

CARDIOVASCULAR ACADEMY CONGRESS

& INTERNATIONAL YOUNG ACADEMY OF CARDIOLOGY E-CONGRESS

2021

17-21
SEPTEMBER
2021

ELEXUS HOTEL
GİRNE, K.K.T.C.
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International
Young Academy of Cardiology

GENH
CONGRESS

CARDIOVASCULAR
ACADEMY SOCIETY

Dear Colleagues,

As you can see the massive and rapid spread of COVID-19 and its impact on many countries in the world, many restrictions are applied by all governments to limit the spread of the virus. So for the second year and for the sake of safety of our faculty, delegates and staff, we are presenting our congress in an electronic online version.

The annual congress of the International Young Academy of Cardiology (IYAC), that was usually held at Northern Cyprus will be now converted to an **E-Congress**. We are delighted to invite you to take part in the third International Young Academy of Cardiology **E-Congress** under the theme: "**Unlimited Knowledge**".

The goals of this congress are to provide innovative and comprehensive overview of the state-of-the-art in cardiovascular medicine. As well as being a strong platform for connecting cardiologists from all over the world. The program this year aims to update the current knowledge and to offer interactive sessions and discussions as many as possible to the young cardiologists and cardiology trainees, and to help them to improve their skills in everyday clinical practice.

Mark your calendars now and join us each day – from 17 September to 21 September 2021.

We look forward to meeting you at **IYAC 2021 E- Congress**.

Sincerely Yours,

International Young Academy of Cardiology Congress Organizing Committee

ORGANIZING AND SCIENTIFIC COMMITTEE

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**INTERNATIONAL
YOUNG ACADEMY OF CARDIOLOGY
E-CONGRESS**

2021

**SCIENTIFIC
PROGRAM**

17 September 2021, Friday

08:30-09:15 Abstract Session 1-Aorta and Aortic Valve

Chairpersons: Oktay Ergene TR, Tuğba Kemaloğlu Öz TR

- OP-001** Prognostic Utility of Systemic Immune-Inflammation Index in Aortic Stenosis Patients Treated With Transcatheter Aortic Valve Implantation
Mehmet Erdoğan
- OP-002** Effect of New-Onset Permanent Left Bundle Branch Block On Long-Term Outcomes In Patients Undergoing Transcatheter Aortic Valve Implantation
Serkan Aslan, Ezgi Gültekin Güner
- OP-003** Arterial Stiffness and Myocardial Remodeling in Patients With Aortic Coarctation Treated by Surgery or Transcatheter Stent Implantation
Pelin Köşger, Ayşe Sülü, Birsen Uçar
- OP-004** Implantation: Manufacturer Based Sheath-To Femoral Artery Ratio as a New Predictor
Sinem Çakal, Beytullah Çakal, Bilal Boztosun
- OP-005** Assessment of Monocyte/HDL Ratio in Ascending Aortic Dilatation
Gonul Aciksari
- OP-006** Coarctation of The Aorta and Relapsing Supravalvular Aortic Stenosis in Congenital Cutis Laxa Syndrome
Turkan Seda Tan

COFFEE BREAK

09:30-10:15 Abstract Session 2-Chest Pain

Chairpersons: Ömer Kozan TR, Mehmet Kış TR

- OP-007** Association Between Psychiatric Symptoms and Pain Severity in Individuals Who Complain of Noncardiac Chest Pain
Ibrahim Yağcı, Yasin Taşdelen, Fatih Aydın
- OP-008** Evaluation of The Culprit Lesion Changes Over Time in Patients Presenting With Acute Myocardial Infarction and Prior Coronary Artery Bypass Grafting
Murat Gençaslan, Beytullah Çakal, Murat Çaylı
- OP-009** Assessment of Possible Predictors of Access Site Complications in Non-STEMI patients Treated Via Femoral Arterial
Barış Şimşek, Duygu İnan
- OP-010** Digit Ratio (2D:4D) in Turkish Male Patients With Myocardial Infarction
Nihan Çağlar, Ece Celebi
- OP-011** The Time to Return to Work and Time of Adaptation to Social Life of Patients to Whom Primary or Elective Coronary Angiography Were Applied
Özkan Karaca, Mehdi Karasu, Mehmet Ali Kobat, Tarik Kıvrak
- OP-012** Value of Age, Creatinine, and Ejection Fraction (ACEF Score) for Clinical Prognosis of Young Patients With ST-Segment Elevation Myocardial Infarction Under The Age of 40
Bektas Murat, Selda Murat, Gurbet Ozge Mert

COFFEE BREAK

10:30-11:15 Abstract Session 3-Covid-19

Chairpersons: *Abdallah Almagraby EG, Ömer Görkem Göldağ TR*

- OP-013** Relationship of Magnesium With Myocardial Damage and Mortality in Patients With COVID-19
Serhat Caliskan, Seyda Gunay, Deniz Sıgırlı
- OP-014** Brain Natriuretic Peptide Levels In Patients With COVID-19
Gökhan Alıcı, Alaa Quisi
- OP-015** Relationship Between Atherogenic Indices and Cardiovascular Diseases in Patients With Covid-19 Pneumonia
Hakan Kilci, Adem Melekoğlu, Özgür Selim Ser
- OP-016** Comparison of Covid-19 Patients According to the Survival Time in the Turkish Population: A Retrospective Study
Adem Atıcı, Ömer Faruk Baycan
- OP-018** Association of Laboratory Markers With Mortality In Patients With COVID-19 Pneumonia
Emirhan Hancıoğlu, Serkan Karahan

COFFEE BREAK**11:30-12:30 Abstract Session 4-Pulmonary Embolism and Pulmonary Hypertension**

Chairpersons: *Mesut Demir TR, Tarık Kıvrak TR*

- OP-019** The Benefit of The Systemic Immun-Inflammation Index In The Differential Diagnosis of Massive And Non-Massive Pulmonary Embolism
Ferit Büyük
- OP-020** Newly Defined Prognostic Indicator For Patients With Acute Pulmonary Embolism: Systemic Immune-Inflammation Index
Muhsin Kalyoncuoğlu, Halil İbrahim Biter, Mehmet Mustafa Can
- OP-021** Pinched Thrombus in The Patent Foramen Ovale Causing Pulmonary and Paradoxical Systemic Embolism
Oktay Şenöz, Zeynep Emren
- OP-022** The Role of Computed Tomography in The Calculation of Pulmonary Artery Systolic Pressure in Patients With Pulmonary Hypertension
Nesrin Gündüz, Lütfi İhsan Kuru
- OP-023** The Role of The Frontal QRS-T Angle in The Diagnosis of Acute Pulmonary Embolism
Ekrem Şahan, Semih Aydemir
- OP-024** Riociguat May Be Effective in Combined Therapy in Pulmonary Hypertension Due to Connective Tissue Disease
İdris Buğra Çerik, Emin Koyun
- OP-025** Controlling Nutrition Status Score and Mortality in Patients With Acute Pulmonary Embolism Admitted to The Intensive Care Unit
Fahrettin Katkat, Sinan Varol

Lunch

13:00-13:45 Abstract Session 5-Electrocardiogram**Chairpersons:** *Özgür Kırbaş TR, Selvi Öztaş TR*

- OP-026** Evaluation of Changes in Ventricular Repolarization Parameters in Morbidly Obese Patients Undergoing Bariatric Surgery
Haci Murat Gunes
- OP-027** P Wave and QT Dispersion in Patients With Subclinical Hypothyroidism
Cennet Yıldız, Ahmet Karakurt
- OP-028** Evaluation of Arrhythmia Frequency With Holter Electrocardiography in Pregnant Patients With Palpitation Complaints
Mehmet Kış
- OP-029** Tpeak-Tend Interval, and Tp-e/QT Ratio in Patients With Essential Tremor
Emine Altuntaş
- OP-031** The Relationship Between Body Mass Index and Changes in Ventricular Repolarization Markers During Exercise
Ajar Koçak

COFFEE BREAK**14:00-14:45 Abstract Session 6-Pericardial Diseases****Chairpersons:** *Onur Taşar TR, Yasmin Rustamova AZ*

- OP-032** Effect of Prone Position Aspiration to The Result of Pericardiocentesis
Aycan Esen Zencirci
- OP-033** An Unusual Cause of Pericardial Effusion Resulting Tamponade: Angiosarcoma
Zeynep Yapan Emren, Oktay Şenöz
- OP-034** Rare But Always Keep in Mind: Purulent Pericarditis
Ozlem Arican Ozluk, Huseyin Akdogan, Omer Faruk Kahraman, Ozgur Dagli
- OP-035** Coronary Subclavian Steal Syndrome Presenting With Acute Coronary Syndrome
Hande Seymen, Muhammed Emre Güleşir
- OP-036** Perimyocarditis As First Sign of High-Grade B-Cell Lymphoma
Dilay Karabulut, Gungor Ilayda Bostanci Alp, Ibrahim Faruk Akturk, Fatma Nihan Turhan Caglar

COFFEE BREAK

15:00-16:00 Abstract Session 7-Biomarkers

Chairpersons: *Veysel Özgür Barış TR, Yasemin Doğan TR*

- OP-039** A Systematic Review and Meta-Analysis on Prognostic Value of Mean Platelet Volume (MPV) in Patients With Acute Coronary Syndrome
Akhmetzhan Galimzhanov, Erhan Tenekecioglu, Farida Rustamova
- OP-040** Evaluation of Inflammatory Markers in Patients With Isolated Myocardial Bridge: A Case-Control Study
Tufan Çınar, Mert Ilker Hayiroğlu, Murat Selçuk, Vedat Çiçek, Mert Babaoğlu, Suha Asal, Sahhan Kılıç, Samet Yavuz, Ahmet Lütfullah Orhan
- OP-041** The Predictive Value of Crp / Albumin Ratio on The Development of Contrast Induced Nephropathy in Non-ST- Elevated Myocardial Infarction Patients Who Have Undergone Percutaneous Coronary Intervention
Halil Ibrahim Biter
- OP-042** Evaluation of The Relationship Between Coronary Artery Ectasia and MPV to Platelet Count Ratio and Systemic Immune-Inflammation Index
Özge Özcan Abacıoğlu
- OP-043** C Reactive Protein to Albumin Ratio is Predictive of Acute Stent Thrombosis After Primary Percutaneous Primary Intervention
Eser Açıkgöz, Sadık Kadri Açıkgöz, Gökhan Çiçek
- OP-044** Increased De Ritis Ratio May Indicate Cardiovascular Risk in PCOS Patients
Muzaffer Kahyaoglu, Beyzanur Kahyaoglu, Murat Can Guney

COFFEE BREAK**16:15-17:15 Abstract Session 8-Coronary Artery Diseases 1**

Chairpersons: *Volkan Emren TR, Elif İlkay TR*

- OP-046** Impella for Cardiogenic Shock and High-Risk Percutaneous Coronary Intervention: a Single-Center Experience
Mariana Brandão, Pedro Queirós, Pedro Gonçalves Teixeira, Mariana Ribeiro Silva, Gualter Santos Silva, Diogo Santos Ferreira, João Gonçalves Almeida, Gustavo Pires Moraes, Alberto Rodrigues, Marco Oliveira, Daniel Caeiro, Ricardo Fontes Carvalho
- OP-047** Percutaneous Coronary Interventions to Native vs. Graft Vessels in Acute Myocardial Infarction Patients With History of Coronary Artery Bypass Grafting
Bedrettin Boyraz, Burhan Aslan
- OP-048** Evaluation of Inflammation and Atherogenic Indices in Coronary Artery Ectasia
Abdulmecit Afşin, Emin Asoğlu
- OP-049** Association Between Nontraditional Lipid Profiles and Isolated Coronary Artery Ectasia
Gonul Aciksari, Ramazan Asoglu

17:30-17:45 Opening Speech

18:00-19:00 Nightmares and Solutions in Cath Lab

Chairpersons: Ömer Kozan TR, Mohammed Zahran EG

Acute Coronary Syndromes

Mirvat Alasnag SA

Elective Coronary Angiography

Abdalraouf Omar LY

Challenging Echo Cases: Seen Once in Lifetime'

Mohamed Ayman EG

COFFEE BREAK

19:15-20:15 Recent Advances in Arrhythmia

Chairpersons: Berkay Ekici TR, Zainab Dakhil IQ

Role of Echocardiography in Arrhythmia

Corrado Fiore IT

Developments in Cardiac Devices

Lorenzo Costantini IT

Tips and Tricks in Temporary Pacemaker Implantation

Elnur Isayev AZ

Side Effects and Drug Interaction of OACs

Fulya Avci Demir TR

18 September 2021, Saturday**09:00-09:45 Abstract Session 9-Heart Failure***Chairpersons: Hakan Altay TR, Şeyda Günay TR*

- OP-051** The Prognostic Performance of Platelet to Lymphocyte Ratio Among Patients With Acute Decompensated Heart Failure
Caglar Ozmen
- OP-052** Sex Differences in Patients With Mid-Range Heart Failure
Onur Argan
- OP-053** The Relationship Controlling Nutritional Status Score and Hospital Mortality in Decompensated Heart Failure Patients
Ipek Büber, Oğuz Kılıç
- OP-054** Evaluation of Sleep Quality in Patients With Reduced Ejection Fraction Heart Failure Using Angiotensin Receptor Neprilysin Inhibitor
Ferhat Işık, Burhan Aslan, Abdurrahman Akyüz
- OP-055** Is There a Different Mechanism on The Basis of Heart Failure Emerging in Sleep Apnea?: Correlation of Myocardial Efficiency Index With NT-Pro BNP
Ibrahim Ersoy, Fulya Avcı Demir

COFFEE BREAK**10:00-10:45 Abstract Session 10-Interesting Cases***Chairpersons: Sara Moscatelli UK, Çağlar Emre Çağlayan TR*

- OP-057** Pericardial Effusion and Cardiac Tamponade After Covid-19 Vaccine: A Rare Case
Muammer Karakayalı, Yusuf oflu, Timor Omar
- OP-058** Free-Floating Right Atrial Thrombus With Acute Pulmonary Embolism Treated With Heparin Infusion
Fatih Özkan, Hasan Ali Barman, Serhan Yıldırım, Sait Mesut Doğan
- OP-060** A Case of Acute Upper Limb Ischemia in a Patient With COVID-19
Muzaffer Kahyaoglu, Murat Can Guney
- OP-061** A Different Approach in The Differential Diagnosis of The Patient With Chronic Diarrhea, TTR Amiloidosis
Muhammet Mücahit Tiryaki, Sadık Volkan Emren, Nihan Kahya Eren
- OP-062** A Rare Complication of Surgical Atrial Septal Defect Repair: Iatrogenic Diversion of The Inferior Vena Cava Into The Left Atrium
Emre Özdemir, Nihan Kahya Eren

COFFEE BREAK

11:00-11:45 Abstract Session 11-Cardiac Imaging

Chairpersons: *Fulya Avcı Demir TR, Öykü Gülmez TR*

- OP-063** Myocardial Strain Assessment by 2D Speckle Tracking Echocardiography in Patients With Congenital Myopathy
Murat Çap, Askeri Türken, Emrah Erdoğan
- OP-064** Decreased Intracardiac Volume Reserve and Increased Sympathetic Response in The Mechanism of Vasovagal Syncope: A Comprehensive Echocardiographic and Holter ECG Monitoring Study
Pelin Karaca Ozer
- OP-065** Subclinical Myocardial Systolic Function In patients With Arteriovenous Fistulas After Renal Transplantation
Cansu Selcan Akdeniz, Ibrahim Halil Sever
- OP-066** CHA2DS2-VASc Score is Higher in Patients With Left Atrial Appendage Thrombus on Transesophageal Echocardiography
Hasan Koca
- OP-067** Assesment of Aortic Bioprothesis Valve Thrombosis: Two and Three Dimensional Transesophageal Echocardiography Study
Semih Kalkan, Ahmet Karaduman, Gokhan Kahveci
- OP-120** Cardiac Changes With Subclinical Hypothyroidism in Women With Metabolic Syndrome
Bedri Caner Kaya, Berna Kaya, Mehmet Memduh Baş

COFFEE BREAK**12:00-13:00 Abstract Session 12-Biomarkers 2**

Chairpersons: *Gönenç Kocabay TR, Mustafa Yenerçay TR*

- OP-069** Mean Platelet Volume to Lymphocyte Ratio in The Prediction of SYNTAX Score in Patients With Non-ST-Elevation Myocardial Infarction
Emre Yılmaz, Ahmet Karagöz, Zeki Yüksel Günaydın, Ertan Aydın, Aslı Vural, Devrim Kurt
- OP-070** Relationship Between Lymphocyte to C Reactive Protein Ratio and Thrombus Burden in Patients With Acute Coronary Syndrome
Onur Osman Şeker, Metin Çoksevrim
- OP-072** Relation of Total Cholesterol to High Density Cholesterol Ratio on Predicting Coronary Artery Atherosclerosis
Ebru Ozenc
- OP-074** Atherogenic Indexes Versus Hematologic Inflammatory Indexes: What is Most Useful Predictor of Coronary Slow Flow?
Ferhat Dındas, Emin Koyun, Idris Bugra Cerik, Anıl Sahin, Celal Kilit, Mustafa Dogdus
- OP-075** Experimental Investigation of Effect of Radiopaque Substances on Fibrinogen Results
Mustafa Beğenç Taşcanov, Zulkif Tanriverdi, Ataman Gonel

Lunch

13:30-14:30 Abstract Session 13-Acute Coronary Syndromes

Chairpersons: *Seçkin Pehlivanoglu TR, Süleyman Çağan Efe TR*

- OP-077** Optimal Timing for Complete Revascularization in Patients With ST-Segment Elevation Myocardial Infarction: Index Hospitalization Versus Early After Discharge
Betül Balaban Koçaş
- OP-078** The Effect of Total Occlusion Pattern in Hospital and Long Term Clinical Outcomes in Acute ST Segment Elevated Myocardial Infarction
Yusuf oflu, İlhan İlker Avcı, Gönül Zeren, Barış Şimşek, Görkem Ayhan, Duygu İnan, Can Yücel Karabay
- OP-079** The Prediction of No-Reflow in Young ST Segment Elevation Myocardial Infarction Patients by Simple Electrocardiographic Score
Ayça Gümüşdağ, Muhammed Süleymanoğlu
- OP-080** Presence of Fragmented QRS is Associated With Major Adverse Cardiac Events in Patients With Acute Coronary Syndrome
Ayhan Küp, Mehmet Celik
- OP-082** Inflammatory Parameters and Homocysteine Dilemma in Patients With Diabetes Mellitus Presenting With First Time Acute Coronary Syndrome
Songül Usalp, Emine Altuntaş, Bayram Bagirtan, Enver Yücel, Ali Bayraktar, Behzat Özdemir, Filiz Çelebi, Şükrü Çetin, Kanber Ocal Karabay

COFFEE BREAK**14:45-15:30 Abstract Session 14-Coronary Artery Disease 2**

Chairpersons: *Özgen Şafak TR, Çağlar Özmen TR*

- OP-083** Nutritional Status as a New Prediction Tool for Coronary Collateral Development
Kürşat Akbuğa, Özge Kurmuş Ferik, Kadriye Gayretli Yayla, Turgay Aslan, Murat Eren, Mustafa Karanfil, Berkay Ekici, Aycan Fahri Erkan, Ebru Akgül Ercan, Celal Kervancioğlu
- OP-084** The Relationship Between Levels of Sex Steroids and Coronary Collateral Circulation in Men Patients With Coronary Artery Disease
Aslan Erdoğan, Ender Özgün Çakmak, Cevat Kıрма
- OP-085** Does Adding Duration of ST Depression to Duke Treadmill Score Effect Diagnostic Accuracy of EET to Predict Obstructive Coronary Artery Disease ?
Nihan Çağlar, Gulsum Oztimer
- OP-086** CHA2DS2-VASc Score as a Predictor of Graft Failure After Coronary Artery Bypass Surgery
Omer Tasbulak, Ahmet Anil Sahin, Arda Guler
- OP-087** Successful Treatment of Acute Total Occlusion of The Left Main Coronary Artery (LMCA) Lesion in a Patient Presented With Cardiogenic Shock
Özgür Selim Ser, Hakan Kilci, Kadriye Kılıçkesmez
- OP-088** Which Type of Dyslipidemia is More Effective in The Severity of Coronary Artery Disease?
Turgay Aslan

COFFEE BREAK

16:45-17:30 Abstract Session 15-Interesting Cases

Chairpersons: Ebru Özpelit TR, Ahmet Öz TR

OP-089 Revascularization of Acute Renal Arterial Occlusion

Kubilay Erselcan

OP-090 Takotsuba Cardiomyopathy After mRNA-1273 SARS-CoV-2 Vaccination

Büşra Mavi, Mehmet Pişirici, Nihan Çağlar

OP-091 Fabry Disease With Atrioventricular Block and Severe Aortic Stenosis: A Dangerous Combination

Dilay Karabulut, Hasan Ali Sinoplu, Ersan oflar, Ibrahim Faruk Akturk, Fatma Nihan Turhan Caglar

OP-093 A Case Report of Permanent Pace Macker Inserted, A Case of Large Vessels Vasculitis That Consists With Sweating, Weight Loss And Cough

Hasan Kudat, Merve Balci, Bahar Artım Esen, Ahmet Bilge Sözen

COFFEE BREAK

18:00-19:00 Artificial Intelligence in Imaging

Chairpersons: Tuğba Kemaloğlu Öz TR, Alessia Gimelli IT

AI and Ethics in Imaging

Bernard Cosyns BE

The Use of AI in Imaging

Erwan Donal FR

What Do Cardiologists Need From AI?

Didem Oğuz US

In Collaboration With



EACVI
European Association of
Cardiovascular Imaging

COFFEE BREAK

19:15-20:15 Meet the Editors

Chairpersons: Mehdi Zoghi TR, Navin Nanda US

What Do Editors Want?

Guiseppa Biondi-Zoccai IT

Where Should We Start?

Erin Michos US

The Key Points of a Case Report

Ritu Thamman US

Difficulties of Being a Young Researcher and Solutions

Jolanda Sabatino IT

19 September 2021, Sunday

09:00-09:45 Abstract Session 16-Hypertension

Chairpersons: Elnur Alizade TR, Emrah Erdoğan TR

- OP-095** Clinical Usability of The Most Important Three Atherogenic Indices for Prediction of Hypertension in Young Population
Mehmet Özgeyik
- OP-096** The Association of Non-Dipper Hypertension With Aortic Sclerosis in Hypertensive Patients
Savaş Özer, İsmail Barkın Işık
- OP-097** The Relationship of Sleep Quality With Dipper and Non-Dipper Blood Pressure Patterns in Essential Hypertensive Patients During Covid-19 Pandemic
Erkan Demirci
- OP-098** The Effect of Ramadan Fasting on Endothelial Function in Patients With Hypertension
Bekir Çalapkorur
- OP-099** Prognostic Value of Frontal Plane QRS-T Angle in Pulmonary Hypertension: Experience From a Tertiary Center
Sena Sert
- OP-100** Pulmonary Hypertension Patient With a Mean Pap of 23 mmhg by Right Heart Catheterization
Turgay Aslan, Celal Kervancıoğlu, Özge Kurmuş, Murat Eren, Kürşat Akbuğa

COFFEE BREAK

10:00-10:45 Abstract Session 17-Peripheral Artery Diseases

Chairpersons: Onur Taşar TR, Hakan Kılıcı TR

- OP-101** The Effect of Superficial Femoral Artery Occlusion on Primary Patency and Amputation Free Survival After Endovascular Management of Infrarenal Aorta Total Occlusions
Şeyhmus Külahçioğlu, Regayip Zehir
- OP-102** The Link Between Ventricular Repolarization Variables and Carotid Artery Stenting
Munevver Sari, Mehmet Ayturk
- OP-103** Relationship Between Atherosclerotic Risk Factors and Vertebral Artery Flow in Asymptomatic Adults
Rengin Çetin Güvenç
- OP-104** Approach to The Case of Renal Artery Perforation Occurred During The Stenting Procedure Performed in The Renal Artery Stenosis
Ömer Tepe, Abdulkadir İltaş, Çağlar Özmen, Engin Onan, Mustafa Demirtaş, Saime Paydaş
- OP-105** The Effect of Successful Endovascular Revascularization on Aortic Augmentation Index in Peripheral Artery Patients
Khaganı Isgandarov, Muhammed Bahadır Omar, Abdulrahman Naser, Selçuk Pala, Müslüm Şahin

COFFEE BREAK

11:00-11:45 Abstract Session 18-Drugs

Chairpersons: Berkay Ekici TR, Serkan Asil TR

OP-107 Oral Anticoagulants in Early Diagnosis of Cancer

Aslı Kurtar Mansirođlu

OP-108 Always Wondered But Not Known: What is The Best Time to Take Warfarin for Patients With Metallic Prosthetic Valves?

Ömer Furkan Demir, Ozlem Arican Ozluk, Huseyin Akdogan, Dogan Ormanci

OP-109 Biological Heart Valves Versus Mechanical Heart Valves With Effective Anticoagulation With Low-Dose Warfarin During Pregnancy

Ahmet Güner, Ezgi Gültekin Güner

OP-110 Changes of Nutrition and Lifestyle Habits Among Cardiovascular Drug Users: A CVSCORE-TR Sub-Study

Mehmet Özgeyik, Taner Sen, Lale Dinc Asarcikli, Duygu Kocyigit, Mustafa Begenc Tascanov, Ahmet Ersecgin, Kamil Tuluçe, Gulay Gok, Mehmet Kis, Yavuzer Koza, Didem Oguz, Mehdi Zoghi

OP-111 Investigation of Changes in Superficial ECG of Combinations Containing Hydroxychloroquine Used in the Treatment of COVID-19

Serdal Baştuđ

OP-112 Resolution of COVID-19 Induced Thrombus via Absiksimab in a 33 Years Old Woman Presenting With NSTEMI

Emre Yılmaz, Ahmet Karagoz, Zeki Yuksel Gunaydin, Ertan Aydın, Aslı Vural, Devrim Kurt

COFFEE BREAK**12:00-13:00 Abstract Session 19-Miscellaneous**

Chairpersons: Gülay Gök TR, Mustafa Dođduş TR

OP-113 The Relationship Between Prolidase Activity and Paroxysmal Atrial Fibrillation

Mustafa Beğenç Taşcanov, Fatih Güngören

OP-115 A New Scoring to Predict Cerebrovascular Event in Patients With New-Onset Atrial Fibrillation After Coronary Artery Bypass Graft Surgery

Ali Rıza Demir, Arda Can Dođan

OP-116 Predictors of Major Adverse Cardiac Events After Renal Transplantation

Umut Kocabas

OP-117 Retrospective Analysis of Cardiac Implantable Device Extractions in Adult Patient

Yusuf Hoşođlu, Abdilmecit Afşin, Ayşe Hoşođlu

OP-119 Assessment of Exercise Stress Test Parameters in Patients With Erectile Dysfunction

Ersan oflar, Cennet Yildiz, Dilay Karabulut, Abdulcelil Sait Ertuđrul, Fatma Nihan Turhan Caglar, Faruk Akturk

OP-121 Maternal and Perinatal outcomes in Pregnant Women with Left Ventricular Noncompaction

Aysun Erdem Yaman

COFFEE BREAK

14:00-15:00 What Should we Know in Our Daily Practice?

Chairpersons: Oktay Ergene TR, Nigar Babazade AZ

Do We Pay Enough Attention to Cardiac Rehabilitation?

Francesco Perone IT

COVID-19 Impact on The Heart

Emna Allouche TN

What Should a Cardiologist Know About Congenital Echo?

Giovanni Di Salvo IT

Lung Ultrasound in Covid Era

Hatem Soliman UK

COFFEE BREAK**15:15-16:15 When Should We Make Fast Decision?**

Chairpersons: Abdallah Almaghraby EG, Guido Tavazzi IT

Pulmonary Embolism in COVID-19 Era

Awad Zeid EG

Post-Cardiac Arrest Syndrome

Pablo Jorge ES

Early Identification of the High Risk Patient in the CCU/ICU

Konstantin Krychtiuk AT

Emerging Therapies in Cardiogenic Shock

Christophe Vandenbriele BE



In Collaboration With

ACVC
Association for
Acute CardioVascular Care

COFFEE BREAK**16:30-17:30 Back to The Future**

Chairpersons: Alexandru Mischie FR, Mahmoud Hassan US

Patients' Follow-up in COVID-19 Era

Aaysha Cader BD

Virtual/Augmented Realities For Cardiac Imaging

Costas Papadopoulos GR

The Role of Social Media in Cardiology

Ahmed Mohsen EG



In Collaboration With

International Society for
Telemedicine & eHealth

COFFEE BREAK**17:45-18:45 Advances and Perspectives in Heart Failure (HF)**

Chairpersons: Yasmin Rustamova AZ, Milica Aleksic SRB

When is Cardiac MRI Needed in HF?

Sarah Moharem-Elgamal EN

Acute HF Management

Shaymaa Abdelmaboud Bahrain

Cardiotoxicity and HF

Sabeeda Kadavath US

Novel Member in HF Campaign

Madiha Fatima PK

19:00-19:15 Closing Speech

**INTERNATIONAL
YOUNG ACADEMY OF CARDIOLOGY
E-CONGRESS**

2021

**ORAL
PRESENTATIONS**

OP-001 PROGNOSTIC UTILITY OF SYSTEMIC IMMUNE-INFLAMMATION INDEX IN AORTIC STENOSIS PATIENTS TREATED WITH TRANSCATHETER AORTIC VALVE IMPLANTATION

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Objective: Systemic immune-inflammation index (SII) is a novel marker of inflammation, including neutrophil, lymphocyte and platelet cell counts. The role of SII as a biomarker has been investigated several cardiovascular disease such as acute and chronic coronary syndrome, acute pulmonary embolism and aortic stenosis. This retrospective study aimed to investigate the prognostic value of SII in a propensity score-matched cohort of severe calcific AS patients undergoing transcatheter aortic valve implantation (TAVI).

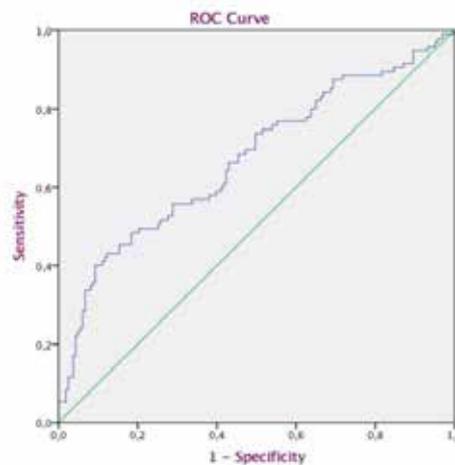
Methods: The primary end-point of the study was all-cause mortality during the follow-up period. Propensity score matching analysis was performed to adjust for the influence of differences in the patients' baseline features and comorbid situations on survival.

Results: Between 2011 and 2018, a total of 448 patients underwent TAVI procedure at our center due to severe calcific aortic stenosis (AS). Among them, 390 patients were included in the statistical analysis as unmatched study cohort after the application of exclusion criteria. Subsequently, propensity score matching yielded 258 patients in total as the final paired study population. The median follow-up duration of the whole study patients was 29.5 (30.7) months. ROC analysis identified a cut-off value of SII 792×10^3 for all-cause mortality after TAVI with a sensitivity of 64% and specificity of 58%. The area under the curve was 0.67 (95% CI:0.60-0.74, $p < 0.001$). Patients were classified into two groups after a 1:1 propensity score matching analysis: Low SII ($< 792 \times 10^3$) (n=129) and High SII ($\geq 792 \times 10^3$) (n=129). All-cause mortality was significantly higher in high SII group compared to low SII group (47.3%, 26.4%, $p < 0.001$, respectively).

Conclusion: Preprocedural SII levels were associated with long-term mortality in severe calcific AS patients undergoing TAVI. This cheap and simple marker can help us to predict long-term mortality in these patients.

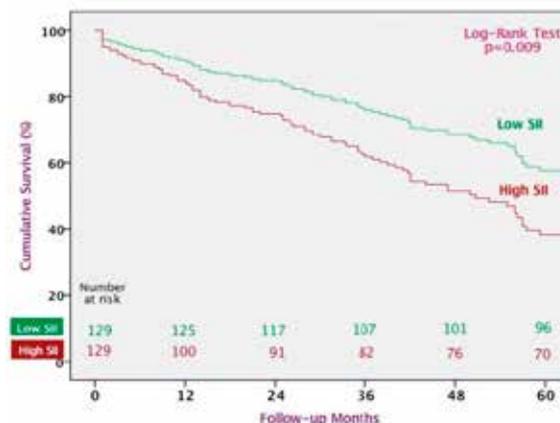
Keywords: Aortic stenosis, outcomes, prognosis, systemic immune-inflammation index, transcatheter aortic valve implantation

Figure 1



The receiver operating characteristic (ROC) curves of the systemic immune-inflammation index (SII) in predicting all-cause mortality in aortic stenosis patients after a transcatheter aortic valve implantation.

Figure 2



Kaplan-Meier curve showing 5-year all-cause mortality according to systemic immune-inflammation index (SII). SII dichotomization; SII $< 792 \times 1000$ (green line) and SII $\geq 792 \times 1000$ (red line).

Table 1. Baseline characteristics laboratory and transthoracic echocardiography findings and peri-procedural and post-procedural features of the study population

Variables	Before Propensity Score Matching (n=390)			p value	After Propensity Score Matching (n=258)			p value
	All Patients (n=390)	Low SII (n=217)	High SII (n=173)		All Patients (n=258)	Low SII (n=129)	High SII (n=129)	
Age, years	79 (10)	78 (10)	79 (11)	0.49	79 (10)	77 (11)	77 (11)	0.71
Gender (male), n (%)	168 (43)	101 (46)	67 (40)	0.12	108 (42)	55 (43)	53 (41)	0.80
Diabetes mellitus, n (%)	117 (30)	57 (26)	60 (35)	0.07	78 (30)	39 (30)	39 (30)	1.00
Hypertension, n (%)	336 (86)	184 (85)	152 (88)	0.38	225 (87)	114 (88)	111 (86)	0.57
Previous CABG, n (%)	99 (25)	61 (28)	38 (22)	0.16	59 (23)	28 (22)	31 (24)	0.65
COPD, n (%)	156 (40)	77 (35)	79 (46)	0.04	112 (43)	58 (45)	54 (41)	0.61
CAD, n (%)	286 (73)	164 (76)	122 (70)	0.26	190 (74)	96 (74)	94 (73)	0.77
STS PROM, %	5.11 (3.80)	4.36 (3.87)	5.89 (3.69)	<0.001	5.30 (3.65)	4.91 (3.98)	5.54 (3.52)	0.93
Atrial fibrillation, n (%)	97 (25)	44 (20)	53 (30.6)	0.01	67 (26)	35 (27)	32 (25)	0.67
GFR (mL/min/m2)	63 (34)	70 (33)	159 (31)	0.001	63 (34)	63 (35)	63 (31)	0.98
Neutrophil (K/uL) x 1000	4.86 (2.57)	4.16 (1.74)	5.99 (2.48)	<0.001	5.01 (2.45)	4.34 (1.71)	5.74 (2.58)	<0.001
Lymphocyte (K/uL) x 1000	1.66 (0.79)	1.84 (0.71)	1.30 (0.77)	<0.001	1.67 (0.81)	1.83 (0.64)	1.41 (0.79)	<0.001
Platelet (K/uL) x 1000	249 (116)	206 (75)	313 (78)	<0.001	255 (116)	205 (75)	309 (87)	<0.001
Triglycerides (mg/dl)	103 (62)	103 (68)	105 (57)	0.43	104 (71)	104 (79)	105 (63)	0.61
HDL (mg/dl)	44 (17)	45 (18)	43 (17)	0.50	43 (17)	44 (17)	42 (18)	0.83
LDL (mg/dl)	100 (47)	102 (46)	95 (48)	0.21	100 (48)	101 (44)	98 (44)	0.59
AVA (cm2)	0.70 (0.25)	0.70 (0.27)	0.70 (0.25)	0.47	0.70 (0.25)	0.70 (0.25)	0.70 (0.25)	0.87
Peak gradient (mmHg)	80 (26)	82 (29)	79 (25)	0.16	82 (24)	82 (23)	82 (27)	0.67
Mean gradient (mmHg)	47 (18)	48 (18)	47 (16)	0.37	49 (27)	48 (16)	50 (18)	0.62
LVEF, %	57 (20)	59 (18)	56 (23)	0.07	57 (19)	57 (16)	57 (20)	0.98
Beta blocker, n (%)	176 (45)	103 (47)	73 (42)	0.29	115 (45)	60 (46)	55 (43)	0.53
RAS blocker, n (%)	254 (65)	150 (69)	104 (60)	0.06	161 (62)	81 (63)	80 (62)	0.89
CCB, n (%)	131 (34)	70 (32)	61 (35)	0.55	91 (35)	48 (37)	43 (33)	0.51
Digoxin, n (%)	23 (6)	13 (6)	10 (6)	0.93	20 (8)	10 (8)	10 (8)	1.00
Amiodarone, n (%)	11 (3)	7 (3)	4 (2)	0.58	7 (3)	3 (2)	4 (3)	0.70
Anticoagulant, n (%)	102 (26)	47 (22)	55 (32)	0.02	71 (27)	37 (29)	34 (26)	0.67
Successful implantation, n (%)	389 (99.7)	216 (99.5)	173 (100)	0.37	257 (99.6)	128 (99.2)	129 (100)	0.31
Balloon pre-dilatation, n (%)	352 (90.3)	192 (88.5)	160 (92.5)	0.18	231 (89.5)	112 (86.8)	119 (92.2)	0.15
Balloon post-dilatation, n (%)	9 (2.3)	7 (3.2)	2 (1.2)	0.17	3 (1.2)	1 (0.8)	2 (1.6)	0.56
Major Vascular complication, n (%)	24 (6.2)	12 (5.5)	12 (6.9)	0.56	16 (6.2)	8 (6.2)	8 (6.2)	1.00
Major Stroke, n (%)	2 (0.5)	0 (0)	2 (1.2)	0.11	0 (0)	0 (0)	0 (0)	1.00
Pericardial tamponade, n (%)	5 (1.3)	1 (0.5)	4 (2.3)	0.10	2 (0.8)	1 (0.8)	1 (0.8)	1.00

	Before Propensity Score Matching (n=390)				After Propensity Score Matching (n=258)			
Permanent pacemaker, n (%)	31 (7.9)	15 (6.9)	16 (9.2)	0.39	19 (7.4)	8 (6.2)	11 (8.5)	0.47
Moderate/Severe Paravalvular insufficiency, n (%)	78 (20.0)	42 (19.4)	36 (20.8)	0.72	55 (21.3)	27 (20.9)	28 (21.7)	0.87
Transfemoral, n (%)	378 (96.9)	213 (98.2)	165 (95.4)	0.11	250 (96.9)	126 (97.7)	124 (96.1)	0.47
Transaxillary, n (%)	11 (2.8)	4 (1.8)	7 (4.0)	0.19	8 (3.1)	3 (2.3)	5 (3.9)	0.47
Transapical, n (%)	2 (0.5)	1 (0.5)	1 (0.6)	0.87	1 (0.4)	1 (0.8)	0 (0)	0.31
All cause mortality, n (%)	146 (37.4)	43 (19.8)	103 (59.5)	<0.001	95 (36.8)	34 (26.4)	61 (47.3)	<0.001

AVA: aortic valve area; CABG: coronary artery by-pass graft surgery; CAD: coronary artery disease; CCB: calcium channel blocker; COPD: chronic obstructive pulmonary disease; GFR: glomerular filtration rate; HDL: high density lipoprotein; IQR: interquartile range; LDL: low density lipoprotein; LVEF: left ventricular ejection fraction; RAS: renin-angiotensin system; SII: systemic immune inflammatory index; STS PROM: Society of Thoracic Surgeons Predicted Risk of Mortality. Parameters were expressed as median (IQR). * $P < 0.05$ was considered significant for statistical analyses.

OP-002 EFFECT OF NEW-ONSET PERMANENT LEFT BUNDLE BRANCH BLOCK ON LONG-TERM OUTCOMES IN PATIENTS UNDERGOING TRANSCATHETER AORTIC VALVE IMPLANTATION

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OBJECTIVE: New-onset permanent left bundle branch block (NOP-LBBB) is one of the most common conduction disorders after transcatheter aortic valve implantation (TAVI). LBBB causes electrical and mechanical incompatibility of the heart and various adverse hemodynamic effects. Therefore, it is crucial to evaluate the prognostic impact of NOP-LBBB following TAVI to improve outcomes. Studies evaluating the effect of new-onset LBBB after TAVI on long-term clinical outcomes regarding mortality, need for permanent pacemaker, and rehospitalization for heart failure have yielded conflicting results. Most of these studies defined LBBB as any new-onset LBBB that persisted at hospital discharge, although new-onset LBBB resolves spontaneously in more than half of the patients at 30 days. Therefore, these results may not reflect real-world evidence regarding the effects of LBBB in TAVI. Another limitation is that the follow-up period is mostly limited to 1 year or less. Therefore, this study aims to evaluate the prognostic significance of NOP-LBBB following TAVI on long-term clinical outcomes in up to 2 years of follow-up.

METHOD: A total of 195 consecutive patients who underwent TAVI without pre-existing bundle branch block or permanent pacemaker were analyzed for clinical outcomes in an up to 2-year follow-up. Patients were divided into two groups: with or without NOP-LBBB. According to the VARC-3 criteria, NOP-LBBB was defined as any new LBBB (QRS duration ≥ 120 ms) occurring after TAVI that persisted after 30 days.

RESULTS: New-onset LBBB occurred in 80 patients (41.0%) immediately after TAVI and persisted at 30-day in 43 patients (22.1%; NOP-LBBB). Clinical comorbidities were similar among the study population (Table 1). The most common comorbidities in patients were hypertension (68.2%) and coronary artery disease (66.2%). There were no differences between patients with and without NOP-LBBB in terms of all-cause mortality (18.6% vs. 18.4%, log-rank $p = 0.941$), cardiovascular death (11.6% vs. 12.5%, log-rank $p = 0.895$), and heart failure rehospitalization (11.6% vs. 9.2%, log-rank $p = 0.634$) at 2-year follow up (Figure 1). The presence of NOP-LBBB was associated with higher rates of permanent pacemaker (34.9% vs. 14.5%, log-rank $p = 0.003$). NOP-LBBB was also associated with worse left ventricular systolic function at 1 and 2-year follow-up (Figure 2).

CONCLUSION: NOP-LBBB following TAVI was associated with a higher incidence of permanent pacemaker and worsened left ventricular systolic function, but did not increase the risk of all-cause mortality and cardiovascular death or heart failure rehospitalization at 2-year follow-up.

Table 1. Baseline characteristics of the study population according to the presence of NOP-LBBB

Parameters	NOP-LBBB		P value	
	Total (N = 159)	No (N = 152)		Yes (N = 43)
Demographic and clinical characteristics				
Age (years)	78.9 \pm 6.7	78.8 \pm 7.1	79.0 \pm 5.0	0.897
Sex (male)	73 (37.4)	59 (38.8)	14 (32.6)	0.454
Coronary artery disease	129 (66.2)	100 (65.8)	29 (67.4)	0.840
Diabetes	74 (37.9)	59 (38.8)	15 (34.9)	0.639
Chronic kidney disease	57 (29.2)	40 (26.3)	17 (39.5)	0.092
Hypertension	133 (68.2)	103 (67.8)	30 (69.8)	0.803
Previous CABG	51 (26.2)	41 (27.0)	10 (23.3)	0.624
Peripheral vascular disease	50 (25.6)	39 (25.7)	11 (25.6)	0.992
Cerebrovascular disease	10 (5.1)	7 (4.6)	3 (7.0)	0.534
STS score	7.9 \pm 3.5	8.0 \pm 3.4	7.6 \pm 3.9	0.590
Echocardiographic characteristics				
LVEF (%)	56.1 \pm 9.2	56.8 \pm 9.0	54.6 \pm 9.6	0.161
sPAP (mm Hg)	40.2 \pm 12.3	40.6 \pm 13.3	39.5 \pm 9.7	0.633
Aortic valve area (cm ²)	0.74 \pm 0.12	0.75 \pm 0.13	0.71 \pm 0.12	0.094
Aortic valve mean gradient (mm Hg)	49.2 \pm 11.2	49.4 \pm 11.2	48.4 \pm 11.5	0.597
Procedural Characteristics				
Opaque (ml)	155.2 \pm 82.7	149.9 \pm 74.0	168.1 \pm 100.3	0.226
Valve type				0.033
Sapien XT	122 (62.6)	102 (83.6)	20 (16.4)	
Portico	55 (28.2)	39 (70.9)	16 (29.1)	
Lotus	18 (9.2)	11 (61.1)	7 (38.9)	
Balloon post-dilatation	53 (27.2)	37 (24.3)	16 (37.2)	0.094

Parameters	NOP-LBBB		P value	
	Total (N = 159)	No (N = 152)		Yes (N = 43)
Electrocardiographic characteristics				
Sinus rhythm	153 (78.5)	121 (79.1)	32 (20.9)	0.465
Atrial fibrillation	42 (21.5)	31 (20.4)	11 (25.6)	
PR interval (ms)	166 ± 31	168 ± 31	162 ± 32	0.356
QRS duration (ms)	98 ± 20	99 ± 22	94 ± 12	0.145
Ventricular rate (beats per minute)	75 ± 13	76 ± 14	72 ± 11	0.120

Values represent mean ± SD, n (%) or median (interquartile range).
Abbreviations: CABG, coronary artery bypass grafting; LVEF, left ventricular ejection fraction; sPAP, systolic pulmonary artery pressure; STS, Society of Thoracic Surgeons.

Figure 1. Kaplan-Meier curves at 2-year follow-up in patients with and without NOP-LBBB following TAVI. (A) all-cause mortality, (B) cardiovascular death, (C) heart failure hospitalization, and (D) permanent pacemaker implantation.

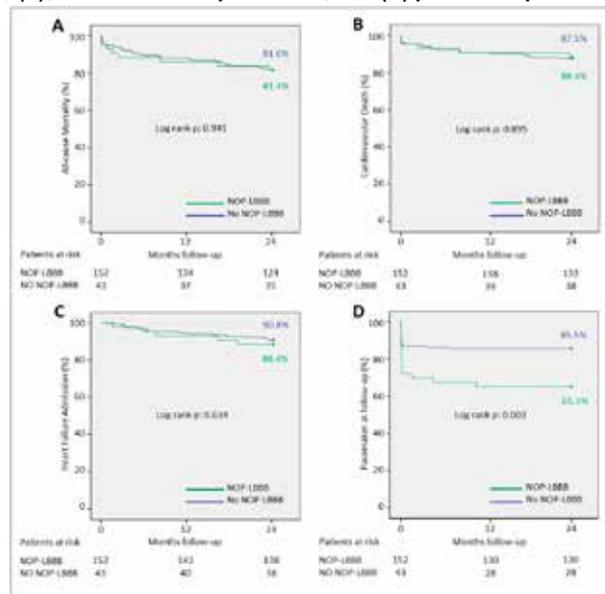
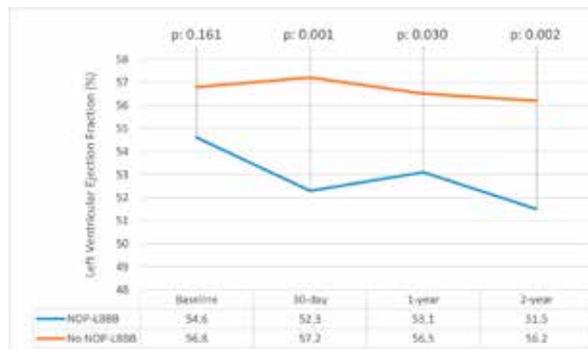


Figure 2. Temporal changes in left ventricular ejection fraction according to the presence of NOP-LBBB following TAVI



Abbreviations: NOP-LBBB, new-onset permanent left bundle branch block; TAVI, transcatheter aortic valve implantation

OP-003 ARTERIAL STIFFNESS AND MYOCARDIAL REMODELING IN PATIENTS WITH AORTIC COARCTATION TREATED BY SURGERY OR TRANSCATHETER STENT IMPLANTATION

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Objective: Despite successful treatment of aortic coarctation, patients still remain at risk of hypertension, left ventricular hypertrophy, and endothelial dysfunction. The present study aimed to investigate the cardiovascular risk factors in children with coarctation who had received different treatment modalities. Methods: Twenty-seven children with successful coarctation surgery or percutaneous stent implantation and 22 healthy peers were examined. Conventional and tissue Doppler echocardiography were applied to investigate myocardial remodeling. Oscillometric pulse wave analysis and carotid intima media thicknesses were performed to vascular damage examination. Results: The all-day systolic blood pressure (SBP) index and load were higher than surgical patients and controls in patients with stent implantation ($p=0.004, p=0.002$, respectively). Their left ventricular mass index and A velocity were higher than controls, and MPI values were higher than both surgical patients and controls ($p<0.001, p=0.02, p=0.01$ respectively). The daytime SBP load and index of surgical patients were higher than controls ($p=0.001, p=0.003$, respectively). Both stent and surgery patients had higher bilateral CIMT z-scores than controls ($p=0.015, p=0.005$, respectively). The pulse wave velocity z-score was higher in stent patients than controls ($p=0.019$). Conclusions: Percutaneous stent implantation has a greater susceptibility to hypertension, myocardial remodeling, and increased arterial stiffness compared to surgical repair at early infancy. However, even patients undergoing early surgical repair are still prone to vascular damage and a high blood pressure profile.

Keywords: Arterial stiffness, coarctation of the aorta, myocardial remodeling, pulse wave velocity

Comparison of arterial stiffness and carotid intima media thickness measurements

Measurements	Surgery, n=15	Stent, n=12	Control, n=22	p-value
24-h average cSBP	97.93±7.74	109.75±7.62	97.68±6.16	<0.001
24-h average cDBP	61.06±6.71	70.58±8.91	65.18±4.13	0.002
Day average cSBP	99.6±8.79	113.5±8.78	99.63±5.67	<0.001
Day average cDBP	66.53±7.14	74.91±10.59	68.63±3.94	0.01
Night average cSBP	96(83,108)	102 (87,118)	91 (82,105)	0.068
Night average cDBP	53.86±6.06	60.72±6.73	58.86±5.64	0.013
PWV, m/s	4.53 ±0.33	5.09±0.37	4.5±0.25	<0.001
z-PWV	-0.21±1.97	0.91±1.14	-0.55±1.02	0.019
Aug@75, %	21.5±10.52	14.25±6.92	19.9±6.95	0.07
cIMT left	0.43 (0.39,0.56)	0.47 (0.42,0.66)	0.40 (0.32,0.47)	<0.001
z-cIMT left	1.07±0.14	1.03±0.4	0.86±0.1	0.015
cIMT right	0.45 ± 0.05	0.51 ± 0.06	0.38 ± 0.04	<0.001
z-cIMT right	1.08±0.13	1.02±0.38	0.84±0.11	0.005

Values are mean±SD, median (minimum- maximum). Aug@75; Augmentation index adjusted for heart rate 75; cDBP; central diastolic blood pressure, cIMT; carotid intima media thickness; PWV; pulse wave velocity; cSBP; central systolic blood pressure.

OP-004 VASCULAR COMPLICATIONS FOLLOWING TRANSCATHETER TRANSFEMORAL AORTIC VALVE IMPLANTATION: MANUFACTURER BASED SHEATH-TO FEMORAL ARTERY RATIO AS A NEW PREDICTOR

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Objectives: Vascular complications (VCs) remain a significant issue in transcatheter aortic valve implantation (TAVI) patients and are associated with worse outcomes. This research analysed the incidence, impact, and predictors of VCs in transfemoral (TF) TAVI and also investigated the predictive role of manufacturer's size charts and a new value manufacturer-derived sheath-to-femoral artery diameter (md-SFAR).

Methods: A total of 223 patients undergoing TF-TAVI were categorized into two groups. Group 1 were divided as eligible and ineligible according to manufacturer's specified limits and Group 2 was dichotomized into eligible and ineligible based on SFAR value of less than or greater than or equal to md-SFAR. Vascular complications (VC) (defined according to VARC-2 criteria) were retrospectively compared.

Results: G1 included 65 patients and Group 2 included 35 patients as ineligible for TF-TAVI. While VCs occurred in 42 cases (18.8%), of those 17 (27.7%) was classified as ineligible in Group 1 whereas 14 (41.2%) was classified as ineligible in Group 2. In a multivariate logistic regression analysis that included md-SFAR, SFAR, current manufacturer's recommendations, iliofemoral moderate to severe calcifications, SFAR>1.05 and peripheral artery disease, only md-SFAR was the sole independent predictors of VCs [OR 3.74, 95% CI: 1.09-12.91, P = 0.036].

Conclusions: According to our results, md-SFAR may provide better patient selection and improve outcomes in TF-TAVI procedures.

Keywords: transcatheter aortic valve implantation, aortic valve disease, complications, vascular access

figure 1

Table 2 Baseline clinical and Procedural characteristics of the Study Population

	Group 1		P	Group 2		P
	TF-TAVI eligible (n=158)	TF-TAVI ineligible (n=65)		TF-TAVI eligible (n=188)	TF-TAVI ineligible (n=35)	
Age (years)	78.9±7.08	78.95±8.79	0.97	79.1±7.34	78.1±8.98	0.45
Men, n (%)	85(53.8)	31(47.7)	0.41	100(52.9)	16(47.1)	0.53
logEuroScore	26.02±4.07	25.68±3.57	0.52	25.79 ±3.85	26.62±4.29	0.26
NYHA III-IV, n (%)	85(53.8)	32(49.2)	0.53	100(52.9)	17(50)	0.75
LVEF %	49.63±13.8	53±10.03	0.07	50.5±13.2	51.2±11.01	0.78
BMI, kg/m ²	26.47±3.63	25.68±3.49	0.13	26.4±3.66	25.35±3.13	0.12
Hypertension, n (%)	143(90.5)	57(87.7)	0.53	172(91)	28(82.4)	0.13
Diabetes, n (%)	38(24.1)	10(15.4)	0.15	45(23.8)	3(8.8)	0.05
CAD, n (%)	87(55.1)	44(67.7)	0.08	108(57.1)	23(67.6)	0.25
PAD, n (%)	26(16.5)	18(27.7)	0.06	33(17.5)	11(31.4)	0.07
GFR(ml/min/1.73 m ²)	68.7±25.9	70.05±25.47	0.73	69.6±25.6	67.1±27.18	0.61
Previous MI, n (%)	41(25.9)	18(27.7)	0.79	48(25.4)	11(32.4)	0.4
Previous CVO, n (%)	8(5.1)	4(6.2)	0.74	9(4.8)	3(8.8)	0.4
Previous PCI, n (%)	47(29.7)	13(20)	0.14	53(28)	7(20.6)	0.37
CABG, n (%)	29(18.5)	13(20)	0.78	34(18)	8(23.5)	0.45
AF, n (%)	43(27.4)	13(20)	0.25	46(24.5)	10(29.4)	0.54
Anticoagulation, n (%)	53(33.8)	12(18.5)	0.023	57(30.2)	8(23.5)	0.43
Edwards Sapien XT	69(43.7)	44(67.7)	0.001	97(51.6)	16(45.7)	0.016
Edwards Sapien 3	22(13.9)	8(12.3)		24(12.8)	6(17.1)	
Evolvute R	54(34.2)	6(9.2)		55(29.3)	5(14.3)	
Portico	13(8.2)	7(10.8)		12(6.4)	8(22.9)	
Iliofemoral calcium Score [*]	1.24±0.74	1.69±1.03	0.002	1.29±0.78	1.82±1.1	0.011
Tortuosity Score [*]	1.34±0.97	1.38±0.97	0.73	1.4±0.98	1.1±0.87	0.07
Minimal IFLD, mm	7.66±1.1	6.32±0.99	<0.001	7.56±1.05	5.66±0.8	<0.001
SFAR	0.89±0.11	1.13±0.15	<0.001	0.91±0.13	1.21±0.17	<0.001
Sheath ≥18F, n (%)	67(42.7)	41(63.1)	0.006	91(48.1)	17(50)	0.84
Sheath outer diameter (mm)	6.71±0.67	7.02±0.71	0.002	6.82±0.71	6.68±0.61	0.29

Baseline clinical and Procedural characteristics of the Study Population

figure 2

Table 3 Baseline characteristics between patients with and without vascular complications

	Patients without VC (n=181)	Patients with VC (n=42)	P
Age (years)	79.2±7.69	77.98±7.2	0.35
Female, n (%)	83(45.9)	24(57.1)	0.19
logEuroScore	25.7±3.81	26.8±4.33	0.11
LVEF %	50.8±12.78	49.6±13.6	0.59
BMI, kg/m ²	26.3±3.5	26.1±3.9	0.81
Hypertension, n (%)	164(90.6)	36(85.7)	0.39
Diabetes, n (%)	40(22.1)	8(19)	0.67
CAD, n (%)	107(59.1)	24(57.1)	0.81
PAD, n (%)	32(17.7)	13(31)	0.053
GFR(ml/min/1.73 m ²)	69.43±25.3	68.3±27.9	0.8
Previous MI, n (%)	51(28.2)	8(19)	0.23
Previous CVO, n (%)	8(4.428.2)	4(9.5)	0.25
Previous PCI, n (%)	51(28.2)	9(21.4)	0.37
CABG, n (%)	33(18.2)	9(21.4)	0.63
AF, n (%)	46(25.6)	10(23.8)	0.82
Ineligibility (md-SFAR)	20(11)	14(33.3)	<0.001
Ineligibility (CG)	47(26)	18(42.9)	0.03
Calcification ≥moderate	18(9.9)	7(16.7)	0.27
Tortuosity ≥ moderate	73(81.2)	17(59.5)	0.98
Minimal IFLD, mm	7.31±1.16	6.87±1.41	0.035
Sheath ≥18F, n (%)	87(48)	21(50)	0.82
Sheath outer diameter (mm)	6.8±0.7	6.79±0.70	0.98
SFAR	0.95±0.15	1.02±0.21	0.009
SFAR>1.05	43(23.8)	16(38.1)	0.058
In-hospital death, n (%)	10(5.5)	5(11.9)	0.16
30 day mortality, n (%)	11(6.1)	8(19)	0.012

LVEF, left ventricular ejection fraction; BMI, body mass index; CAD, coronary artery disease; PAD, peripheral artery disease; MI, myocardial infarction; GFR, glomerular filtrate rate; MI, myocardial infarction; CVO, cerebrovascular occlusion; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; AF, atrial fibrillation; SFAR, sheath-to-femoral artery ratio; md-SFAR, manufacturer derived sheath-to-femoral artery ratio; CG, current-guidelines; IFLD, iliofemoral lumen diameter. VC, vascular complications

Baseline characteristics between patients with and without vascular complications

Table 1

Valve Name	Valve Size (mm)	Sheath Outer Diameter (mm)	Minimum vessel Diameter (mm)	Sheath Size (F)	Sheath outer diameter/minimum vessel diameter ratio
Sapien XT (Edwards Lifesciences)	23	6.7	6	16	1.12
	26	7.2	6.5	18	1.11
	29	8	7	20	1.14
Sapien 3 (Edwards Lifesciences)	20	6	5	14	1.2
	23	6	5.5	14	1.09
	26	6	5.5	14	1.09
	29	6.7	6	16	1.12
Evolute R (Medtronic)	23	6	5	14	1.2
	26	6	5	14	1.2
	29	6	5	14	1.2
	34	6.7	5.5	16	1.22
Portico (Abbot)	23	6	6	18	1
	25	6	6	18	1
	27	6.4	6.5	19	0.98
	29	6.4	6.5	19	0.98

Table 1 Outer Diameter of Sheaths and Minimum Vessel Diameters Required for The TAVI Systems

OP-005 ASSESSMENT OF MONOCYTE/HDL RATIO IN ASCENDING AORTIC DILATATION

Gonul Aciksari

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Objective: Ascending aortic dilatation (AAD) is a common and incidental condition, which can be fatal if unnoticed at an early stage. Previous studies have shown the relationship between the dilatation of the ascending aorta and oxidative stress. Monocytes and macrophages play the critical role in the inflammation process. These cells participate in the release of the proinflammatory cytokines in inflammation sites. High-density lipoprotein (HDL) cholesterol is a molecule with an anti-inflammatory effect. Monocyte count-to-high-density lipoprotein-cholesterol ratio, as an oxidative stress and inflammatory marker, has been shown as a novel useful indicator to predict cardiovascular risk and adverse outcome. The aim of this study was to investigate the relationship between Monocyte/ high density lipoprotein cholesterol (HDL-C) ratio and AAD.

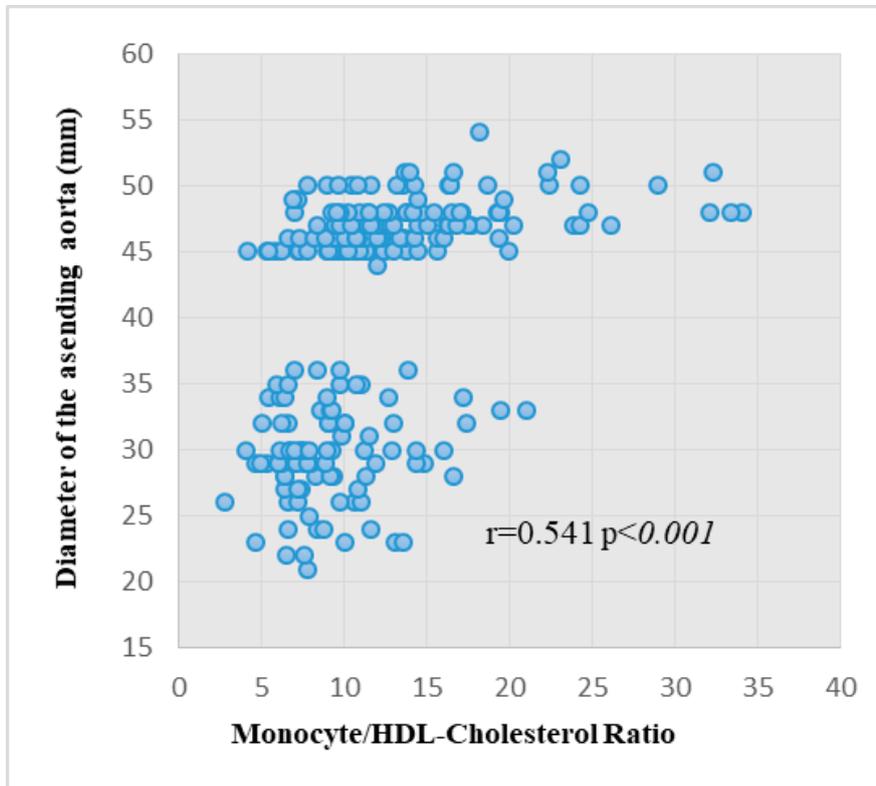
Methods: The study was designed as an observational cross-sectional controlled study. One hundred thirty-one consecutive patients with ADD and 89 consecutive controls with normal ascending aorta diameter were selected for the study by comprehensive transthoracic echocardiography (TTE). All routine biochemical and hematological parameters were measured on the day of blood draw and computed monocyte/HDL- cholesterol ratio. ANOVA, Mann-Whitney U test, Chi-square test, Pearson's correlation analysis, multivariate logistic regression analysis, and receiver-operator curve analysis were used for statistical analysis.

Results: Monocyte count-to-high-density lipoprotein-cholesterol ratio was significantly higher in patients with ascending aorta group than control group (13.7 ± 5.8 vs 9.2 ± 3.5 $p < 0.001$). There was a statistically significant positive correlation between the ascending aortic diameter and monocyte/HDL ratio ($r=0.541$, $p < 0.001$). According to multiple logistic regression analysis (ascending aortic diameter set as the dependent variable), age (OR: 1.07, 95% CI: 1.03 – 1.11, $p = 0.001$), Monocyte count-to-HDL-cholesterol ratio (OR: 1.22, 95% CI: 1.11 - 1.34, $p < 0.001$), high-sensitive C-reactive protein, (OR: 1.34, 95% CI: 1.10 – 1.69, $p = 0.016$) levels were found to be significantly and independently predictors of AAD.) Receiver operating characteristics (ROC) curve. ROC curve analysis revealed a correlation between Monocyte count-to-HDL-cholesterol ratio over 9.3 and ascending aorta dilation (81.7% specificity, 62.9% sensitivity, positive predictive value: 70%, negative predictive value: 76.4%) (area under the curve: 0.723, 95% CI: 0.652-0.794, $p < 0.001$).

Conclusion: We demonstrated for that combination of high circulating monocyte count and low HDL-cholesterol concentration is independently associated with size in ascending aortic dilatation patients. Monocyte count - high density lipoprotein ratio, as a marker of chronic low-grade inflammation and oxidative stress, may play a role in the pathogenesis of aneurysm of the ascending aorta in patients

Keywords: Ascending aortic dilatation, Monocyte count - high density lipoprotein ratio, İnflammation, oxidative stress

Figure 1. Correlation analysis showing a significant positive correlation between monocyte/HDL ratio and diameter of the Asending aorta.



The correlation between the diameter of the ascending aorta and monocyte/HDL ratio There was a significant positive correlation between the diameter of the ascending aorta size and monocyte/HDL ratio ($r=0.541$, $p < 0.001$).

Table 1. Baseline characteristics and laboratory parameters of the study population

	ADD Group (n =131)		Control Group (n =89)		P value
Age, years	58	± 8,8	53	± 10	<0,001
Gender (Male)	71	54,2%	37	41,6%	0,066
BMI, kg/m ²	28,5	± 5,1	28	± 3,7	0,984
Smoker, n, %	27	20,6%	18	20,2%	0,989
Alcohol, n, %	1	0,8%	1	1,1%	1,000
Hypertension, n, %	86	65,6%	38	42,7%	<0,000
Diabetes Mellitus, n, %	30	22,9%	24	27,0%	0,533
Hyperlipidemia, n, %	27	20,6%	24	27,0%	0,279
Family History of AAA	23	17,6%	19	21,3%	0,491
Glucose, mg/dl	101,0	± 18,8	98,7	± 17,8	0,276
Creatinine, mg/dl	0,83	± 1,6	0,80	± 0,15	0,683
TSH,mU/l	1,4	± 0,8	1,4	± 0,9	0,729
Uric acid, mg/dL	5,7	± 1,6	4,4	± 1,3	<0,001
AST, U/L	22	± 7,8	19,3	± 6,2	0,078
ALT, U/L	21,1	± 9,6	19,8	± 9,1	0,317
GGT, U/L	23,1	± 6,7	21,4	± 6,9	0,084
C-Reactive protein, mg/l	1,01	(0,20 -2,95)	0,20	(0,18 -0,91)	<0,001
Total cholesterol, mg/dl	197,0	± 41,1	214,6	± 44,6	0,030
LDL-C,mg/dl	121,6	± 33,8	134,3	± 39,4	0,010
HDL-C,mg/dl	43,2	± 9,5	51,6	± 12,6	<0,001
Triglycerides, mg/dl	150,7	± 71,0	138,6	± 61,3	0,164
White blood cell x10 ³ /µl	7,4	± 2,1	7,3	± 1,9	0,539
Hemoglobin, g/dL	13,5	± 1,4	13,7	± 3	0,516
Monocyte count x10 ³ /µl	0,53	± 0,14	0,45	± 0,13	<0,001
Lymphocyte x10 ³ /µl	2,3	± 0,7	2,4	± 0,9	0,530
Neutrophil x10 ³ /µl	4,8	± 1,8	4,3	± 2,2	0,052
Platelet x10 ³ /µl	247	± 55,8	251	± 63,5	0,685
NLR	2,4	± 1,4	2,0	± 1,1	0,092
Monocyte/HDL-Cholesterol	13,7	± 3,8	9,2	± 3,3	<0,001
Ascending aortic diameter (mm)	45,2	± 2,2	29,6	± 3,7	<0,001
ACEI, n %	27	20,6%	20	22,4%	0,741
ARB, n %	39	29,8%	13	4,6%	0,009
CCB, n %	33	25,2%	14	15,7%	0,093
Beta-blocker, n, %	74	56,5%	30	33,7%	0,001
ASA, n, %	27	20,6%	15	16,9%	0,487
Statin, n, %	22	16,8%	12	13,5%	0,505
Oral Antidiabetic, n %	18	13,7%	19	21,3%	0,139

ACEI, angiotensin converting enzyme inhibitor; ADD, ascending aortic dilatation; ALT, alanine aminotransferase; ARB, angiotensin receptor blocker; ASA, acetylsalicylic acid; AST, aspartate aminotransferase; BMI, body mass index; GGT, γ glutamyl transferase; NLR, neutrophil-lymphocyte ratio; TG, triglyceride.; TC, total cholesterol; TSH, thyroid stimulating hormone.

OP-006 COARCTATION OF THE AORTA AND RELAPSING SUPRAVALVULAR AORTIC STENOSIS IN CONGENITAL CUTIS LAXA SYNDROME

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Introduction: Cutis laxa is a very rare connective tissue disorder. Loose skin and accordingly prematurely aged appearance are the most common features of this disease. We report a 27-year-old female patient, who was born with congenital cutis laxa and had been operated for supra- valvular aortic stenosis presenting with symptomatic coarctation of the aorta and recurrence of supra- valvular aortic stenosis.

Case Presentation: The 27-year-old patient was admitted to our hospital with persistent hypertension and claudication. Her skin was redundant, loose, and its recovery was very slow when hauled. Facial dysmorphism was prediagnosed to be due to cutis laxa. Therefore, a skin biopsy and genetic counselling were performed and confirmed the diagnosis. The patient was then operated for supra- valvular aortic membrane in 2003 after which she was lost to follow-up. During the physical examination in our center, her blood pressure was 160/90mmHg from the right arm and 100/60mmHg from the right leg. We suspected aortic coarctation and performed a thoracic computed tomography (CT). The CT demonstrated a significant coarctation of the aorta just below the left subclavian artery in descending aorta as well as hypoplastic abdominal aorta measured as 1.2cm. Suprasternal echocardiography demonstrated 70mmHg maximum gradient at the level of coarctation. The transthoracic echocardiography also showed recurrence of the supra- valvular membrane with 20 mmHg gradient.

Discussion: Cutis Laxa (CL) is known as a genetic disorder which occurs from elastic fibers mutations but it can also be acquired too. Inherited forms include autosomal dominant CL (ADCL) and autosomal recessive CL forms (ARCL-I, -IIA, and -IIB). In addition, CL may be observed along with geroderma osteodysplasticum and arterial tortuosity syndrome.

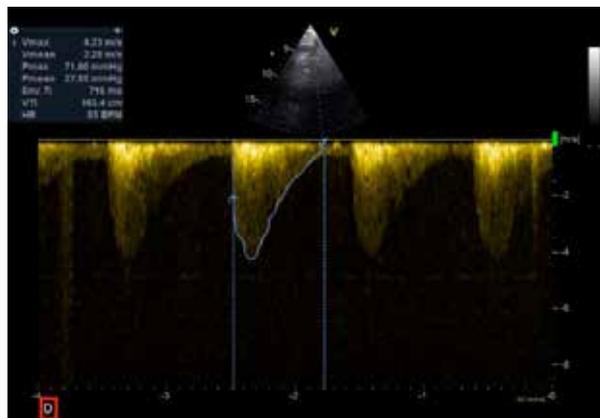
Arterial tortuosity syndrome (ATS) is a rare autosomal recessive connective tissue disease characterized by tortuosity and elongation of the large- sized and medium-sized arteries, and was first described in 1967 by Ertugrul et al. SLC2A10 gene mutation has been well established in this syndrome. Other cardiovascular problems are the arterial and pulmonary valve stenosis, and vasomotor instability.

There are some case reports about the coarctation of the aorta in ATS. Although cutis laxa can occur with ATS, the most characteristic complications are tortuosity and elongation of the large- sized and medium-sized arteries in ATS. In our case report neither elongation nor tortuosity was demonstrated in CT. There is only one case that has been reported with supra- valvular aortic stenosis in cutis laxa. Our case has relapsing supra- valvular aortic stenosis and has a complex form of aortic coarctation.

To our knowledge, this is the first case report with congenital cutis laxa presented with the coarctation of the aorta and relapsing supra- valvular aortic stenosis. Because of these complications, we think our patient has ARCL-I with FBLN-4 and FBLN-5 combined mutations.

Keywords: coarctation of aorta, supra- valvular aortic stenosis, cutis laxa

Coarctation of aorta



Facial appearance of the 27-year-old patient.



OP-007 ASSOCIATION BETWEEN PSYCHIATRIC SYMPTOMS AND PAIN SEVERITY IN INDIVIDUALS WHO COMPLAIN OF NONCARDIAC CHEST PAIN

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¹Kars Harakani Devlet Hastanesi

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Objective: Complaints of chest pain are common in the society. Psychiatric symptoms have been investigated in patients complaining of noncardiac chest pain (NCCP), however Type D personality has not been investigated. Therefore, this study was planned to examine the association between psychiatric symptoms and the severity of pain felt with these symptoms in people who complain of chest pain.

Material-Methods: The patient group with 100 individuals diagnosed with NCCP, and the control group with 100 healthy individuals were formed. Socio-demographic Data Form, Beck Depression Inventory, Childhood Trauma Questionnaire, Type D Personality Scale, Somatosensory Amplification Scale, and Toronto Alexithymia scale were applied to the subjects.

Results: It was found that depression, alexithymia, somatosensory amplification, Type D Personality and childhood traumatic experiences scale scores were significantly higher in individuals in the patient group compared to the control group, and that Type D personality traits were associated with pain severity.

Conclusion: Psychiatric symptoms are common in patients with NCCP. We are of the opinion that detection and treatment of psychiatric disorders might reduce recurrent hospital admissions.

Keywords: Non-cardiac chest pain, depression, alexithymia, mental disorders, Type D Personality

OP-008 EVALUATION OF THE CULPRIT LESION CHANGES OVER TIME IN PATIENTS PRESENTING WITH ACUTE MYOCARDIAL INFARCTION AND PRIOR CORONARY ARTERY BYPASS GRAFTING

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Aim: Management of patients suffering from acute myocardial infarction (AMI) with previous CABG is complicated. Lesions in venous grafts tend to be more thrombogenic and progression of atherosclerosis in left internal mamarian artery (LIMA) is slower than native coronary artery. The present study aimed to analyze the culprit lesions in native coronary artery and grafts in patients with AMI and prior CABG.

Methods: The study included a total of 195 prior CABG patients diagnosed with AMI. Patients were classified into two groups including non-ST segment elevation MI (NSTEMI)(n=154) and ST segment elevation MI (STEMI) (n=41). We investigated demographic and clinical characteristics, patency rates of the arterial and venous grafts according to graft age, distribution of culprit vessels and lesion localizations.

Results: Demographic and clinical characteristics were similar for both groups. Patency rates of the arterial grafts were significantly better than the venous grafts at all time-intervals (p<0.05). Responsible lesions in both NSTEMI and STEMI groups did not differ as being either native or graft (30 vs. 96 native vessels in STEMI and NSTEMI groups, respectively; 11 vs. 58 grafts in STEMI and NSTEMI groups, respectively; p=0.19). Culprit lesions in LIMA were all located at the distal anastomosis site whereas more frequently in the mid portion of the venous grafts (79.2% in NSTEMI; 66.7% STEMI; p=0.019). Patency rate of the arterial grafts were significantly better than the venous grafts at all time-intervals (p=0.03, 6 month-5 year; p=0.01, 5-10 years, p=0.006, >10 years). During all time intervals culprit lesion locations being at either grafts or native vessels did no differ between STEMI and NSTEMI patients.

Conclusions: Culprit lesion locations as occurring in either graft or native vessels were similar in CABG patients presenting with NSTEMI and STEMI. Venous grafts lesions tended to locate at mid portion for both of NSTEMI and STEMI patients. Patency of arterial grafts were better than venous grafts during all time intervals.

Keywords: coronary artery disease, bypass, grafts, acute myocardial infarction

Figure 1

Table 1. Demographic and clinical characteristics of the patients

Characteristics	NSTEMI (n=154)	STEMI (n=41)	P
Age, year	62.7±9.7	62.6±10.8	0.932
Gender, (%)			
Male	101 (65.6)	28 (68.3)	0.86
Female	53 (34.4)	13 (31.7)	
Hypertension, (%)	106 (68.8)	26 (63.4)	0.464
Diabetes, (%)	74 (48.0)	23 (56.1)	0.288
Smoking, (%)	37 (24.0)	9 (21.9)	0.867
Dyslipidemia, (%)	84 (54.5)	25 (60.9)	0.362
Family history, (%)	80 (51.9)	18 (45.2)	0.268
Used graft, (%)			
LIMA	116 (75.3)	29 (70.7)	0.549
Ao-LAD	38 (24.7)	12 (29.3)	0.415
Ao-OM	97 (54.2)	28 (63.6)	0.427
Ao-RCA	72 (40.2)	10 (23.8)	0.013
Ao-D	10 (5.6)	4 (9.6)	0.307
Graft age (years)	6.3±3.8	6.1±3.2	0.775
Mean number of grafts	2.1±0.5	2.0±0.4	0.376
LVEF (%)	43.7±10.4	43.6±8.9	0.971

Abbreviations, LVEF, left ventricular ejection fraction

Figure 2

Table 2: Patency rates of the arterial and venous grafts by graft age

Graft age	Arterial Graft (n=145)	Venous Graft (n=271)	P
6 month -5 years, (%)			
Total graft	58 (100)	100(100)	
Patent graft	56 (96.5)	67 (77.8)	0.03
5-10 years, (%)			
Total graft	69(100)	140(100)	
Patent graft	66 (95.6)	91 (65)	0.01
>10 years, (%)			
Total graft	18(100)	31(100)	
Patent graft	17 (94.4)	12 (38.7)	0.006

OP-009 ASSESSMENT OF POSSIBLE PREDICTORS OF ACCESS SITE COMPLICATIONS IN NON-STEMI PATIENTS TREATED VIA FEMORAL ARTERIAL

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BACKGROUND AND Aim: Femoral artery approach is widely used for coronary angiography (CAG) and percutaneous coronary interventions (PCI). Access site complications (ASC) are reported to occur more frequently in femoral approach compared to radial approach. However, the predictors of ASC in femoral approach have not been well-defined especially in patients with non-STEMI.

Methods: In this single center analysis, we have investigated ASC rates and possible correlates in 1532 non-STEMI patients treated with CAG and PCI. Clinical, demographic properties and anthropometric measures were collected from medical records

Results: Access site complications developed in 119 patients (7.7%). In 84 patients hematoma formation, in 32 patients pseudoaneurysm formation, in 2 patients arterial rupture and in 1 patients retroperitoneal hemorrhage developed. ASC occurred more often in women but the remaining demographic, clinical properties and laboratory parameters were similar between two groups (Table 1). In addition, body mass index, waist and hip circumference and waist/hip ratio were not different between the groups. In subgroup analysis of male and female subjects, anthropometric parameters were not different. Only 11 patients had undergone surgical repair and the remaining patients were treated conservatively. None of the patients died due to vascular complication. Hospitalization period was longer in patients with ASC

Conclusions: In patients with non-STEMI who had undergone CAG and PCI via femoral approach we did not find a predictor of ASC except female gender. In 7.7% of the patients an ASC was diagnosed and none of the cases died secondary to ASCs.

Keywords: access, approach, complication, femoral, site

Baseline demographic, clinical, interventional, laboratory characteristics and medical therapy of the study patients

Variables	ASC (+) (n=119)	ASC (-) (n=1233)	p value
Age	60.6 ± 11.6	60.1 ± 11.3	0.590
Gender (Female) n(%)	44 (%36)	346 (%28)	0.041
Hypertension n(%)	69 (%57)	822 (%66)	0.517
Diabetes mellitus n(%)	49 (%41)	474 (%38)	0.558
History of CAG n(%)	32 (%26)	424 (%34)	0.098
History of AF n (%)	4 (%3)	31 (%2)	0.578
Weight (kg)	81.3 ± 15.2	81.6 ± 14.6	0.830
Height (cm)	165 ± 8.6	166.7 ± 9.1	0.320
Waist circumference (cm)	99.5 ± 12.4	99.4 ± 11.5	0.930
Hip circumference (cm)	100.1 ± 9.4	98.9 ± 10.1	0.220
BMI (kg / m ²)	29.6 ± 5.5	29.4 ± 5.0	0.610
SBP when Procedure Started (mm/Hg)	148 ± 25	147 ± 29	0.671
DBP when Procedure Started (mm/Hg)	84 ± 14	83 ± 16	0.887
Procedure Performed PCI n(%)	61 (%51)	679 (%55)	0.425
Critical Vessels Number (per patient)	1.31 ± 0.96	1.43 ± 1.02	0.290
Hospitalization Period (day)	7.4 ± 2.4	4.6 ± 1.4	< 0.01
Previous Medical Therapy			
Warfarin, n%	2 (%1)	10 (%0.8)	0.231
NOAC, n%	2 (%1)	17 (%1)	0.651
Antiplatelet agents, n %	28 (%23)	445 (%36)	0.095
Admission Laboratory Parameters			
Glucose (mg/dL)	155 ± 85	145 ± 78	0.220
Creatine (mg/dL)	0.93 ± 0.33	1.01 ± 0.41	0.230
Hemoglobin (g/dL)	13.8 ± 1.9	13.9 ± 3.1	0.690
INR	1.20 ± 0.31	1.13 ± 0.33	0.170
PLT (103/μL)	236 ± 89	237 ± 66.7	0.990

ASC: Acces site complication, AF: Atrial fibrillation, BMI: Body mass index, CAG:coronary angiography, DBP: Diastolic blood pressure, INR: International normalized ratio, NOAC: New oral anticoagulants, PCI: Percutan coronary intervention, SBP: Systolic blood pressure

OP-010 DIGIT RATIO (2D:4D) IN TURKISH MALE PATIENTS WITH MYOCARDIAL INFARCTION

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Bakirkoy Dr Sadi konuk Training and Research hospital

Background: Digit ratio is the ratio of the length of the 2nd (index finger) and 4th (ring finger) digit (2D:4D)

It is thought to be negatively correlated with prenatal testosterone level.

The relationship between the 2D:4D and various physiological, behavioral, and pathological conditions has been demonstrated in many studies.

2D:4D in humans also vary between ethnic groups. A previous study showed that the 2D:4D was lower in Afro-Caribbean Jamaicans than Caucasian Uygurs.

Traditional coronary artery disease (CAD) risk factors are well-defined. However, The effect of different risk factors such as genetic predisposition or inflammation are still being evaluated .

Myocardial infarction (MI) prevalence is higher in men than women in the pre-menopausal period, yet the difference decreases at later ages .

It is also known that low endogenous testosterone levels are related with MI in men. Hence, High 2D:4D ratio may be predisposing to history of MI at an early age in males.

Objective: Although Small number of studies have evaluated the association between CAD and 2D:4D, the link between 2D:4D and age or MI localization has not yet been reported in the literature. Therefore, The aim of the current study was to compare 2D:4D ratios according to age, other CAD risk factors and MI localization in Turkish male patients with a history of MI.

Materials and Method: Study population: This was a cross-sectional, single-center, retrospective study. A total of 140 male patients with a history of MI were consecutively included. All of the patients were from Turkish ethnic group.

Data regarding demographic characteristics of patients were obtained from hospital registries.

Measurement of finger lengths: The length of index and ring fingers of both hands were measured using a ruler. Measurement was performed from the palmar surface, starting from the proximal palm's basal crease to the tip of the finger. 2D:4D ratio was calculated by dividing the length of the 2nd finger of one hand by the length of the 4th finger of the same hand. An independent examiner blinded to the study measured finger lengths, 2D:4D ratio was calculated.

Results: The mean age of the group was 57 years old and 13% of them were between the age of 30 to 45 year old.

Mean right hand 2D:4D was 0.98 mm and left hand was 0.96 mm.

The right versus left 2D:4D was not statistically different according to any age groups, CAD risk factors, nor MI localization (p> 0.05).

Discussion: In this study, we did not find a statistically significant difference in 2D:4D of both hands according to age groups, presence of diabetes, HT, smoking, family history, or localization of MI. To the best of our knowledge, our study was the first to evaluate the association between 2D:4D ratio and MI, in respect to different age groups, CAD risk factors and MI localization.

In previous studies, 2D:4D ratio was reported to be a predictor for CAD.

High testosterone levels are assumed to be protective against CAD and myocardial infarction in males.

Wang et al. Showed that 2D:4D ratio in Chinese women with CAD was lower compared to control subjects.

Viveka et al ; demonstrated a positive association between 2D:4D and diagonal ear lobe crease which is also considered as an indicator of atherosclerosis.

Ozdogmus et al. found on the autopsy of 100 male patients that the right hand 2D:4D was associated with atherosclerotic plaque burden in right coronary arteries.

Conclusion: 2D:4D of both hands are similar in Turkish male patients with a history of MI. 2D:4D is not associated with CV risk factors, and MI localization.

Right and left hand 2D:4D according to age

Age groups	Right 2D:4D	Left 2D:4D	p value
30-45 years (n=19)	0,99±0,04	0,95±0,05	0,580
46-55 years (n=45)	0,98±0,04	0,96±0,05	
56-65 years (n=40)	0,97±0,06	0,95±0,04	
>65 years (n=36)	0,98±0,05	0,96±0,04	

OP-011 THE TIME TO RETURN TO WORK AND TIME OF ADAPTATION TO SOCIAL LIFE OF PATIENTS TO WHOM PRIMARY OR ELECTIVE CORONARY ANGIOGRAPHY WERE APPLIED

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1.ABSTRACT

Coronary artery disease is one of the most common cause of death in the World due to factors such as the increase in mean age of communities and the bad eating habits brought by industrialization. After coronary angiography, invasive, surgery or medical treatment decisions are given for majority of patients. In this study we aimed to calculate the time to return to work and daily life after coronary angiography. The country's economies are affected adversely by both treatment costs and labor force loss because of the disease.

We aimed to determine the time to return to the work and daily life in patients with a history of DM and HT, in elderly, in patients who were decided to taking in PCI or CABG, in patients with prolonged hospitalization, in patients with angina and dispnea.

Key words: Coronary artery disease, time of returning the work, cardiac rehabilitation

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INTRODUCTION: According to data of WHO, it is estimated that Cardiovascular diseases will be the first reason for mortality and morbidity all over the World in 2020 [1]. Removing risk factors is very important as well as early diagnosis and treatment of cardiovascular disease in industrial nations. Cardiovascular diseases are very important for the economy of these countries because diagnosis and treatment of the disease is very expensive [2].

The factors affecting the time of returning to job are treatment time, complications of contrast agent and vascular access site, the cause of coronary angiography, hospitalization time, age, gender, comorbid diseases, drugs and sociocultural characteristics. While some of the studies about returning job after coronary angiography [3] say that age and gender can affect the results, some of them obtained results as nonsignificant [4]. In addition, it was found that the time to return to work was a mean of 4 weeks, while reduced ejection fraction (EF) significantly affected the time to return to work, whereas factors such as elective or primary angiograph were not significant. Atherosclerosis begins in the early decades, then results in coronary artery disease (CAD) which is one of the most common causes of death in the industrialized population [5]. According to some studies the frequency of angina increases proportionally with age in both genders and while prevalence of angina in women is 5-7% between ages 45-64 years and 10-12% between 65-84 years, in men it is 4-7% between ages 45-64 and 12-14% between 65-84 years [6]. Symptoms are usually short-term and disappear with rest [7]. Considering that a productive economy depends on the labor force, the time to return to work is very important. Our aim in this study is to help to determine the optimal approaches that will cause the least labor loss by determining the average return to work process and revealing the factors affecting the return to work process.

MATERIALS AND METHODS: Our study was conducted between March 2018 and October 2018 by applying questionnaire to the patients who had primary or elective coronary angiography decision in the cardiology clinic of Firat University Medical Faculty Hospital, and by contacting the contact numbers given at the third month, asking the time to return to work and social life. The patients were grouped as STEMI, NSTEMI and Stable angina pectoris (elective). Angiographic results of the patients were evaluated by two experienced physicians. Patients were evaluated according to risk factors and general demographic characteristics of coronary artery disease. Patients' age, sex, diabetes mellitus (DM), hypertension (HT), smoking, alcohol, cerebrovascular disease (SVH), chronic renal failure (CRF), ischemic heart disease (HRC), heart failure (HF), pace history (DEVICE), angina, dyspnea, dizziness symptoms, occupation, education status were asked after coronary angiography (CAG). Total hospitalization time of the patients was recorded and Echocardiography (ECHO) was taken and Ejection fraction (EF), valve pathology, pulmonary hypertension and routine diameter measurements were taken.

Exclusion criteria: - Patients who have not undergone to coronary angiography and who was decided to followed up medically

- Patients without contact numbers.

Statistical analysis

Data were analyzed with Windows SPSS software version 22.0. Descriptive statistics were used for values such as frequency, mean, standard deviation, median, and range. Chi-Square test was used to compare the qualitative variables between the independent groups of more than three groups and the expected values were smaller than five in the evaluation of the study groups according to different variables. The sample T-test was used to determine the significance of the difference between two means in two independent groups. ANOVA was used to compare more than three independent groups. All statistical analyses were performed using SPSS software. To be statistically significant, the p-value was accepted as less than 0.05 for all data.

RESULTS: When we look the distribution of the age the youngest patient was 23 years and the oldest was 94 years old. The mean age of the patients was 61 ± 12.7 years. The distribution of age is not normal.(Figure 1)

In this study, Patients are divided into three groups according to their diagnosis.(Figure 2)

We see that adaptation to daily life and work takes place faster in younger patients.(Table 2)

When the demographic characteristics of the patients were considered, it was observed that individuals with a history of HT and IHD had a later return to work.(Table 3)

There was no correlation between the severity of admission and return to work or social life. The data we obtained were that the patients who underwent elective CAG made a business plan so that they could spend time for rest, while the patients who had STEMI could not comply with the recommended period of rest due to the lack of preparation due to the emergent emergency and the low socioeconomic level in which they were present. (Table 4)

It was seen that invasive procedures such as PCI or decision of coronary artery bypass surgery caused prolongation of patients' return to work and the earliest return to work was seen in NKA group.(Table5)

After CAG, return to work was found to be late in patients with angina and dyspnea, but was not associated with dizziness.(Table 6)

There was no significant relationship between retirement and housewives' time to return to social life and their current status, on the other hand, there was no significant relationship between working as a civil servant or worker and returning to work.(Table 7)

The p-value of the relationship between education level and time to return to work is 0.202 and is not statistically significant. There was no significant relationship between education level and time to return to work.(Table 8)

Return to work is also prolonged in patients with extended hospital stays.(Figure 3)

TABLES

TABLE 1. Demographic and clinical characteristics of the general population

Demographic and clinical features	N	Percent %
DM	108	31,0
HT	169	48,6
Cigarette	141	40,5
Alcohol	11	3,2
CVD	22	6,3
CRF	39	11,2
İHD	149	42,8
PACE	6	1,7
HF	42	12,1
STEMI	70	20,1
NSTEMI	55	15,8
ELECTIVE CAG	223	64,1

TABLE 2. Comparison of demographic and clinical characteristics according to the presence of coronary artery disease.

Age	≤40	41-59	≥ 60	P value
The time to return to work	6,8	11,6	12,7	0,021

TABLE 3. Relationship between demographic characteristics and return to work

Demographic and clinical features	The time to return to work	P value
Gender (F/M)	11,2/13,3	0,218
HT	14,7	<0,01
DM	13,7	0,140
Cigarette	11,4	0, 594
İKH	14,3	0, 012
Alcohol	6,1	0, 195
CRF	10,9	0, 425
CVD	17,3	0, 091

TABLE 4. Relationship between application diagnoses and return to work and daily life

	Stemi	Nstemi	Elevtive Cag	P value
The time to return to work	10,3	12,6	15,2	0,597

TABLE 5. Correlation between decision-making after coronary angiography and return to work and daily life

	PCI	MTD	NCA	BY-PASS	P
Number of patients	176	74	88	10	-
The time to return to work	13,9	10,4	4,0	54,2	<0,01

TABLE 6. Relationship between ongoing symptoms and return to work and daily life after CAG

Symptoms after coronary angiography	The time to return to work	P value
Angina	18,5	<0,01
Dyspnoea	20,5	<0,01
Dizziness	8,9	0,378

TABLE 7. Relationship between patients' occupational status and return to work and daily life

Job	Number of patients	The time to return to work	p
Retired	135	11,2	0,506
Housewife	51	12,9	0,668
Officer	31	7	0,055
Worker	131	13,7	0,210

TABLE 8. Relationship between education level and return to work and daily life

Level of education	The time to return to work	P
Illiterate	14,5	0,180 0,180
Only literate	13,6	0,505
Primary school	13,1	0,306
Middle School	10,3	0,417
High school	8,7	0,079
High education	9,1	0,303

TABLE 9. Relationship between ejection fraction and return to work and daily life

	EF ≤%40	%41-49	≥%50	P value
The time to return to work	12,3	11,5	13,2	0,884

FIGURES

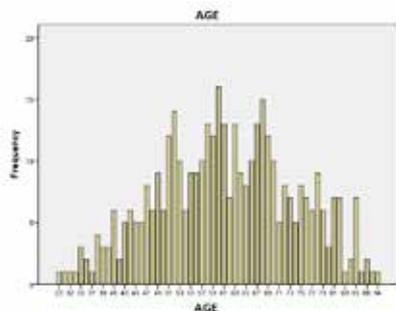


Figure 1. Age distribution of patients

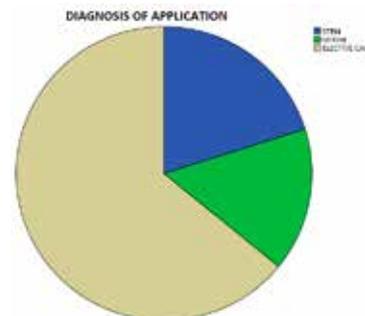


Figure 2. Distribution of patients according to their diagnosis

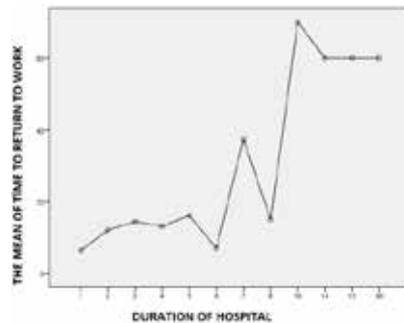


Figure 3. Distribution of return-to-work time according to a length of hospital stay (The p-value of the relationship between the two factors is <0.01 and is statistically significant).

DISCUSSION: One of the most objective indicator of coronary artery disease treatment is that patients can return to work and achieve their previous living standards. Failure of patients to gain previous activity capabilities will cause loss of self-confidence and loss of financial gain. The aorta-coronary by-pass surgery (CABG), percutaneous coronary intervention (PCI) and percutaneous coronary angioplasty (PTCA), which are used for the treatment of coronary artery disease, are expensive procedures and with these the emergence of the factors that prevent the return of the patients to the work creates economic high costs. Preservation of left ventricular functions and absence of effort angina are important factors in patients' return to work and their adaptation to social life in patients presenting with the acute coronary syndrome. After an uncomplicated myocardial infarction, return to work was 2 weeks on average in light office conditions, 3 weeks for arm workers, and 6 weeks for heavy-duty workers. Although a small number of patients have been reported to return to work earlier, the average time to return to work after acute myocardial infarction worldwide is 50 days. However, it should not be ignored that there are serious differences between countries. Important factors in returning patients to work include age, gender, educational status, occupation, previous myocardial infarction (MI), MI width, residual angina, low left ventricular functions, and low exercise capacity. Other factors such as psychological status, the presence of health insurance, unemployment benefits and working conditions are also important. Compared to other factors, medical factors seem to play a small role in returning to work. It is also important to identify the factors that may cause the recurrence of cardiac events against the economic benefit of early return to work.

In this study, we investigated the factors affecting the return of 348 patients to work and previous standards of life, who were admitted to the cardiology clinic for ACS or stable angina and ultimately underwent primary or elective angiography. Similar studies have been done before and average values have been revealed. However, different results have emerged as a result of the researches performed by each country and clinic. In our study, unlike other studies, active employees were asked to return to work, while individuals who were not actively involved in business life such as housewives or retirees were asked to return to their previous activities.

Herlitz et al. [8] in a prospective cohort study of myocardial infarction under the age of 65 full-time or part-time working patients were studied and a total of 921 patients were taken as a mean age 72 (16-98) and in total 49% of patients returned to work after MI. After MI, 37% of full-time workers returned to work, while only 12% of part-time workers returned to work. It is stated that advanced age and large infarcts adversely affect return to work. In our study, the total number of patients with AMI was found to be 125 patients. In our study, it was seen that all pre-CAG workers returned to work and therefore it was thought that the time to return to work was more important for our population and the periods were compared. In all groups, it was revealed that the time to return to work was later in advanced age.

In the studies of Maeland et al. [9,10], 249 patients under the age of 67 were followed up for 6 months after acute myocardial infarction, and 2% of the patients had this period on leave due to illness. A total of 72.7% of the patients returned to work during this period. As a result of the analysis, it was stated that return to work was adversely affected in advanced age, low education level, rural people, high work stress, anxiety, depression and low self-confidence. In our study, it was observed that the level of education had no significant effect on the time to return to work. The insufficient number of patients at different educational levels and the resulting statistical insignificance explain this weak relationship.

Soejima et al. [11] performed a study about return to work after myocardial infarction in, full-time employment male Japanese patients who had myocardial infarction for the first time. Patients were reached by mail or telephone at the 8th month and 83% of the patients returned to work within 8 months. In total, 134 people were included in the study and the mean age was calculated as 54.3. The most important factors that negatively affect the return to work were depression and anxiety about one's illness.

In our study, patients were not asked questions about their commitment to work, their emotions, anxiety caused by the disease, and income levels. Future studies were considered to be more powerful with these questions.

Although acute myocardial infarction recommendations, treatment techniques and medical therapies improved the patient's clinical status, it was not found to be effective in determining the time to return to work and daily life. It is thought that medical, psychological and socioeconomic factors should be evaluated in a multidisciplinary manner in return to work and daily life. Although cardiac rehabilitation programs shorten the time to return to work, it is seen that there is no clear benefit on the return to work period considering the transportation cost and loss of time for the patients living in rural areas.

It was found that 59% and 100% of the patients who had been working before coronary artery bypass surgery(CABG) had returned to work after surgery [12,13]. However, the average rate of patients returning to work was shown to be 75%.

In the prospective PERISCOP study [14], 530 patients underwent CABG and were followed-up for a mean of 12 months and 67.55% returned to work. The average time to return to work was shown to be 3.2 ± 2.2 months and was generally said to be 3 months. At the same time, it was shown that the number of coronary arteries affected by the disease did not affect return to work, total revascularization was performed in 78.2% of those who returned to work and 75% of those who could not return to work, and the relationship between the two was not statistically significant and there was no significant relationship between the return to work and detected arrhythmia, while patients with advanced age, dyspnea complaints and short

exercise duration had negative effects on return to work.

A total of 213 patients were included in the return to work after CABG study conducted by Speziale et al. The mean follow-up was 38 months and 78.7% of the patients returned to work ^[15]. 78.3% of patients under 50 returned to work, while only 60.7% of patients over 50 returned to work. At the same time, it was stated that the return to work of patients with the low socioeconomic level was negatively affected. Advanced age and low socioeconomic status have been shown to adversely affect return to work.

In our study, the average time to return to work was 54.2 days in patients with CABG decision, and similar results were obtained with other studies. Also, the ongoing dyspnea complaints after PCI and CABG decision were questioned and the meantime to return to work was shown to be 20.5 days in these patients, and the relationship between dyspnea and return to work was statistically significant. It has been observed that persistent dyspnea and advanced age adversely affect the time of return to work and cause later returns to work.

Some of the return to work studies after coronary artery by-pass surgery (CABG) indicated that DM was an important factor ^[16] and the results were insignificant in some studies ^[17]. It has also been shown in some studies that the history of acute myocardial infarction is not an effective factor at the time of return to work after CABG ^[14]. A study by Mark et al. revealed that peripheral arterial disease adversely affects return to work after CABG ^[18].

DM did not have a clear effect on the time of return to work in our study. No significant differences were observed in the time of return to work because no complications related to DM were observed in our patients.

136 patients who underwent CABG were included in a study by Boudrez and De Backer ^[19]. 81% of the patients followed up for 12 months returned to work. The mean ejection fraction (EF) of the patients returning to work was $71 \pm 12\%$, whereas the mean EF of those who could not return to work was $62 \pm 18\%$ and the relationship between EF and return to work was statistically significant. As a result, preserved left ventricular function is a positive factor that facilitates return to work.

In our study, unlike other studies, it was shown that the ejection fraction did not have a significant effect on the time of return to work but only sustained dyspnea harmed return to work. It has been shown that if the clinical status of patients with low EF is deteriorated, the return to work will be adversely affected and if there is no symptom and clinical pathology, there is no significant difference between them and other patients. (Table 9)

In the return to work after CABG study by Skinner et al. 353 patients were included in the study, followed up for 12 months, and 84% of the patients returned to work ^[20]. Angina has been shown to be a factor that adversely affects return to work.

In our study, the meantime to return to work was 18.5 days in patients with persistent angina after PCI (PCI and CABG) and the relationship between angina and return to work was statistically significant. Ongoing angina has been shown to adversely affect patients' return to work, leading to later returns to work.

In the randomized intervention treatment of angina (RITA) study, the return rates of CABG and PCI patients were compared ^[21]. In Bypass Angioplasty Revascularization Investigation (BARI) study ^[22], the return rates of CABG and PCI patients were compared and similar results were found with the RITA study.

In our study, it was seen that the time to return to work of CABG patients was similar to other studies, while earlier returns in the PCI arm took place, shortening of the processing time with improved stent and balloon technology, and the fact that revascularization was performed under optimal conditions with an experienced team contributed greatly to this process. On the other hand, it is an undeniable reality that the patients kept this period shorter despite the physician's recommendations due to economic reasons in our country conditions. In our study, all patients were divided into groups as retired, housewives, civil servants and workers. There was no significant relationship between retiredes and housewives' time to return to social life and their current status however, there was no significant relationship between working as a civil servant or a worker and the time to return to work. It was shown that the data obtained from other studies were similar.

CONCLUSION: This study is the first in Turkey about the time to return to work and return to daily life after the KAG. We should say in particular that there is no standardization regarding the time of return to work in our country. Based on the data obtained from patients with acute coronary syndrome or stable angina who underwent CAG; A total of 162 patients, 31 of whom were civil servants and 131 were workers, were found to have returned to their jobs. On the other hand, the time to return to work was longer in CABG and PCI arm depending on the procedure. It was observed that the education level did not affect the time of return to work. It was found that the time to return to work was later in patients with a history of HT and IHD. No significant relationship was found between EF and time to return to work. It was found that the symptoms that affect the comfort of life such as dyspnea caused by cardiac dysfunction are more effective on the return to work and prolong the duration rather than the quantitative EF value of the patients. It was also found that prolonged hospital stay, regardless of the cause, caused prolonged return to work and previous daily life. It was also observed that angina and dyspnea, which persist after CAG, negatively affected the time to return to work. It was seen that the time to return to work was later in older patients and younger patients were adapted to life earlier after CAG. In our country, it was seen that the return of the patients to work was much earlier than the other countries after AMI, it was predicted that the patient could not be provided with sufficient information and that the patient had underlying financial concerns.

DECLARATION OF INTERESTS

The authors declare no conflict of interests.

OP-012 VALUE OF AGE, CREATININE, AND EJECTION FRACTION (ACEF SCORE) FOR CLINICAL PROGNOSIS OF YOUNG PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION UNDER THE AGE OF 40

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Objective: The age, creatinine, and ejection fraction (ACEF) score (age/left ventricular ejection fraction+1 if creatinine >2.0 mg/dL) has been established as a useful predictor of clinical outcomes in patients undergoing elective coronary artery bypass surgery. Subsequently, multiple clinical trials have indicated that the ACEF score is also evaluated in many different groups such as acute coronary syndrome, elective percutaneous coronary intervention (PCI) and in elderly patients. Nevertheless, whether ACEF score can be used to predict the outcomes in young patients diagnosed with acute STEMI has not been reported. In this study we aimed to investigate the predictive value of the ACEF score for in-hospital and one year clinical outcomes in patients aged <40 years diagnosed with acute ST segment elevation myocardial infarction (STEMI).

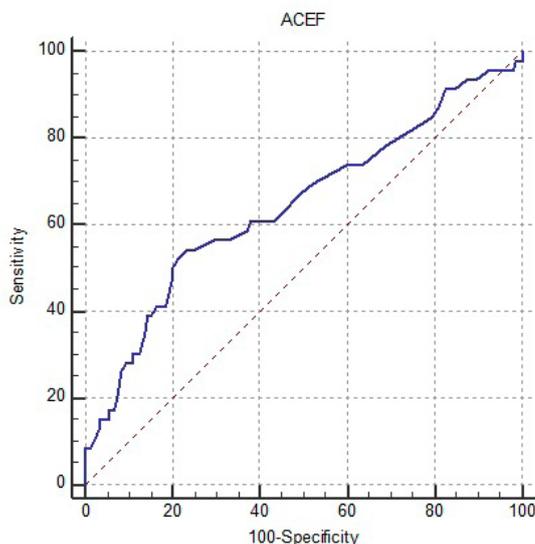
METHODS: This study has been performed retrospectively and observationally in 2 centers that provide 24/7 primary percutaneous coronary intervention service. A total of 191 patients aged ≤ 40 years with STEMI undergoing reperfusion therapy from January 2015 to April 2019 were consecutively included. In-hospital and one year outcomes were evaluated. The ROC curve was utilized to evaluate ACEF scoring system and predict one year clinical outcomes.

RESULTS: Mean age of the study population was 35.1±4.3 years old. According to ACEF score, 191 patients were divided into ACEFlow group (n=136) with ACEF score of 0.38–0.68(0.57±0.06) and ACEFhigh group (n=55) with ACEF score of 0.69–1.66 (0.86±0.18). The age, left ventricular ejection fraction (LVEF), presence of diabetes mellitus in the ACEFhigh group were significantly higher than those in ACEFlow group (all P<0.05). There was no difference in in-hospital outcomes between the two groups (all P>0.05). The one year clinical outcomes of cardiovascular death, all-cause death, myocardial infarction, hospitalization for cardiovascular events and coronary angiography occurred in 16.2% of ACEFlow group, 43.6 % of patients with ACEFhigh group (P<0.001).

CONCLUSION: The results of the study suggested that ACEF score can predict the clinical outcomes at 1 year after reperfusion therapy in STEMI patients aged <40 years old.

Keywords: ACEF Score, ST-segment Elevation Myocardial Infarction, Young

figure 1



Receiver-operating characteristic curve for ACEF in the predict of clinical outcomes

Tabel 1

Variables	ACEFlow group ≤0.68 (N=136)	ACEFhigh group >0.68 (N=55)	P value
Age, years	34.25±4.4	37.45±2.85	<0.001
Male, n(%)	120(88.2)	42(76.4)	0.035
Diabetes mellitus, n(%)	14 (10.3)	14 (25.5)	0.009
Hypertension, n(%)	19 (14.0)	8(14.5)	0.541
LVEF, %	59.0±5.02	44.7±6.44	<0.001
In-hospital outcomes			
In-hospital mortality, n(%)	1 (0.7)	0	0.712
Re-infarction, n(%)	2 (1.5)	4 (7.3)	0.059

Variables	ACEFlow group ≤ 0.68 (N=136)	ACEFhigh group > 0.68 (N=55)	P value
Cardiogenic shock, n(%)	1 (0.7)	1 (1.8)	0.494
Major bleeding, n(%)	1 (0.7)	0	0.712
Stroke, n(%)	0	1 (1.8)	0.431
One year clinical outcomes			
Hospitalization, n(%)	20 (14.7)	22 (40.0)	<0.001
Myocardial infarction, n(%)	3(2.2)	2 (3.6)	0.551
Coronary angiography, n(%)	14 (10.3)	13 (23.6)	0.018
Cardiovascular death, n(%)	2 (1.5)	2 (3.6)	0.326
All cause death, n(%)	1 (0.7)	0	0.712
Clinical outcomes	22 (16.2)	24 (43.6)	<0.001

Baseline characteristics, in-hospital and one year outcomes according to ACEF score

OP-013 RELATIONSHIP OF MAGNESIUM WITH MYOCARDIAL DAMAGE AND MORTALITY IN PATIENTS WITH COVID-19

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Background: Magnesium (Mg) is the second most abundant intracellular electrolyte and plays a significant role in immune system and cardiac protection. Mg deficiency contributes to chronic low-grade inflammation leading to cardiovascular diseases and low Mg level exacerbates virus-induced inflammation

Aim: To investigate whether serum magnesium level is associated with myocardial damage and prognosis of COVID-19.

Method: This was a single-center, observational retrospective study of patients with COVID-19. The study population was divided into two groups according to in-hospital mortality: a survivor group (SG) and a non-survivor group (NSG). Myocardial damage was defined as blood levels of cardiac Troponin I (cTnI) above the 99th-percentile upper reference limit. Magnesium, variables regarding inflammation and myocardial damage were compared between the groups.

Results: A total of 629 patients with COVID-19 were included. Mortality rate was 11.85% (n=82). There were 61 (74.4%) and 294 male patients (53.7%) in NSG and SG, respectively (p=0.001). The median age of NSG was 64.5 years (min-max: 37–93) and the median age of SG was 56.0 years (min-max: 22–92) (p<0.001). Median serum magnesium levels of NSG and SG were 1.94 mg/dl (min-max: 1.04-2.87) and 2.03 mg/dl (min-max: 1.18-2.88) respectively (p=0.027). Median cTnI levels of NSG and SG were 25.20 pg/ml (min-max: 2.10-2240.80) and 4.50 pg/ml (min-max: 0.50-984.3) respectively (p<0.001). The cTnI levels were lower in those patients whose serum Mg levels were higher than 1.94.

Conclusion: Although serum magnesium level was not a predictor for in-hospital mortality, there was a significant negative correlation between magnesium levels and myocardial damage.

Keywords: magnesium, covid-19, mortality, troponin, myocardium

Table-1: Comparison of variables regarding inflammation and myocardial damage with respect to the magnesium cut-off value

Variable	Magnesium ≤1.94 (mg/dL)	Magnesium > 1.94 (mg/dL)	p-value
NLR	3.21(0.54-37.00)	3.03(0.45-141.29)	0.941
PLR	179.12(4.96-1097.50)	188.79(5.02-1243.58)	0.068
MLR	0.37(0.00-8.43)	0.35(0.07-2.55)	0.385
cTnI (pg/mL)	6.85(0.60-2240.80)	4.40(0.50-2197.00)	<0.001
CRP (mg/L)	65.70(0.20-378.20)	63.00(0.30-451.40)	0.601
Ferritin (ng/mL)	223.35(8.90-1500)	269.60(6.60-1500)	0.151

CRP: C-reactive protein, NLR: neutrophil to lymphocyte ratio, PLR: platelet to lymphocyte ratio, MLR: monocyte to lymphocyte ratio, cTnI: cardiac troponin I

OP-014 BRAIN NATRIURETIC PEPTIDE LEVELS IN PATIENTS WITH COVID-19Gökhan Alıcı¹, Alaa Quisi²¹Adana Şehir Hastanesi, Adana²Adana Özel Medline Hastanesi, Adana

Objective: In December 2019, pneumonia associated with severe acute respiratory syndrome coronavirus 2 emerged in China, and since spread worldwide resulting in the coronavirus disease 2019 (COVID-19) pandemic. Brain natriuretic peptide (BNP) is a peptide hormone that is released in response to volume expansion and the increased wall stress of cardiac myocytes. In the present study, we aimed to assess BNP levels in patients with COVID-19 regarding in-hospital mortality.

Methods: This retrospective cohort study included a total of 65 COVID-19 patients. The diagnosis of COVID-19 was made according to the World Health Organization's interim guidance and confirmed by RNA detection of SARS-CoV-2. Pro-BNP levels on admission were measured.

Results: Pro-BNP level on admission was significantly higher in non-survivors COVID-19 patients than in survivors (1710.0 (84.0-28800.0) vs. 95.0 (65.0-25700.0), $p=0.005$). Forward stepwise logistic regression analysis demonstrated that age (OR=1.506 (1.016-2.230), $p=0.040$), and ferritin level on admission (OR=1.011 (1.001-1.021), $p=0.027$) was an independent predictor of in-hospital mortality of patients with COVID-19.

Conclusion: Pro-BNP level on admission is significantly higher in non-survivors COVID-19 patients than in survivors. However, further studies are warranted.

Keywords: Brain natriuretic peptide, COVID-19, mortality

OP-015 RELATIONSHIP BETWEEN ATHEROGENIC INDICES AND CARDIOVASCULAR DISEASES IN PATIENTS WITH COVID-19 PNEUMONIA

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Background: Covid-19 disease, caused by the coronavirus SARS-CoV 2, is a pandemic viral respiratory infection in which venous and arterial thromboembolic events are common. This study aimed to investigate the relationship between atherogenic indices and cardiovascular diseases in patients with Covid-19 pneumonia.

Method: In this retrospective study, a total of 805 inpatients (median age 63 [IQR: 52-74] years; 45.1% female) who were diagnosed with Covid-19 pneumonia between March 2020 and December 2020 were evaluated. Patients were divided into two groups based on cardiovascular events as non-cardiovascular event (n=709) and cardiovascular event (n=96). All clinical and demographic data, laboratory results were analyzed. Atherogenic Index of Plasma (AIP (log10 (triglyceride/ HDL)), Atherogenic Coefficient (AC (HDL/ non-HDL)) Risk Index of Castelli-I (CRI-I (Total cholesterol/ HDL)), Risk Index of Castelli-II (CRI-II (LDL/ HDL)), were calculated.

Results: Atherogenic Coefficient, CRI-I, CRI-II values were significantly higher in the cardiovascular event group (p=0.001, p=0.001, p=0.007, respectively). AIP values were higher in the cardiovascular event group, but it was not statistically significant (p=0.051). In the cardiovascular event group, HDL values were found significantly lower (p=0.001), but CRP and D-dimer values were found significantly higher (p<0.001, p=0.006 respectively). In the multivariable analysis, Atherogenic Coefficient (OR: 1.294, 95% CI: 1.089-1.537, p=0.003), D-dimer, Hypertension and current smoking were found to be independent predictors of cardiovascular events in patients with Covid-19 pneumonia.

Conclusion: Atherogenic indices could be used to predict cardiovascular events in patients with Covid-19 pneumonia.

Table 1. Clinical, demographic and laboratory characteristics of the study group according to cardiovascular events in patients with Covid-19 pneumonia

Variables	Total n=805	Non-cardiovascular event n=709	Cardiovascular event n=96	P value
Age, years	63 (52-74)	63 (51-74)	68 (57-80)	0.012
Female, n (%)	363 (45.1)	325 (45.8)	38 (39.6)	0.248
Diabetes Mellitus, n (%)	300 (37.3)	260 (36.7)	40 (41.7)	0.342
Hypertension, n (%)	406 (50.4)	345 (48.7)	61 (63.5)	0.006
Current smoking, n (%)	102 (12.7)	70 (9.9)	32 (33.3)	<0.001
Coronary artery diseases, n (%)	173 (21.5)	149 (21)	24 (25)	0.372
COPD/Asthma, n (%)	82 (10.2)	62 (8.7)	20 (20.8)	<0.001
Chronic renal failure, n (%)	120 (14.9)	112 (15.8)	8 (8.3)	0.054
Cerebrovascular diseases, n (%)	62 (7.7)	45 (6.3)	17 (17.7)	<0.001
Atrial fibrillation, n (%)	65 (8.1)	52 (7.3)	13 (13.5)	0.036
Malignancy, n (%)	104 (12.9)	93 (13.1)	11 (11.5)	0.649
eGFR ml/min/1.73 m ²	77.6± 40.3	76.8 ±40.1	83.9 ±40.9	0.106
Total cholesterol, mg/dL	169 (137-204)	169 (137-204)	165 (139-205)	0.883
LDL cholesterol, mg/dL	101 (77-131)	102 (77-131)	100 (76-136)	0.878

Variables	Total n=805	Non-cardiovascular event n=709	Cardiovascular event n=96	P value
HDL cholesterol, mg/dL	37 (30-46)	38 (31-47)	35 (28-40)	0.001
Triglyceride, mg/dL	129 (95-174)	129 (94-174)	125 (95-174)	0.869
C-Reactive Protein, mg/L	106 (31-190)	102 (28-183)	150 (64-229)	0.002
D-dimer, ng/ml	893 (500-1803)	869 (494-1627)	1290 (585-2803)	0.006
Creatinine, mg/dl	0.93 (0.73-1.29)	0.94 (0.74-1.29)	0.86 (0.67-1.30)	0.156
Atherogenic index of plasma (AIP)	0.18 ±0.26	0.17 ±0.25	0.23±0.24	0.051
Atherogenic coefficient (AC)	3.54 (2.66-4.46)	3.47 (2.62-4.43)	4.00 (3.18-4.88)	0.001
Castelli risk index I	4.55 (3.67-5.46)	4.48 (3.62-5.43)	5.00 (4.18-5.88)	0.001
Castelli risk index II	2.82 ±1.03	2.78 ±1.03	3.09± 0.96	0.007

Table 2. Univariable and multivariable logistic regression analysis to detect the independent predictors of cardiovascular events in patients with Covid-19 pneumonia

Variables	Univariable Analysis			Multivariable Analysis		
	OR	(95% CI)	P value	OR	(95% CI)	P value
Atherogenic coefficient (AC)	1.286	1.103-1.499	0.001	1.294	1.089-1.1537	0.003
Age	1.015	1.001-1.029	0.030	1.010	0.993-1.027	0.262
Gender	0.774	0.501-1.196	0.249	1.042	0.635-1.709	0.872
D-Dimer	1.000076	1.000013-1.000138	0.018	1.000077	1.000013-1.000140	0.018
Diabetes Mellitus	1.234	0.800-1.903	0.343	0.977	0.595-1.602	0.925
Current Smoking	4.564	2.794-7.457	<0.001	6.113	3.510-10.649	< 0.001
Hypertension	1.839	1.183-2.858	0.007	1.852	1.078-3.179	0.026
C-Reactive protein	1.003	1.001-1.004	0.005	1.001	0.999-1.003	0.189
Atherogenic index of plasma (AIP)	2.270	0.994-5.186	0.052			
Castelli Risk Index I (CRI-I)	1.283	1.100-1.497	0.002			
Castelli Risk Index II (CRI-II)	1.319	1.075-1.617	0.008			

OP-016 COMPARISON OF COVID-19 PATIENTS ACCORDING TO THE SURVIVAL TIME IN THE TURKISH POPÜLATION: A RETROSPECTIVE STUDY

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Background-Aim: Coronavirus disease 2019 (COVID-19) was first detected as a form of atypical pneumonia. COVID-19 is a highly contagious virus, and some patients may experience acute respiratory distress syndrome (ARDS) and acute respiratory failure leading to death. We aim to evaluate the clinical, imaging, and laboratory parameters according to survival time to predict mortality in fatal COVID-19 patients.

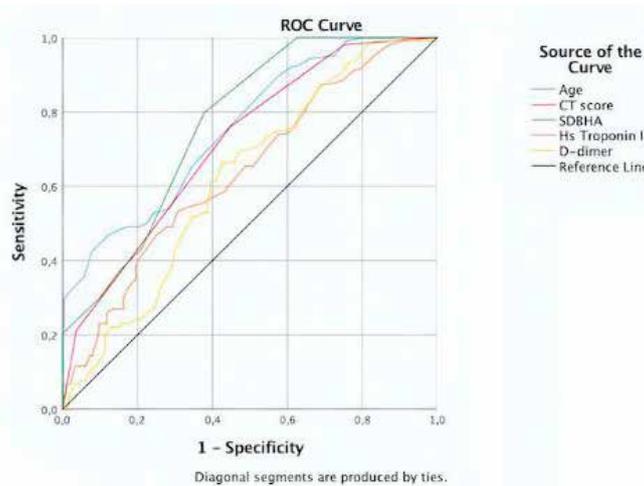
Materials-Methods: Three hundred and fifty patients who died and 150 COVID-19 patients who survived were included in the study. Patients who died were divided into three groups according to the median value of survival days. Demographic characteristics and in-hospital complications were obtained from medical databases.

Results: Of the non-surviving patients, 30% (104) died within three days, 32% (110) died within 4–10 days, and 39% (136) died after ten days. Pneumonia on computational tomography (CT), symptoms duration before hospital admission (SDBHA), intensive care unit (ICU) admittance, hypertension (HT), C-reactive protein test (CRP), d-dimer, multi-organ dysfunction syndrome (MODS), cardiac and acute kidney injury, ejection fraction, right ventricular fractional area change (RV-FAC) and Tocilizumab/Steroid were independent predictors of mortality within three days compared to 4–10 days and over ten days mortality. A combined diagnosis model was evaluated for the age, CT score, SDBHA, hs-Tnl, and d-dimer. The combined model had a higher area under the ROC curve (0.913).

Conclusion: This study showed that age, pneumonia on CT, SDBHA, ICU, HT, CRP, d-dimer, cardiac injury, MODS, acute kidney injury, left ventricular ejection fraction (LVEF), RV-FAC, and Tocilizumab/Steroid were independently associated with short-term mortality in non-surviving COVID-19 patients in the Turkish population.

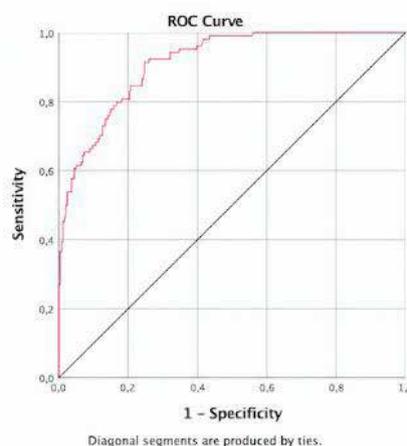
Keywords: COVID-19, mortality, acute respiratory distress syndrome, echocardiography

Figure-1



In ROC curve analyses, areas under the curve (AUC) for Age, computed tomography (CT) score, symptom duration before hospital admission (SDBHA), high sensitive-Troponin I (hs-Tnl), and D-dimer were determined (0.755 / 0.734 / 0.766 / 0.639 / 0.620 respectively).

Figure-2



The combined diagnosis model of the age, computed tomography (CT) score, symptom duration before hospital admission (SDBHA), high sensitive-Troponin I (hs-Tnl), and D-dimer was analyzed by the ROC curve. The red line represents the combined diagnosis model, and the area under the curve (AUC) was 0.913.

OP-018 ASSOCIATION OF LABORATORY MARKERS WITH MORTALITY IN PATIENTS WITH COVID-19 PNEUMONIA

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Background: COVID-19 has become a pandemic spreading worldwide and the number of affected patients is gradually increasing. Early identification of laboratory markers is crucial to determine the severe form of this disease. The aim of this study was to investigate the relationship between laboratory parameters on admission and prognosis of COVID-19 pneumonia.

Methods: This study designed as a retrospective study. A total of 140 patients followed up in our hospital due to COVID-19 pneumonia were included in the study. (83 males, mean age 61,1 ± 13,7). Patients' demographic data, comorbid diseases and routine laboratory parameters such as hemogram lipid profiles, renal function tests at admission were recorded and analyzed. The results of laboratory examinations were compared between the non-mortality and mortality groups.

Results: The main demographic findings were similar among two groups. ($p > 0.05$) GFR, hemoglobin, hematocrit, lymphocyte, albumin values were significantly lower in the mortality group ($p < 0.05$). WBC, neutrophil, RDW, CRP, uric acid, D-dimer, ferritin values were significantly higher in the mortality group ($p < 0.05$) (Table 2). However, HDL, monocyte, monocyte / HDL, platelet, MPV values did not differ significantly ($p > 0.05$) between the two groups.

Conclusion: Several laboratory markers may be helpful to assess the severity and mortality status of the disease. In particular, low lymphocyte counts and high CRP and D-dimer levels can be considered as indicators of the poor prognosis.

Keywords: COVID-19, laboratory markers, mortality

Table 1

	Min-Max	Median	Mean ±ss/n-%
Age(years)	29,0 - 87,0	60,0	61,1 ± 13,7
Gender	Female		57 41,0%
	Male		83 59,7%
CAD			32 23,0%
HT			54 38,8%
HL			29 20,9%
DM			59 42,4%
SVO			8 5,8%
COPD			16 11,5%
CA			15 10,8%
Smoking			36 25,9%
CKD			25 18,0%
AF			12 8,6%
CHF			18 12,9%
AKD			13 9,4%
ARDS			77 55,4%
Yatış Süresi	1,0 - 32,0	11,0	12,3 ± 7,6
EX			48 34,5%
HDL(mg/dL)	14,0 - 74,0	33,0	33,8 ± 9,7
Monocyte($10^3/\mu\text{L}$)	0,1 - 1,2	0,4	0,5 ± 0,2
Monocyte/HDL	0,0 - 0,1	0,0	0,0 ± 0,0
GFR($\text{mL}/\text{min}/1.73\text{m}^2$)	4,0 - 136,0	87,0	78,2 ± 30,4
HGB(g/dl)	3,0 - 17,0	12,3	12,1 ± 2,3
HTC(%)	17,0 - 57,0	38,1	37,6 ± 6,2
WBC($10^3/\mu\text{L}$)	1,1 - 20,8	7,3	7,8 ± 3,7
NEU($10^3/\mu\text{L}$)	0,5 - 19,8	5,4	6,0 ± 3,5
LEN($10^3/\mu\text{L}$)	0,3 - 2,8	1,0	1,1 ± 0,5
RDW(%)	11,0 - 25,1	13,8	14,4 ± 2,3
PLT($10^3/\mu\text{L}$)	95,0 - 639,0	207,5	229,1 ± 94,1
MPV(fl)	8,8 - 13,1	10,6	10,7 ± 0,9
CRP (mg/L)	0,9 - 539,0	192,0	201,0 ± 121,5
Albumin(g/dl)	1,7 - 4,7	3,3	3,3 ± 0,6
Uric acid(mg/dl)	1,6 - 12,7	4,8	5,4 ± 2,3
D-Dimer (pg/dL)	0,1 - 9,0	1,7	3,0 ± 2,9
Ferritin	15,2 - 1500,0	544,0	690,7 ± 507,3

Demographic and clinical data of the study population

Table 2

	Mortality (-)		Mortality(+)		p
	Mean. ±ss	Median	Mean. ±ss	Median	
HDL(mg/dl)	33,4 ± 9,8	33,0	34,5 ± 9,7	32,0	0,533 ^m
Monocyte(10 ³ /μL)	0,5 ± 0,2	0,4	0,5 ± 0,2	0,5	0,684 ^m
Monocyte/HDL	0,0 ± 0,0	0,0	0,0 ± 0,0	0,0	0,874 ^m
GFR(mL/min/1.73m ²)	86,6 ± 25,7	95,0	62,2 ± 32,4	69,0	0,000 ^m
Hemoglobin(g/dl)	12,6 ± 2,0	13,0	11,2 ± 2,5	11,2	0,002 ^m
Hematocrit(%)	38,7 ± 5,7	39,6	35,6 ± 6,6	36,4	0,008 ^m
WBC(10 ³ /μL)	7,2 ± 3,6	6,7	8,9 ± 3,6	8,3	0,002 ^m
NEU(10 ³ /μL)	5,4 ± 3,4	4,8	7,3 ± 3,4	6,7	0,000 ^m
LEN(10 ³ /μL)	1,2 ± 0,5	1,1	1,0 ± 0,5	0,8	0,003 ^m
RDW(%)	13,9 ± 2,2	13,2	15,4 ± 2,1	15,2	0,000 ^m
PLT(10 ³ /μL)	233,9 ± 99,0	209,5	219,8 ± 84,0	192,0	0,433 ^m
MPV(fl)	10,7 ± 0,9	10,5	10,7 ± 0,9	10,7	0,772 ^m
CRP (mg/L)	152,3 ± 100,4	133,9	293,3 ± 103,7	302,0	0,000 ^m
Albumin(g/dl)	3,5 ± 0,5	3,5	3,0 ± 0,5	3,0	0,000 ^m
Uric acid(mg/dl)	4,9 ± 2,1	4,3	6,5 ± 2,3	6,0	0,000 ^m
D-Dimer (pg/dL)	2,0 ± 2,4	1,0	5,0 ± 2,7	5,0	0,000 ^m
Ferritin (ng/mL)	541 ± 435	427	981 ± 515	1120	0,000 ^m

^t t test / ^m Mann-whitney u test

Comparison of laboratory values between patients with COVID-19 mortality and non-mortality

OP-019 THE BENEFIT OF THE SYSTEMIC IMMUN-INFLAMMATION INDEX IN THE DIFFERENTIAL DIAGNOSIS OF MASSIVE AND NON-MASSIVE PULMONARY EMBOLISM

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Objective: Previous studies have shown that neutrophil-to-lymphocyte and platelet-to-lymphocyte ratios may be useful in the diagnosis of pulmonary embolism. In this study, we aimed to investigate whether the systemic immune-inflammation index (SII), which is a novel index that incorporates these two indexes, may be useful in the differential diagnosis of massive and non-massive pulmonary embolism.

Method: The study was designed as a retrospective cohort study. Our study was planned with a total of 200 patients who applied to our emergency department in the last 3 years and were diagnosed with pulmonary embolism. For the classification of massive and non-massive embolism, the ESC guideline was taken as a reference. From the hospital records of all patients included in the study, the diagnosis of pulmonary embolism was selected from patients whose diagnosis was confirmed by CT pulmonary angiography. Laboratory records and echocardiographic records of all patients were analyzed from hospital records. Patients without laboratory records or echocardiography records were excluded from the study. The study was designed with a total of 145 patients after excluding patients with renal failure, rheumatological disease, active infection and active cancer diagnoses that would change the laboratory results.

Results: A total of 145 patients were included in our study, including 68 massive embolism (46.9%) and 77 non-massive (53.1%) acute pulmonary embolism. 58 (40%) of the patients included in the study were women. It was significantly higher in the SII massive embolism group than in the non-massive embolism group ($p < .01$). By using Pearson's correlation analysis, SII was found to be highly correlated with d-dimer and troponin ($p < .001$). In the differential diagnosis of massive and non-massive embolism, receiver operating characteristic curve were used. The cut off value of SII was tried to be found. We found that SII could distinguish massive embolism from non-massive embolism with 91% sensitivity and 62% specificity above 516.76. We found that SII could distinguish massive embolism from non-massive embolism better when compared to the number of neutrophils and platelets alone.

Conclusion: In our study, we found that SII can be an early and good index in the differential diagnosis of massive and non-massive pulmonary embolism and may be useful in early diagnosis.

Keywords: pulmonary embolism, Systemic Immun-Inflammation Index, massive embolism

Introduction: Acute pulmonary embolism (APE) is one of the leading causes of death worldwide. It caused more than 100,000 deaths in 2018 (1,2). Early diagnosis is life-saving in APE, as deaths usually occur within the first hours. Studies have found that more than 70% of the deaths from APE occur within the first hour (3). Many risk factors have been identified for acute pulmonary embolism. The most common risk factor for APE is a history of deep vein thrombosis (DVT). (4). The pathogenesis of risk factors encountered in APE can be explained by the triad of Virchow. Diseases and conditions that cause endothelial damage, hypercoagulability, and stasis are roughly considered risk factors for pulmonary embolism (5).

Although many classifications have been suggested for APE, the most commonly used classification is massive and non-massive. The diagnosis of massive pulmonary embolism is established with the presence of one of these criteria: right heart failure, cardiovascular collapse, shock and hypotension, 50% or more occlusion of the main pulmonary artery, or inotropic dependency (6). Massive pulmonary embolism can be fatal within hours, sometimes minutes, if not treated early (7).

Echocardiographic findings, presence of thrombus in venous doppler ultrasonography, thrombus monitoring in CT pulmonary angiography and biomarkers showing right ventricular loading are used for the differential diagnosis of APE. All these investigations are expensive and take time for obtaining results (8). There is an increasing number of studies showing that blood inflammatory markers can be used in the differential diagnosis of APE. Procoagulatory and proinflammatory conditions occur during the course of APE due to mediators secreted from platelets and leukocytes (9). In the course of APE, the number of neutrophils increases due to the increased inflammatory response, and the number of lymphocytes decreases due to the increased sympathetic activity and glucocorticoids. It has been previously investigated whether the neutrophil/lymphocyte ratio (NLR) and platelet/lymphocyte ratio (PLR) are associated with poor prognosis, based on all these physiological responses. Studies have shown that increased NLR and PLR are predictors of poor prognosis (10). In this study, we aimed to investigate whether the systemic immune inflammation (SII) index, which is a new index and formulated as neutrophil x platelet/lymphocyte, can be useful in the differential diagnosis of massive and non-massive pulmonary embolism.

Material and Methods: This study was designed as a retrospective cohort study with a total of 200 patients who were admitted to the emergency service of our hospital and diagnosed with pulmonary embolism within the last 3 years. Massive and non-massive embolism classification was based on the ESC guideline (11). All patients included in the study were selected from hospital records of the patients whose diagnosis of pulmonary embolism was confirmed by CT pulmonary angiography. Laboratory records and echocardiographic records of all patients were reviewed from hospital records. Patients with missing laboratory or echocardiographic records were excluded from the study. After patients with kidney failure, rheumatological disease, active infection, and active cancer that would alter laboratory results were excluded, the study was executed with the remaining 145 patients. SII was formulated as neutrophil x platelet/lymphocyte counts and calculated using Microsoft® Excel software.

Statistical Analysis

Statistical analyses were performed using SPSS statistical software version 22 (IBM Inc., NC, USA). Patient characteristics were compared with the Chi-squared test and median values were compared using the non-parametric ranking test. Independent Samples Test was used for the group comparisons by means, while Pearson test was used to analyze a correlation relationship between all the parameters. The best cut-off values of SII levels were determined using the receiver operating characteristic (ROC) curve analysis to predict the disease. All p-values were based on two-sided testing and $p < 0.05$ values were considered statistically significant.

Results: A total of 145 patients were included in the study with 68 (46.9%) having massive and 77 (53.1%) non-massive pulmonary embolism. Laboratory data of the patients are presented in Table 1. Of all patients included in the study, 58 (40%) were female. SII was significantly higher in the massive embolism group than in the non-massive embolism group. ($p < .01$). Using Pearson correlation analysis, it was determined that the SII index was highly correlated with D-dimer and troponin ($p < .001$).

	N	Minimum	Maximum	Mean	Std. Deviation
GLUCOSE	145	70	388	130.21	56.095
POTASSIUM	145	3.00	6.70	4.3659	.50726
SODIUM	142	120	145	137.21	3.866
UREA	144	13	94	34.49	14.400
CREATININE	144	.40	2.74	.8432	.27533
WBC	145	.55	25.29	10.3303	3.89767
NEU	145	.11	17.52	7.2692	3.42928
LYM	145	.17	5.36	2.0361	.94156
MONOCY	145	.05	3.44	.8048	.42851
PLT	145	61	620	259.26	95.822
LYM%	145	1.7	45.7	21.234	9.3983
MONO%	145	.6	45.5	8.123	4.3421
NEU%	145	14.5	92.4	67.703	12.7045
RDW	145	11.5	27.4	14.751	2.4165
PLT/LYM	145	44.59	1646.15	161.5592	151.53298
NEU/LYM	145	.65	55.77	4.8376	5.74705
D-DIMER	143	.10	34.40	4.9571	5.86849
CRP	145	1.30	404.30	61.7747	72.46351
PAP	145	24.0	73.0	41.703	12.2928
TROPONIN	145	1.10	419.10	25.0692	48.38028
Systolic BP	145	83	142	112.83	15.023
Diastolic BP	145	48	86	69.21	10.183
SYSTEMIC IMMUNE-INFLAMMATION INDEX	145	43.35	23869.23	1262.9509	2117.37195
Valid N (listwise)	139				

We tried to find the cut-off value of SII in the differential diagnosis of massive and non-massive embolism with the receiver operating characteristic (ROC) curve (Figure 1). It was found that the SII cut-off value ≥ 516.76 was able to distinguish massive emboli from non-massive embolism with a sensitivity of 91% and a specificity of 62% (Table 2). It was determined that SII could better distinguish massive embolism from non-massive embolism compared with the neutrophil and platelet counts alone.

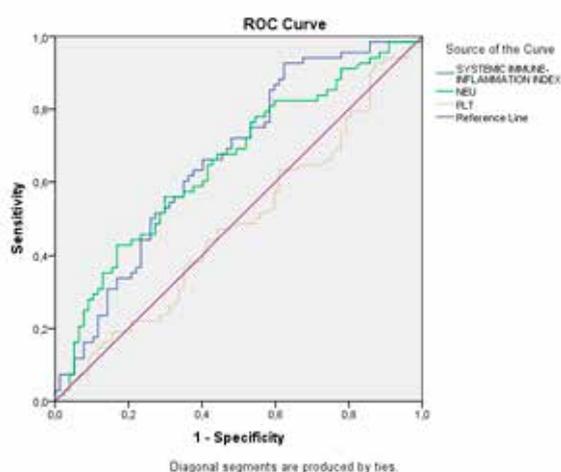


Figure 1. ROC analysis curve plotted for determining cut-off value of SII.

VARIABLE	AUC	95% CI
SII	.665	0.577-0.753
NEUTROPHIL COUNT	.653	0.564-0.743
PLATELET COUNT	.484	0.389-0.579

Discussion: In our study, we found that SII, which is a novel index, can distinguish massive pulmonary embolism from non-massive embolism well. Despite the development of new treatment and diagnostic modalities for APE, mortality rates are still very high. Although it has been shown that BNP, Troponin and NT-proBNP may be useful for early risk categorization in APE, it is not always possible to study these laboratory values in daily practice. Therefore, it would be useful to find simpler and prognostic laboratory tests. It has been shown that NLR and PLR can be useful in determining the prognosis in cardiovascular diseases and peripheral arterial diseases, because they reflect both proinflammatory and procoagulatory status (12, 13). In the course of APE, procoagulant and proinflammatory microparticles are released from leukocytes, platelets and endothelial cells (14). Platelets are reactivated and neutrophils are remodeled during this increased inflammatory process in APE. Previous studies have found that NLR and PLR are associated with poor prognosis in APE. Pahn et al. found that NLR and PLR were independent predictors of mortality in APE (10).

The number of studies in the literature investigating the relationship of SII, a new index, with cardiovascular diseases is increasing. Early detection of the high-risk group in patients with acute coronary syndrome will reduce mortality and morbidity. G. Su et al. found that SII was an independent predictor of 30-day mortality in patients with acute coronary syndrome. (15).

There are many studies in the literature reporting that the neutrophil count markedly increased and the lymphocyte count decreased in patients who died due to APE. Therefore, NLR is expected to be higher in the exitus patient group. Phan et al. showed that the neutrophil and platelet counts changed minimally and the lymphocyte count decreased in the group that died from APE (10). Conflicting data on inflammation-related markers in the literature show that the proinflammatory and procoagulatory status is very variable in the course of APE. All these results prompt researchers to search for new markers to better demonstrate the complex immune inflammation process. Gok et al. found that SII can indicate the severity of acute pulmonary embolism and is an independent predictor of mortality (16).

Recently, there has been an increasing number of studies showing that a new index, SII, is associated with the prognosis of many clinical conditions. Chen et al. found that SII is important in determining the prognosis in colorectal cancer patients. Again in that study, they found that NLR, PLR and SII were independent predictors of mortality. As a result of the study, they found that SII could be useful in differentiating high risk patients (17).

In our study, we used SII considering that it would better reflect both the proinflammatory and procoagulatory situation. We determined that SII, which is a cost effective and easy to obtain parameter that can be studied early in massive and non-massive APE, can be useful in the differential diagnosis. Our study will shed light on future prospective studies.

Conclusion: In our study, we found that SII may be an early and good marker in the differential diagnosis of massive and non-massive pulmonary embolism and may be useful in early diagnosis.

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OP-020 NEWLY DEFINED PROGNOSTIC INDICATOR FOR PATIENTS WITH ACUTE PULMONARY EMBOLISM: SYSTEMIC IMMUNE-INFLAMMATION INDEX

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Background: Inflammation and thrombosis are interrelated, with up-regulation of pro-inflammatory and pro-coagulatory mediators in the setting of acute pulmonary embolism (APE). Inflammation increases procoagulant factors, and also inhibits natural anticoagulant pathways and fibrinolytic activity, causing a thrombotic tendency. In addition, chronic inflammation may cause endothelial damage, resulting in the loss of physiologic anticoagulant, antiaggregant and vasodilatory properties of endothelium. Systemic immune-inflammation index (SII), is a recently described inflammation-related indicator that integrates neutrophil, platelet, and lymphocyte counts and can reflect the comprehensive immune and inflammation situation in the body. The present study aimed to examine the predictive value of a novel SII index in patients with APE.

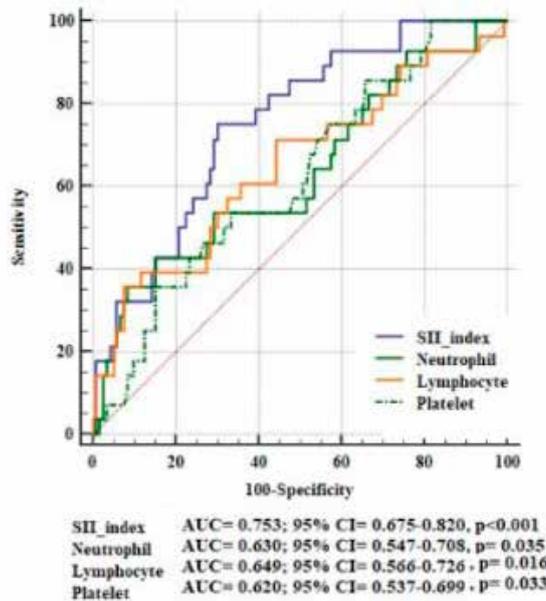
Methods: A total of 148 patients with APE, a median age of 65.6 [54.0-76.0], were included in this retrospective observational study. Admission blood samples were collected for SII and other laboratory measurements. The SII index was calculated using the formula platelet x neutrophil/lymphocyte counts. The study population was divided into two groups defined as survivors and non-survivors.

Results: Nonsurvivor patients were older ($p= 0.032$) and had more malignancy ($p< 0.001$) and CVA histories ($p= 0.021$). Deceased individuals had more massive APE ($p= 0.001$) and had higher Wells ($p= 0.003$) and PESI scores ($p= 0.023$). SII levels were significantly higher in patients with in-hospital death ($p= 0.015$). Multivariable cox regression analysis revealed that SII index (Odds ratio [OR]= 1.000, 95% confidence interval [CI]= 1000-1001, $p= 0.002$), older age (OR= 1.055, 95% CI= 1.019-1.092, $p= 0.002$), malignancy history (OR= 4.170, 95% CI= 1.697- 10.249, $p= 0.002$), elevated troponin levels (OR= 3.156, 95% CI= 1.732- 5.749, $p< 0.001$) and high PESI score (OR= 3.508, 95% CI= 1.535- 8.018, $p= 0.003$) were independent predictors for in-hospital mortality. In the receiver operating characteristic curve, the optimal cutoff value of SII to predict the in-hospital mortality 1494, with 75% sensitivity and 70% specificity (area under the curve: 0.753). Furthermore, the predictive performance of the SII index was superior to those of neutrophils, lymphocytes, and platelets ($p= 0.040$, $p= 0.041$, $p= 0.034$, respectively).

Conclusion: We concluded that SII may be useful immun-inflammatory parameter in identifying high-risk patients for in-hospital mortality in patients with APE.

Keywords: Acute pulmonary embolism, mortality, systemic immune inflammation index

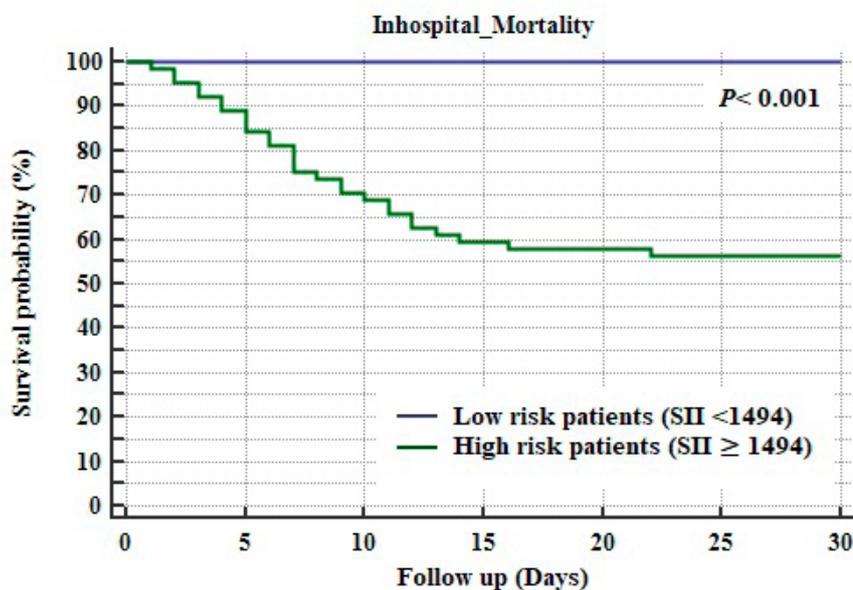
Figure 1.ROC curves of the SII index (blue), neutrophil (green), lymphocyte (orange) and platelet (dashed green) for detecting the in-hospital mortality



	Difference between areas	95% CI	Z-statistic	P
SII index vs Neutrophil	0.122	0.006 - 0.239	2.053	0.040
SII index vs Lymphocyte	0.104	0.005 - 0.203	2.041	0.041
SII index vs Platelet	0.132	0.010 - 0.255	2.119	0.034

Abbreviations: AUC, area under the curve; CI, confidence interval; SII, systemic immune-inflammation index

Figure 2. Kaplan–Meier plots of clinical outcome of the high risk patients categorized by SII index. Blue line means low-risk individuals and green line means high risk individuals.



Abbreviations: SII, systemic immune-inflammation index

Table 1. Demographic, clinical and laboratory parameters of the study cohort

Variables	All population (n = 148)	Survivor (n = 130)	Non-survivor (n = 28)	p
Male gender, n (%)	68 (46.6)	55 (44.6)	13 (46.4)	0.986
Age, year, [IQR]	65.6 [54.0-76.0]	65.0 [51.0-74.0]	73.0 [61.3-79.3]	0.032
BMI (kg/m ²)	28.1±3.2	28.4±3.1	24.9±4.1	0.148
Hypertension, n (%)	67 (49.9)	54 (48.3)	10 (35.7)	0.229
Diabetes mellitus, n (%)	42 (28.8)	31 (26.3)	11 (39.3)	0.171
CAD history, n (%)	16 (11.0)	14 (11.9)	2 (7.1)	0.472
CHF history, n (%)	16 (11.0)	12 (10.2)	4 (14.3)	0.531
CVA history, n (%)	11 (7.5)	6 (5.1)	5 (17.9)	0.021
Malignancy history, n (%)	16 (11)	7 (5.9)	9 (32.1)	< 0.001
Presence of COPD, n (%)	18 (12.3)	16 (13.6)	2 (7.1)	0.353
Presence of CRF, n (%)	10 (6.8)	8 (6.8)	2 (7.1)	0.945
Smoking, n (%)	33 (22.8)	28 (23.9)	5 (17.9)	0.491
Immobilization, n (%)	46 (31.5)	33 (28)	13 (46.4)	0.059
Prior DVT, n (%)	59 (40.4)	44 (37.3)	15 (53.6)	0.114
Dyspnea, n (%)	132 (90.4)	106 (89.8)	26 (92.9)	0.625
Chest pain, n (%)	77 (52.9)	66 (55.9)	11 (39.3)	0.353
Hemoptysis, n (%)	13 (8.9)	8 (6.8)	5 (17.9)	0.064
Massive, n (%)	42 (28.4)	27 (22.5)	15 (53.6)	0.001
Submassive, n (%)	55 (37.2)	46 (39.2)	9 (32.1)	0.542
Nonmassive, n (%)	51 (34.5)	47 (39.2)	4 (13.4)	0.013
Wells score	4.4±1.8	5.3±1.8	4.2±1.7	0.003
High PESI, n (%)	52 (53.1)	37 (30.8)	15 (53.6)	0.023
Systolic BP (mmHg)	116.1±20.3	117.9±19.4	108.8±22.6	0.032
Diastolic BP (mmHg)	72.2±13.8	73.8±13.9	65.4±11.7	0.003
PAPs, (mmHg)	46.6±11.3	43.7±9.5	58.6±10.8	<0.001

Variables	All population (n = 148)	Survivor (n = 130)	Non-survivor (n = 28)	p
Main pulmonary artery, n (%)	72 (48.6)	56 (46.7)	16 (57.1)	0.318
Lobar, n (%)	98 (66.2)	78 (65)	20 (71.4)	0.517
Segmenter, n (%)	101 (68.2)	83 (69.2)	18 (64.3)	0.617
Subsegmenter, n (%)	46 (31.1)	35 (29.2)	11 (39.3)	0.298
Pleural effusion, n (%)	59 (39.9)	43 (35.8)	16 (57.1)	0.038
Consolidation, n (%)	55 (37.2)	42 (35)	13 (46.4)	0.260
Diaphragm elevation, n (%)	5 (3.4)	2 (2.5)	2 (7.1)	0.221
Glucose, mg/dL, [IQR]	121 [103-169]	120 [103-157]	148 [103-237]	0.042
eGFR, mL/min/1.73m ²	82.7±19	83.8±19	78.1±17	0.158
Haemoglobin, g/dL	12.5±2.3	12.4 ± 2.4	12.8±2.4	0.793
Neutrophil, 103/μL, [IQR]	6.9 [5.5-9.4]	6.9 [5.1-8.6]	8.1 [6.2-12.7]	0.010
Lymphocyte, 109/L, [IQR]	1.55 [1.12-2.31]	1.64 [1.15-2.34]	1.22 [0.78-2.03]	0.061
Platelet, 109/L	251 [208-333]	245 [205-324]	298 [230-368]	0.039
SII	1180 [707-2031]	1003 [663- 1719]	1922 [1324-3518]	0.015
Cardiac troponin (ng/mL)	1.2 [0.8-1.5]	1.1 [0.8-1.5]	1.5 [1.1-1.9]	0.002
D-dimer (ng/mL)	4029 [2299-6083]	3913 [2071- 5974]	4596 [2850-7646]	0.058

Abbreviations: BMI, body mass index; CAD, coronary artery disease; CHF, chronic heart failure; CVA, cerebrovascular accident; COPD, chronic obstructive pulmonary disease; CRF, chronic renal failure; DVT, deep vein thrombosis; PESI, pulmonary embolism severity index; BP, blood pressure; PAPs, pulmonary artery systolic pressure; eGFR, estimated glomerular filtration rate; SII, systemic immune-inflammation index.

OP-021 PINCHED THROMBUS IN THE PATENT FORAMEN OVALE CAUSING PULMONARY AND PARADOXICAL SYSTEMIC EMBOLISM

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Introduction: Patent foromene ovale (PFO) is found in 20-25% of healthy individuals as a remnant of fetal circulation. It is generally considered asymptomatic but can cause paradoxical embolism, migraine, decompression sickness and platypne-orthodoxy syndrome. In addition, the presence of PFO increases mortality in patients with pulmonary embolism. Increase in right atrial pressure due to pulmonary embolism may cause paradoxical embolism by exacerbating the right-left shunt through PFO. Systemic embolism concurrent with pulmonary embolism is a rare condition. Sometimes large thrombi are stuck in the tunnel during its passage from PFO and can be detected by echocardiography.

Case: A 66-year-old male patient, who underwent 3-vessel coronary artery bypass grafting (CABG) 10 days ago for acute coronary syndrome (ACS), was admitted to the emergency room with complaints of dyspnea, hypotension and edema in the legs. There was tension arterial 80/55 mmhg on physical examination and sinus tachycardia on ECG. In the echocardiography, performed at the bedside with the pre-diagnosis of pericardial tamponade; it was found that right heart chambers dilated, moderate tricuspid regurgitation and systolic pulmonary artery pressure 45 mmhg. A large thrombus was observed in the interatrial septum, with one end extending to the right atrium and the other to the left atrium (Figure 1-A). Transoesophageal echocardiography (TEE) was performed on this to visualize the PFO passage (Figure 1-B). However, it was observed that the thrombus extending to the right and left atrium disappeared on TEE. An embolism was detected in the left main pulmonary artery in thoracic angio computed tomography (CT) imaging (Figure 2-A). Brain CT and MRI images taken in terms of paradoxical embolism were normal. In peripheral angio CT, embolism was detected in the superior mesenteric artery and the right common iliac artery (Figure 2-B). Thrombolytic therapy could not be given due to a cardiac operation 10 days ago. Low molecular weight heparin (LMWH) treatment was initiated. Short segment bowel resection was performed. After the operation, LMWH treatment was continued, the picture of acute abdomen improved, signs of pulmonary embolism regressed, peripheral artery ischemia did not develop. The patient was discharged with oral anticoagulant therapy on the 11th day of hospitalization.

Discussion: Thrombi attached to PFO are rare, but their mortality is high because they cause pulmonary and systemic embolism. Early diagnosis and treatment reduce mortality. Anticoagulant, thrombolytic or surgical treatment can be chosen according to the clinical condition of the patient. PFO closure should be considered to prevent recurrences.

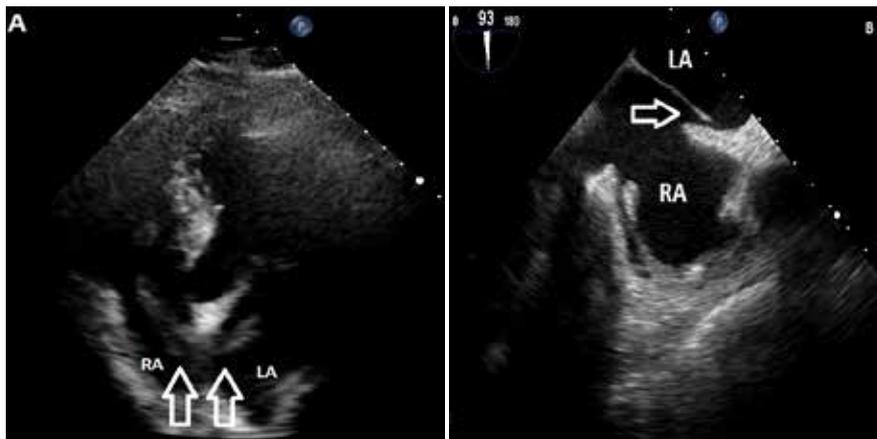


Figure 1: Large thrombus extending from the right atrium to the left atrium (A), disappearance of thrombus in TEE (B).

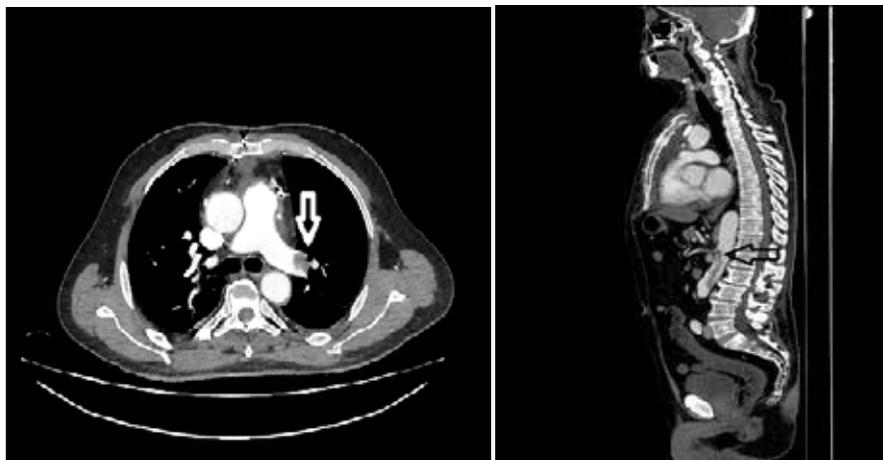


Figure 2: Thromboembolism in the left main pulmonary artery (A), and superior mesenteric artery (B)

OP-022 THE ROLE OF COMPUTED TOMOGRAPHY IN THE CALCULATION OF PULMONARY ARTERY SYSTOLIC PRESSURE IN PATIENTS WITH PULMONARY HYPERTENSION

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INTRODUCTION: Pulmonary hypertension (PH) refers to a life-threatening condition as a result of progressive right heart failure where the mean pulmonary artery pressure (oPAP) as demonstrated by right heart catheterization is ≥ 25 mmHg (1). Although right heart catheterization is the gold standard diagnostic method, it is an expensive method with known complications (2). Echocardiography is the most commonly used imaging method in screening and follow-up due to its easy accessibility, reproducibility and cheapness. Doppler echocardiography derived systolic pulmonary artery pressure (sPAP) is the most used screening tool in the assessment of pulmonary hypertension (PH). Because of suboptimal echocardiographic image quality in patients with chronic obstructive pulmonary diseases, obesity, and thoracic deformities, or in the setting of trivial tricuspid regurgitation, sPAP may not be calculated accurately with echocardiography. Chest computed tomography (CT) is a valuable diagnostic tool in patients with PH. In this study we investigated the role of CT in the estimation of sPAP in comparison with echocardiography.

METHODS

Study Population: Those patients who had been hospitalized or under follow-up on an outpatient basis for different diagnoses with a sPAP ≥ 35 mmHg as assessed with Doppler echocardiography, and who had been undergone contrast-enhanced chest 64-section CT within three days of echocardiographic examination were retrospectively analyzed. 57 patients (37 female, 20 male) with adequate CT scan quality were included in the study.

CT assessment and CT Metrics: The CT device used is a 0.6 mm collimation scanner with a gantry rotation time of 0.33 seconds and a minimal slice thickness of 0.75 mm. Using an intravenous automatic injection system, (400 mg iodine/ml) was infused at a rate of 3-4 ml/sec. The tube voltage was set as 120 kV and the tube current as 250 mAs. Pitch value was 0.75. The reconstruction interval was 0.5 mm. After analyzes of CT scans of all patients, the diameters of main pulmonary artery (PA), right and left pulmonary arteries, right descending pulmonary artery (RDPA), left descending pulmonary artery, wall thicknesses and diameters of right and left ventricles, thoracic diameters (TD), and diameters of ascending aorta (AA) and descending aorta (DA) were obtained (Figure 1). Additionally, the ratios of PA/DA, PA/AA ve PA/TD were calculated. The relationships between these parameters and the sPAP were assessed.

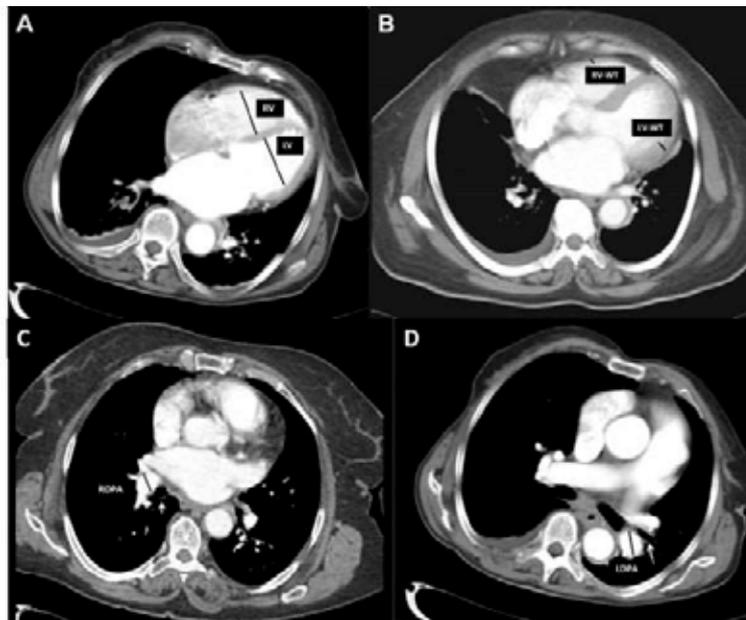


Figure 1: Measurements of A: Right ventricle (RV) and left ventricle (LV), B: Right ventricular wall thickness (RVWT) and left ventricular wall thickness (LVWT), C: Right descending pulmonary artery (RDPA), D: Left descending pulmonary artery (LDPA).

Statistics: The mean values and standard deviations of the continuous variables, the lower and upper limits of their distribution, and the frequencies of the categorical variables were calculated. The differences between continuous variables were compared using the Mann Whitney U test, and categorical variables were compared using the Chi-square test. The relationship between sPAP and CT measurements and derivatives was calculated by linear regression. Calculation of sPAB was done by multivariate regression. A $p < 0.05$ was considered significant. SPSS 17 software was used for analysis.

RESULTS: Correlation analyzes revealed that PA/DA ($R=0.57$, $p<0.001$) and RDPA ($R=0.33$, $p=0.015$) were moderately related to sPAP (Figure 2). The effect of the change in PA/DA and RDPA to the change in sPAP was assessed with multivariate regression analysis. The best fit equation was as $sPAP=13.66+PA/DA \times 24.06+RDPA \times 3.82$. Subgroup analysis of 40 (%70.2) patients with precapillary PH showed that PA and RDPA were more strongly related to sPAP. These variables were not related to echocardiographic sPAP in patients with postcapillary PH.

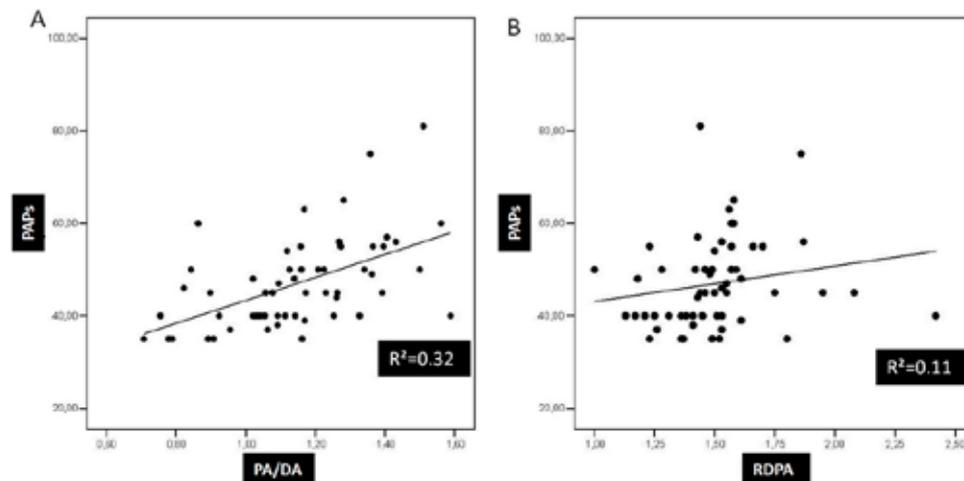


Figure 2: Scatterplot graphs showing significant correlation between systolic pulmonary artery pressure (PAPs) and horacic metrics including A: pulmonary artery (PA)/descending artery (DA) diameter ratio and B: right descending pulmonary artery (RDPA) diameter.

DISCUSSION: Among the CT findings predicting mean PAP measured by right heart catheterization in the literature, PA diameter, PA/AA ratio, segmental pulmonary PA/segmental bronchus diameter ratio are among the most studied. For example, a threshold value of PA diameter >28.6 predicts PH (3). In addition, a PA/AA ratio >1 or segmental PA diameter/adjacent segmental bronchus diameter ratio >1.25 is in favor of PH. According to the result obtained in our study, an estimated sPAB value can be calculated with a moderate correlation by combining the PA/DA ratio and RDPA diameter in PH patients. In the subgroup analysis, the calculation of sPAB with these two criteria could be obtained with higher correlation power in patients with precapillary PH. In the postcapillary PH (due to left heart pathology) patient group, PA/DA and Right DPA criteria were not associated with sPAB.

Our study has some limitations. The most important ones are the retrospective design of the study and a patient group with heterogeneous causes of PH. The presence of PH in the patients was determined by the echocardiographic estimated sPAB, and the fact that right heart catheterization was not performed in the patients is another limitation.

CONCLUSION: sPAP can be calculated with contrast enhanced thorax CT in a group of patients with heterogenous PH etiologies. This calculation may especially be important in patients with limited echocardiographic image quality to determine patients who need right heart catheterization. These data should be supported by further studies with higher number of patients undergoing right heart catheterization including specific PH subgroups.

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OP-023 THE ROLE OF THE FRONTAL QRS-T ANGLE IN THE DIAGNOSIS OF ACUTE PULMONARY EMBOLISM

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Background: Acute pulmonary embolism (APE) is a cardiovascular disease that causes mortality and long-term morbidity. There is no clear ECG parameter supporting the diagnosis of acute pulmonary embolism in current guidelines and studies. ECG parameters used in the diagnosis of cardiovascular diseases are mostly caused by the depolarization-repolarization change triggered by ischemia in the myocardium. Frontal QRS-T angle is a parameter that reflects myocardial depolarization-repolarization changes. Our aim in this study is to reveal the role of the Frontal QRS-T angle in the diagnosis of APE.

Method: 385 patients who underwent pulmonary CT angiography with the suspicion of AE in clinical and laboratory results were included in the study. As a result of pulmonary CTA, 195 patients had a diagnosis of APE. There was no embolism in the pulmonary CTA of 190 patients. The clinical information of the patients and their ECGs at the emergency service application were obtained from their files. Frontal QRS-t angle of all patients was calculated from 12-lead ECG.

Results: The number of female patients in the study population was 203 (52.3%), the mean age of the study population was 55.69 ± 15.93. There was a statistically significant difference in age and gender between the patient groups with and without APE, the group with embolism was older, the number of men in the group with embolism was higher than the number of women. While there was no significant difference in terms of comorbidity in terms of HT, DM, ASCVD, CVD, HF, the number of COPD was higher in the group without embolism. The frontal QRS-T angle was significantly higher in the group with acute pulmonary embolism. In the ROC analysis performed for the frontal QRS-T angle with the diagnosis of embolism, the value of 28.50 for the frontal QRS-T angle was found to be the cut-off value for the diagnosis of embolism with 57.6% sensitivity and 61.1% specificity (area under the ROC curve 0.604, Confidence interval 95%: 0.548 - 0.660, p <0.001).

Conclusion: Possible hypoxia due to acute pulmonary embolism and hemodynamic effects on the right ventricle affect the frontal QRS-T angle on the ECG. High values in the frontal QRS-T angle can be used in the diagnosis of acute pulmonary embolism.

Keywords: Pulmonary embolism, ECG, frontal QRS-T angle

Tablo 1

	Without APE n = 190	With APE n = 195	P value
Age	52.18±15.33	59.06±15.80	<0.001
Gender			
Male	74 (%38,9)	111 (%56,1)	
Female	116 (%61,1)	87 (%43,9)	0.001
Hypertension	57 (%30,0)	50 (%25,3)	0.308
Diabetes Mellitus	36 (%18,9)	27 (%13,6)	0.156
ASCVD	17 (%8,9)	18 (%9,1)	0.961
HF	3 (%1,6)	4 (%2,0)	0.744
COPD	58 (%30,5)	23 (%11,6)	<0,001
CVD	3 (%1,6)	4 (%2,0)	0.744
Heart rate (b.p.m)	85.52±13.58	97.07±16.59	<0.001
QRS (msec)	89.58±15.86	89.12±10.13	0.736
QTcorrected (msec)	410.77±45.08	406.75±36.88	0.338
f-QRS-T angle (°)	30.38±27.25	41.30±33.19	<0.001

Table 1

OP-024 RIOCIGUATE MAY BE EFFECTIVE IN COMBINED THERAPY IN PULMONARY HYPERTENSION DUE TO CONNECTIVE TISSUE DISEASE

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Introduction: Riociguat is one of the new options in combination therapy in group 1 pulmonary hypertension patients.

Case: A 74-year-old female patient was admitted to the cardiology outpatient clinic with dyspnea in 2013. Routine tests were requested for the patient. Echocardiography (ECHO) revealed ejection fraction (EF) 60%, minimal mitral regurgitation, mild to moderate tricuspid regurgitation, pulmonary arterial pressure (PAP): 4.1/67mmHg, and right heart chamber diameters dilated at the upper limit. Electrocardiography was in sinus rhythm. Physical examination (PE) revealed pretibial edema and skin telangiectasias in both legs. In addition, the patient complained of joint pain and intermittent bruising on his hands. Blood tests are all normal except NtproBNP (1583pg/ml). The six-minute walking test (SMWT) result was 250 meters. It was considered as New York heart association functional classification(NYHA) 3. Coronary angiography(CA) and right heart catheterization(RHC) were planned. In invasive evaluation, normal coronary arteries and mean-PAP 30 mmHg were found. Etiology was investigated due to high PAP. The patient with Raynaud's phenomenon and telangiectasia was consulted with rheumatology when the measured markers ANA and SCL70 were positive for etiology investigation then the patient was diagnosed with scleroderma. The patient was started on bosentan monotherapy with the diagnosis of group 1 pulmonary hypertension. The patient, who had been hospitalized several times in the intervening period, was admitted to our clinic 6 times in the last year with increased complaints. At the first hospitalization due to dyspnea and decompensation, NtProBNP:15027pg/ml, SMWT 200 meters was detected and syncope occurred once, the patient was NYHA 3 symptomatic. Thereupon, sildenafil treatment was started in addition to bosentan. After starting combined therapy, the patient was admitted to our clinic 5 more times. At the last admission, the patient had NTproBNP:10076pg/ml, syncope occurrence once, NYHA3, and an SMWT were measured as 210 meters. ECHO: EF%60, third-degree tricuspid regurgitation, PAP:4.4/78 mmHg, and right heart chambers were large. It was planned to stop sildenafil and add riociguat to bosentan treatment. During the controls, the riociguat level was gradually increased and 2.5 mg was given 3 times a day. At the 1st month control after the combination of bosentan and riociguat; It was evaluated as NYHA2, PE was normal and the SMWT was measured as 320 meters. In the control 3 months after the combination of bosentan and riociguat; The SMWT was evaluated as 385 meters and NYHA 2, no recurrence of syncope, no dyspnea, NTproBNP:414 pg/ml and ECHO PAP:4.0/66 mmHg.

Conclusion: The combination of endothelin receptor antagonist and riociguat produces rapid clinical improvements in a patient with group 1 pulmonary arterial hypertension due to connective tissue disease.

Keywords: pulmonary hypertension, riociguat, bosentan

OP-025 CONTROLLING NUTRITION STATUS SCORE AND MORTALITY IN PATIENTS WITH ACUTE PULMONARY EMBOLISM ADMITTED TO THE INTENSIVE CARE UNIT

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Objective: Acute pulmonary embolism is a major cause of death worldwide. In hospitalized patients, it is the third cause of cardiovascular mortality after acute myocardial infarction and stroke. Malnutrition is an important clinical condition that affects especially elderly patients, and it is potentially treatable. Lately, Malnutrition is related to mortality in patients with coronary artery disease, congestive heart failure, valvular heart disease, and hypertension. A simple parameter, Controlling Nutritional Status (CONUT) score has been proposed to screen for nutritional and inflammatory status. We investigated the CONUT score and short-term mortality of acute pulmonary embolism in the patients who were admitted to the intensive care unit (ICU).

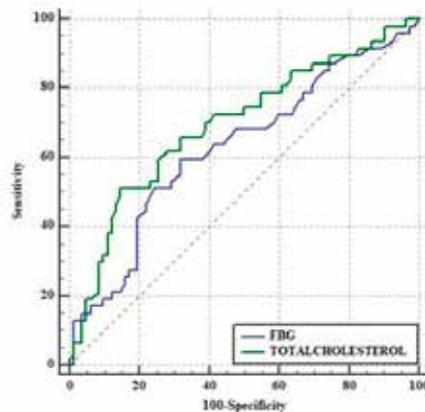
Material-Methods: We retrospectively investigated 129 patients (48 male, 81 female) with acute pulmonary embolism in patients with acute pulmonary embolism who admitted to ICU. The patient data including demographics, comorbid risk factors, and laboratory data have been obtained from medical records. CONUT score has been calculated using serum albumin level, lymphocyte count, and total cholesterol. (Albumin g/dL: $\geq 3.5=0$, $3-3.49=2$, $2.5-2.99=4$, $<2.5=6$ point; Total kolesterol mg/dl: $>180=0$, $140-179=1$, $100-139=2$, $<100=3$ point; lenfosit 109/L: $\geq 1600=0$, $1200-1599=1$, $800-1199=2$, $<800=3$ point). Also, we considered Pulmonary Embolism Severity Index (PESI) score in our analysis.

Result: In patients, total mortality was 48 out of 128 (%36.4). A higher CONUT score was related to mortality ($p<0.001$). Patients who died have higher rates of malign disease ($p=0.001$) and chronic renal failure ($p=0.01$). Laboratory findings indicate that low levels of serum albumin ($p<0.001$), total cholesterol ($p<0.001$) and eGFR (0.016), and high levels of glucose ($p=0.014$), D-dimer ($p=0.019$) and C-reactive protein ($p=0.020$) levels related with mortality. Multivariate regression analysis showed that high glucose levels ($HR=1.006, \%95CI= 1.001-1.012, P=0.027$), high PESI score ($HR=1.980, \%95CI= 1.078-3.637, P=0.028$) and total cholesterol ($HR=0.992, \%95CI= 0.986-0.998, P=0.014$) have independently predictor of in-hospital mortality with exception of CONUT score.

Conclusion: Although a Higher CONUT score has been observed in acute pulmonary embolism-caused deaths, it was not predicted in-hospital mortality. Further large-scale prospective studies are needed to clarify this issue.

Keywords: acute pulmonary embolism, conut score, mortality

Figure 1. ROC curves of the FBG and total cholesterol for detecting the in-hospital mortality.



Cut off value of > 129 mg/dl for blood glucose has %59 sensitivity, % 68 specificity ($AUC = 0.628$; $95\% CI = 0.538 - 0.711$; $p = 0.015$) and cut off value of ≤ 161 for total cholesterol has %51 sensitivity, % 85 specificity ($AUC = 0.694$; $95\% CI = 0.607 - 0.772$); $p < 0.001$)

Table 1. Demographic, clinical and laboratory parameters of the study cohort

Variables	All population (n = 129)	Survivor (n = 82)	Non-survivor (n = 47)	p
Female gender, n (%)	81 (62.8)	53 (64.6)	28 (59.6)	0.567
Age, year	65.8±15.7	64.2±16	68.2±15	0.157
Hypertension, n (%)	89 (69)	56 (68.3)	33 (70.2)	0.820
Diabetes mellitus, n (%)	51 (39.5)	36 (43.9)	15 (31.9)	0.180
CAD history, n (%)	30 (23.3)	10 (24.4)	10 (21.3)	0.687
CHF history, n (%)	7 (5.4)	3 (3.7)	4 (8.5)	0.242
CVA history, n (%)	17 (13.2)	8 (9.8)	9 (19.1)	0.129
Malignancy history, n (%)	26 (20.2)	9 (11)	17 (36.2)	0.001
Presence of COPD, n (%)	20 (15.5)	14 (17.1)	6 (12.8)	0.515
Presence of CRF, n (%)	28 (21.7)	12 (14.6)	16 (34)	0.010

Variables	All population (n = 129)	Survivor (n = 82)	Non-survivor (n = 47)	p
Smoking, n (%)	35 (27.1)	24 (29.3)	11 (23.4)	0.471
Atrial fibrillation, n (%)	12 (9.3)	6 (7.3)	6 (12.8)	0.305
DVT, n (%)	72 (55.8)	44 (53.7)	28 (59.6)	0.515
High PESI (>125 point), n (%)	45 (34.9)	21 (25.6)	24 (51.1)	0.004
PAPs, mmHg	43.2±13.4	38.2±9.9	51.7±14.2	<0.001
Glucose, mg/dL, [IQR]	136.5±48.7	128.2±41.3	149.9±56.9	0.014
eGFR, mL/min/1.73m ²	74.2±29.7	78.8±27.1	65.9±31.8	0.016
Albumin g/dl	3.5±0.7	3.6±0.6	3.2±0.7	<0.001
Total Cholesterol mg/dl	194.8±52.6	207.1±49.7	174.2±50.4	<0.001
Haemoglobin, g/dL	11.9 ± 1.9	11.9 ± 1.7	11.8±2.2	0.830
Neutrophil, 103/μL, [IQR]	6.5 [4.6-9.3]	5.7 [4.5-8.1]	7.8 [5.2-11.6]	0.057
Lymphocyte, 109/L, [IQR]	1.7 [1.1-2.4]	1.9 [1.3-2.4]	1.6 [1.0-2.1]	0.094
Platelet, 109/L, [IQR]	232 [180-297]	232 [188-308]	231 [165-285]	0.297
CRP, mg/L, [IQR]	40 [15-100]	29 [14-84]	51 [20-134]	0.020
CONUT score, [IQR]	2 [1-4]	2 [0-3]	4 [1-7]	<0.001
Cardiac troponin, ng/mL, [IQR]	24.7 [16.2-118.5]	22.4 [8.8-96.5]	39.5 [23.0-177.7]	0.707
D-dimer, ng/mL, [IQR]	6.5 [3.7- 8.6]	6.0 [3.6- 8.1]	7.9 [4.0- 9.1]	0.019

Abbreviations: CAD, coronary artery disease; CHF, chronic heart failure; CVA, cerebrovascular accident; COPD, chronic obstructive pulmonary disease; CRF, chronic renal failure; DVT, deep vein thrombosis; PESI, pulmonary embolism severity index; PAPs, pulmonary artery systolic pressure; eGFR, estimated glomerular filtration rate; CRP, C-reactive protein

OP-026 EVALUATION OF CHANGES IN VENTRICULAR REPOLARIZATION PARAMETERS IN MORBIDLY OBESE PATIENTS UNDERGOING BARIATRIC SURGERY

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Background and Objectives: Weight loss after bariatric surgery has been associated with reduced cardiovascular mortality and overall mortality in obese patients. In this study, we aimed to analyze the changes of pre-operation and post-operation ventricular arrhythmia predictors of patients who underwent bariatric surgery.

Methods: The study included 58 patients who underwent bariatric surgery. We measured QTmax, QTmin, QRS, JT, and Tp-e intervals and we estimated Tp-e/QT max, Tp-e/QTc max, Tp-e/JT, Tp-e/JTc rates, QTc max, QTc min, cQTd, and JTc intervals of both pre-op and post-op sixth months ECGs.

Results: Heart rate ($81 \pm 13,19$ bpm vs. $68,71 \pm 10,55$ bpm, $p < 0,001$), PR ($154,69 \pm 23,67$ vs. $151,36 \pm 19,48$ bpm, $p = 0,033$) QT max ($409,26 \pm 35,60$ ms vs. $392,40 \pm 32,2$ ms, $p < 0,001$), QTc max ($446,01 \pm 30,89$ ms vs. $407,64 \pm 28,76$ ms, $p < 0,001$), QTc min ($395,45 \pm 30,87$ ms vs. $375,36 \pm 28,49$ ms, $p < 0,001$), cQTd ($50,56 \pm 7,12$ ms vs. $32,28 \pm 6,92$ ms, $p < 0,001$), JT ($283,5 \pm 26,76$ ms vs. $276,39 \pm 24,77$ ms, $p < 0,001$), JTc ($320,25 \pm 24,52$ ms vs. $291,62 \pm 23,51$ ms, $p < 0,001$), Tp-e ($79,25 \pm 13,92$ ms vs. $67,28 \pm 12,69$ ms, $p < 0,001$), Tp-e/QT max ($0,19 \pm 0,034$ vs. $0,17 \pm 0,033$, $p < 0,001$), Tp-e/QTc max ($0,18 \pm 0,032$ vs. $0,17 \pm 0,032$, $p < 0,001$), Tp-e/JT ($0,28 \pm 0,046$ vs. $0,24 \pm 0,047$, $p < 0,001$) and Tp-e/JTc ($0,25 \pm 0,045$ vs. $0,23 \pm 0,045$, $p < 0,001$) values, which were close to the upper limit in the pre-operative period, decreased statistically significantly in the postoperative sixth month. (Reviewer1)

Conclusion: The results of our study have shown that weight loss in patients after bariatric surgery has positive effects on the regression of ventricular repolarization parameters and possible development of ventricular tachycardia.

Keywords: Obesity, surgery, electrocardiogram, ventricular arrhythmia, repolarization

Electrocardiographic findings of the study population.

(n = 58)	Pre-op	Post-op (6. month)	p value
Heart rate, bpm	$81 \pm 13,19$	$68,71 \pm 10,55$	$<0,001^*$
PR; ms	$154,69 \pm 23,67$	$151,36 \pm 19,48$	$0,033^*$
QRS; ms	$92,86 \pm 13,39$	$92,36 \pm 12,09$	$0,452$
QT max; ms	$409,26 \pm 35,60$	$392,40 \pm 32,2$	$<0,001^*$
QT min; ms	$358,7 \pm 35,49$	$360,12 \pm 33,38$	$0,384$
JT; ms	$283,5 \pm 26,76$	$276,39 \pm 24,77$	$<0,001^*$
QTc max; ms	$446,01 \pm 30,89$	$407,64 \pm 28,76$	$<0,001^*$
QTc min; ms	$395,45 \pm 30,87$	$375,36 \pm 28,49$	$<0,001^*$
JTc; ms	$320,25 \pm 24,52$	$291,62 \pm 23,51$	$<0,001^*$
cQTd; ms	$50,56 \pm 7,12$	$32,28 \pm 6,92$	$<0,001^*$
Tp-e; ms	$79,25 \pm 13,92$	$67,28 \pm 12,69$	$<0,001^*$
Tp-e/QT max	$0,19 \pm 0,034$	$0,17 \pm 0,033$	$<0,001^*$
Tp-e/QTc max	$0,18 \pm 0,032$	$0,17 \pm 0,032$	$<0,001^*$
Tp-e/JT	$0,28 \pm 0,046$	$0,24 \pm 0,047$	$<0,001^*$
Tp-e/JTc	$0,25 \pm 0,045$	$0,23 \pm 0,045$	$<0,001^*$

bpm: beat per minute, ms: millisecond, QT max: maximum QT, QT min: minimum QT, QTc max: corrected QT max, QTc min: corrected QT min, JT interval (JT): were measured from the end of the QRS complex (J point) to the end of the T wave (JTend interval), JTc: corrected JT interval, cQTd: cQT dispersion (QTd) was determined as the difference between the maximum and minimum QTc interval, Tp-e: T peak and end interval.

OP-027 P WAVE AND QT DISPERSION IN PATIENTS WITH SUBCLINICAL HYPOTHYROIDISM

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Aim: Subclinical hypothyroidism, or mild thyroid failure, is described as a state in which the thyroid stimulating hormone (TSH) level is increased with normal free T3 and T4 levels. Its prevalence ranges from 4% to 10% in the general population and increases with age. It is mostly asymptomatic, but affected individuals may show symptoms such as those seen in overt hypothyroidism, including tiredness, weight gain, and constipation. Deficiency of thyroid hormones is associated with increased cardiovascular mortality, arrhythmia, autonomic disturbances, and heart failure. However, the impact of subclinical hypothyroidism on the cardiovascular autonomic system remains a matter of debate. The aim of the present study was to assess QT and P wave dispersion (PWD) in subclinical hypothyroid patients.

Material-Method: A total of 125 subjects who applied to our clinic between August and December 2020 were included in the study. The study group consisted of 60 subclinical hypothyroid patients, and the control group consisted of 66 individuals whose age and sex matched subjects. All subjects underwent two-dimensional echocardiographic and electrocardiographic examination. QT dispersion (QTD), corrected QTD (QTDC) and PWD were calculated.

Results: Parameters such as age, gender, smoking habit, systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate were not different between two groups. Max QT, min QT, QTD, max QTc, minQTc, QTDC, P max, P min and PWD and TSH level were found to be significantly higher in study group in contrast to control group (Table 1).

Serum TSH levels were positively correlated with max QT ($r=0.538$, $p<0.001$), min QT ($r=0.221$, $p=0.013$), QTD ($r=0.383$, $p<0.001$), max QTc ($r=0.472$, $p<0.001$), min QTc ($r=0.259$, $p=0.003$), QTDC ($r=0.363$, $p<0.001$), P max ($r=0.490$, $p<0.001$), P min ($r=0.174$, $p=0.052$) and PWD ($r=0.430$, $p<0.001$) intervals.

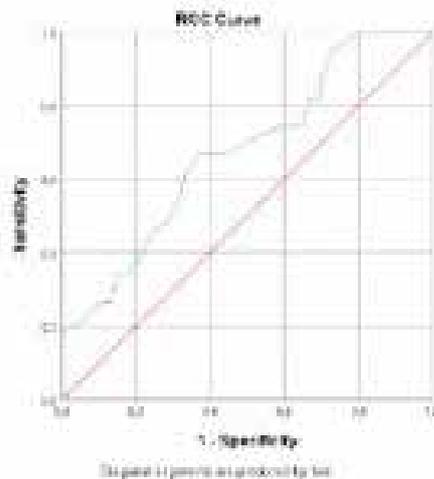
According to multivariate logistic regression analysis, PWD (OR: 1.046, 95% CI: 1.03–1.090, $p=0.034$) was an independent predictor for the presence of subclinical hypothyroidism. ROC curve analysis demonstrated that PWD value of 49.5 ms predicted subclinical hypothyroidism with %66.7 sensitivity and % 62.1 specificity (AUC:0.673, $p=0.001$, %95 CI: 0.580-0.766) (Figure 1).

Conclusion: PWD, and QTD are simple and easily obtainable parameters from surface ECG, reflecting variations in atrial and ventricular repolarizations and denoting information about the autonomic nervous system and cardiac functioning. These simple parameters may be used in clinical practice to evaluate subclinical hypothyroid patients who are at risk of arrhythmias and paroxysmal atrial fibrillation.

Keywords: Subclinical hypothyroidism, P wave dispersion, QT dispersion

FIGURE 1

Figure 1: ROC analysis of PWD for predicting subclinical hypothyroidism



ROC analysis of PWD for predicting subclinical hypothyroidism

TABLE 1

	Control group (n=66)	Study group (n=60)	p
Age (yrs)	37.56 (31.75 - 43.00)	36.87 (31.25 - 41.25)	0.839
Gender (n, %)			0.515
Female	39 (59)	32 (53)	
Male	27 (41)	28 (47)	
Smoking n (%)	10 (15)	9 (15)	0.981
BMI (kg/m ²)	24.555 (23.135 - 25.797)	24.600 (23.14 - 25.69)	0.893
SBP (mmHg)	120.00 (100.00 - 135.00)	120.00 (90.00 - 130.00)	0.502
DBP (mmHg)	74.50 (69.25 - 84.00)	75.00 (62.00 - 84.00)	0.955
HR (bpm)	78.00 (74.00 - 89.25)	74.00 (69.25 - 84.00)	0.073
MAX QT (ms)	368.00 (354.75 - 379.25)	386.50 (355.00 - 379.00)	<0.001
MIN QT (ms)	332.86±13.382	341.67±12.297	0.039
QTD (ms)	37.42±13.660	45.52±13.351	<0.001
MAX QTc (ms)	386.5 (378.75 - 395.75)	408.00 (379.00 - 395.00)	<0.001
MIN QTc (ms)	340.26±13.087	352.28±14.082	<0.001
QTD c (ms)	46.35±14.623	56.90±13.605	0.003
P MAX (ms)	86.00 (84.00 - 104.00)	98.00 (84.00 - 104.00)	0.003
P MIN (ms)	53.48±12.045	50.45±13.682	0.003
PWD (ms)	36.41±12.593	48.82±12.861	<0.001
TSH (mU/l)	3.13 (1.862 - 3.47)	8.95 (6.37 - 11.20)	<0.001
Free T3 (mU/l)	3.182±0.727	3.365±0.606	0.086
Free T4 (mU/l)	1.275 (1.122 - 1.427)	1.260 (1.08 - 1.32)	0.378

Table 1: Clinical characteristics and ECG findings of the groups.

OP-028 EVALUATION OF ARRHYTHMIA FREQUENCY WITH HOLTER ELECTROCARDIOGRAPHY IN PREGNANT PATIENTS WITH PALPITATION COMPLAINTS

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Objective: Palpitations are common during pregnancy. Although this condition sometimes affects quality of life, it is generally benign in structurally normal hearts and usually does not require treatment. Physiological changes during pregnancy may be the cause of palpitations. During pregnancy, blood volume increases by an average of 50% (1,2). This can cause atrial stretch, which may be important for arrhythmogenesis (1). The hormonal change that occurs during pregnancy can exert a proarrhythmic effect on myocardial tissue. The increased sympathetic outflow that occurs during pregnancy also likely contributes to proarrhythmic states (1,3)

So, during pregnancy, the risk of supraventricular and ventricular arrhythmias are increases. Hormones, atrial stretch, and automatic tonus change are the main arrhythmia mechanisms (2). Therefore, pregnant patients can frequently apply to cardiology outpatient clinics with the complaint of palpitations. The aim of our study is to determine the underlying etiology and the frequency of arrhythmia by holter electrocardiography in pregnant patients admitted to the cardiology outpatient clinic with the complaint of palpitations.

Methods: Sixty-four pregnant patients who applied to the cardiology outpatient clinic with the complaint of palpitations and had a holter ECG between January 2019 and March 2021 were included in the study. This study was a retrospective study. Holter ECG, imaging and biochemical data of the patients were analyzed through the hospital registry system.

Inclusion criterias

- >18 years old
- Pregnant women who applied to the cardiology department with the complaint of palpitation
- Patients who had no signs of arrhythmia in their baseline electrocardiography
- Patients who had a 24-hour Holter ECG

Exclusion criterias

- Patients younger than 18 Years
- Severe kidney and liver failure
- Patients with known any arrhythmias
- Patients who cannot undergo optimal echocardiographic examination
- Patients whose Holter electrocardiography cannot be evaluated optimally
- Patients who did not give informed voluntary consent

Statistical analysis

IBM SPSS Statistics 25.0 Program was used. Numerical variables are given as mean and standard deviation or median (min -max). Categorical variables are given as numbers (n) and percentages (%).

Results: The mean age of the patients was 29.11 (min 20- max 46) years, mean SBP 118.70 (min 90-max 165) mmHg and mean pulse was 96.17 per min (min 57- max 138). Nine (14.1%) of the patients were cigarette users. Hypertension was present in 11 (17.2%) patients in terms of comorbid diseases, and 7 (10.9%) of these hypertensive patients had gestational hypertension. In addition, 10 (15.6%) patients had thyroid disease, 3 (4.7%) patients had DM. Anemia, one of the etiologies of arrhythmia, was detected in 34.4% of the study population.

Among the biochemical parameters, the mean hemoglobin (Hb) value is 11.72 (\pm 1.69) g/dl and the mean TSH value is 1.31 (\pm 0.75). The mean ferritin value was 26.35 (\pm 24.56) and the mean vitamin B12 value was 250.37 (\pm 108.7). Average LVEF of the patients was 63.09%, moderate-severe MR was 4.7%, moderate-severe MS was 1.6% and moderate-severe TR was 9.4%.

The study population consisted of patients who could not detect any arrhythmia on ECG. The most common arrhythmias detected in Holter ECG were VES at a rate of 21.9% and SVT at a rate of 14.1%. The frequency of PAF was 4.7%. Non-sustained VT was detected in 2 patients. 23.4 of the patients were using beta-blocker drugs. The rate of using iron preparations was 23.4% and the rate of using B12 vitamin was 18.8%.

Discussion: Pregnancy is an arrhythmogenic condition in which arrhythmias may occur for the first time or exacerbation of a pre-existing condition may occur (4). Physiological changes associated with normal pregnancy, such as increased heart rate, decreased peripheral resistance, increased stroke volume, hormonal changes, and psychological stresses, as well as increased sympathetic activity, are considered the most common triggers of arrhythmias in pregnant women (4-6).

Ectopic beats and non-sustained arrhythmias are encountered in more than 50% of pregnant women who are investigated for palpitations that are generally benign and do not require treatment (7,8). The frequency of arrhythmia in our study was 50.12%.

Ventricular tachycardias (VT) may occur as a new onset arrhythmia during pregnancy or may be exacerbated by pregnancy. This situation is worrisome in terms of both mother and fetus health (9). Ventricular tachycardia can occur at any time during pregnancy. In a study conducted on 11 pregnant women with new onset of VT during pregnancy, it was shown that the onset of VT was evenly distributed in the three trimesters and completely disappeared in the postpartum period (10). In our study, we had two patients with non-sustained VT.

A routine 24-48 hour Holter monitor is helpful in catching frequently occurring paroxysmal arrhythmias (6,9). Paroxysmal supraventricular tachy-

cardia (PSVT) is the most common tachyarrhythmia in pregnancy presenting with palpitations, dyspnea and presyncope (11). Thyroid dysfunction, electrolyte imbalance, anemia, anxiety, toxic drug use, and thromboembolism should be ruled out before the diagnosis of PSVT is made (11). Anemia was observed in 34.4% and thyroid disease in 15.6% of the patients included in our study. PSVT was detected with a rate of 14.1%.

Conclusion: Detecting the underlying arrhythmia in pregnant patients with palpitation is important for both mother and fetus health. The incidence of arrhythmias that should be treated in this patient group is too high to be ignored. Even if the electrocardiography is normal, rhythm monitoring with Holter ECG is very important for the detection of underlying silent arrhythmias in pregnant women with palpitations

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Table 1. Demographic, clinical, laboratory, imaging and holter ECG data of the patients

Demographic feature		Comorbidities	n (%)
Age, year, mean±st	29.11±5.31	Hypertension	11 (17.2)
Systolic BP, mean±st	118.7±16.4	Gestational hypertension	7 (10.9)
Diastolic BP, mean±st	69.80±10.91	PTE	1 (1.16)
Smoking, n(%)	9 (14)	Stroke/TIA	1 (1.16)
Laboratory and imaging findings	mean±st	Thyroid disease	10 (15.6)
Hemoglobin		Parathyroid disease	2 (3,1)
TSH	1.31±0.75	Asthma	2 (3,1)
Ferritin	26.35±24.56	Anemia	22 (34,4)
B12	250.37±108.7	Diabetes mellitus	3 (4.7)
Wbc	10.15±3.26	Hyperlipidemia	6 (9.4)
LDL	102.9±26.25	Holter ECG findings	n (%)
LVEF	63.09±4.27	SVT	9 (14.1)
Moderate-severe MR, n(%)	3 (4.7)	VES 14 (21.9)	14 (21.9)
Moderate-severe MD, n (%)	1 (1.6)	Non-sustained VT	2 (3.1)
Treatments	n (%)	VT	0 (0)
Beta blockers	15 (23.4)	Pause >3 seconds	0 (0)
Non-DHP CCB	4 (6.3)	Atrial fibrillation	0 (0)
DHP CCB 6 (9.4)	6 (9.4)	PAF	3 (4.7)
Iron preparations	15 (23.4)	Atrial tachycardia	1 (1.6)
Vitamin B12	12 (18.8)	Premature atrial contraction	3 (4.7)

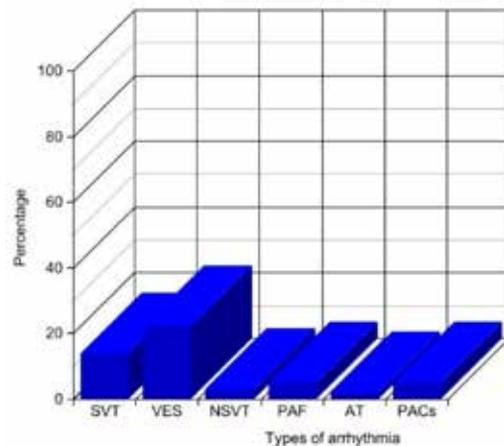


Figure 1. Types and frequency of arrhythmia detected by holter electrocardiography in pregnant patients with palpitation complaints

OP-029 TPEAK-TEND INTERVAL, AND TP-E/QT RATIO IN PATIENTS WITH ESSENTIAL TREMOR

Emine Altuntaş

Sancaktepe Şehit Prof. Dr. İlhan Varank Eğitim ve Araştırma Hastanesi

Recent studies have been accepted as electrocardiographic markers for the evaluation of transmural dispersion of repolarization, QT interval (QT) in arrhythmogenesis, peak-to-end interval time of the T wave (Tp-e) and Tp-e / QT ratio, myocardial repolarization and arrhythmogenesis. These markers constitute a relatively new contribution to the field of non-invasive electrocardiology. Increased QT, QTc Tp-e, Tp-e / QT and Tp-e / QTc ratio reflect tendency to arrhythmias(1-6).

Essential tremor (ET) affects approximately 4% of adults and its prevalence increases with age. Although the main cause is unknown, it may be a syndrome with many causes. Familial transmission is high. However, the transition pattern and genetic basis are unclear. Essential tremor is always a bilateral but symmetrical kinetic and postural tremor of the upper limb, head, and voice. Although essential tremor is a motor neuron disease, its relationship with the autonomic nervous system is not clear (7-10). In this study, it was aimed to determine whether there is a connection between the autonomic nervous system and ET by investigating the relationship between ET and Tp-e.

The study was retrospective and included 40 healthy controls and 40 patients with newly detected essential tremor, who applied to the cardiology outpatient clinic with any complaint.

The age range of the participants was 18-65 years. Pregnancy, any chronic disease are exclusion criteria from the study. Electrocardiography (ECG), transthoracic echocardiography and laboratory data of the cases were recorded. Patients were compared demographically. The average age of the ET group was 33.7 years; The control group was 33,65 years and there was not a statistically significant difference between the groups ($p > 0.05$). The groups were also similar in terms of smoking and gender. The cases were also compared in terms of lipid parameters. While the groups were similar in terms of total cholesterol, low density lipoprotein and triglyceride; the high density lipoprotein was higher in the ET group ($p = 0.001$). Participants were compared in terms of electrocardiographic data. All cases were in sinus rhythm. The groups were similar in terms of QT, QTc, Tp-e, Tp-e / QT and Tp-e / QTc ratio ($p > 0.05$).

In this study, it was found that QT, QTc Tp-e, Tp-e / QT and Tp-e / QTc ratios were found similar between normal individuals and ET, which is basically a motor neuron disease but thought to be related to the autonomic nervous system. The low number of cases may have led to this result.

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OP-031 THE RELATIONSHIP BETWEEN BODY MASS INDEX AND CHANGES IN VENTRICULAR REPOLARIZATION MARKERS DURING EXERCISE

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INTRODUCTION: Obesity is associated with a wide variety of electrocardiographic (ECG) abnormalities. Most of which reflect alterations in cardiac morphology and sympathetic overactivity. Cardiac arrhythmias have been described in obese subjects are often accompanied by left ventricular hypertrophy or the sleep apnea syndrome. Many of ECG abnormalities related to obesity are reversible with substantial weight loss.

During physical exercise, changes in ECG markers representing ventricular repolarization can be seen. Clinical studies have shown that these changes can represent disturbances in myocardial repolarization homogeneity; and in some cases, has been related to higher risks of ventricular tachyarrhythmias.

The aim of our study was to compare the changes in ECG markers of ventricular repolarization during exercise in healthy individuals and its relation to body mass index (BMI).

Methods: 307 young healthy individuals were included in the study. Participants were classified into two groups according to their BMI:

4. Participants with BMI value above 30 kg/m² were assigned to the obese group (n=143)
5. Participants below that level were assigned to the non-obese group (n=164).

Study participants were those who came to our out-patient clinic with atypical cardiac symptoms and gone through a routine evaluation that included clinical examination, laboratory testing, electrocardiography, echocardiography, treadmill exercise test and other tests if needed. Participants that have no history and showed no evidence of cardiovascular diseases were included in the study.

As routinely done in our clinic a baseline ECG and a post-exercise ECG were performed after the treadmill exercise test. Heart rate, T peak-T end (Tpe), QT duration, corrected QT (QTc), QT dispersion (QTd), T peak-T end/QT (Tpe/QT) and T peak-T end/corrected QT (Tpe/QTc) ratios were calculated for all participants on baseline and post-exercise ECG.

Results: There were no significant differences between both groups in terms of sociodemographic and baseline clinical characteristics (Table 1). Both groups had similar baseline heart rate levels, obese group 85.77±11.69 and non-obese group 84.72±11.24 beats per minute (p=0.21). There was statistically a significant difference between baseline and post-exercise values of QT duration, Tpe and Tpe/QT ratios in both groups (P values of the obese group respectively <0.001, <0.001, <0.001 and for the non-obese group <0.001, <0.001, <0.001) but these changes were related to changes in heart rates and were not believed to have an important clinical significance (Table 2,3).

When QT converted to corrected QT using the Bazett's formula, no differences was observed in QTc and Tpe/QTc values before and after exercise (P values of the obese group respectively 0.107, 0.453 and for the non-obese group 0.114, 0.218) (Table 2,3).

One important observation is QT dispersion values, in the obese group there were statistically significant differences between baseline and post-exercise values (P=0.002), in contrast the QTd values of the non-obese group was similar on baseline and post-exercise period (P=0.124) (Table 2,3).

Discussion: Obesity is associated with a wide variety of electrocardiographic (ECG) abnormalities. Many studies suggested that the excessive adipose tissue accumulation in the subcutaneous tissues of the chest wall (and possibly increased epicardial fat) can affect the ECG voltage signals generated by cardiac activity and are responsible of many of these changes. Another studeis suggests that most of ECG changes seen in obese individuals can reflect alterations in cardiac structure and sympathetic overactivity.

In obese subjects simple changes like the horizontal displacement of the heart can affect the ECG signals, also more complicated and significant structural and morphological changes like atrial or ventricular hypertrophy can be responsible of ECG changes. In the other hand, many studies suggested that the adipocyte-derived peptide leptin have shown to raise sympathetic nervous system activity which can affect the normal physiological cardiac rhythms.

During exercise there is also an increase in sympathetic activation which can contribute to these changes in ECG. Disturbances in myocardial repolarization homogeneity may occur which can lead to abnormalities in ventricular repolarization. Studies have shown that changes in ECG parameters of healthy individuals can represent signals of an increased risk of cardiac arrhythmias.

Many studies have examined the effects of obesity on ECG parameters at rest. The aim of our study was to compare the changes in ECG markers of ventricular repolarization during exercise in healthy individuals and its relation to BMI.

Study results showed that changes in electrocardiographic markers representing ventricular repolarization in young healthy obese and non-obese individuals during exercise was similar in many aspects. An exception is the changes in QT dispersion values which was more significant in the obese group. Further studies with larger number of participants are needed to examine the effects of these differences on the risk of developing arrhythmias.

Table 1: The comparison of sociodemographic and baseline clinical characteristics of the study groups

Variables	Obese group (n=143)	Non-obese group (n=164)	P-value
Age, years	46.74±12.52	45.88±13.61	0.73
Basal HR, b/m	85.77±11.69	84.72±11.24	0.21
Systolic BP, mmHg	121.32±13.54	122.17±15.24	0.60
Diastolic BP, mm Hg	74.88±7.45	72.56±6.81	0.09
Hemoglobin, gr/dL	14.2±1.7	14.3±2.1	0.78
TC, mg/dL	171.8±27.4	173.9±31.7	0.71
LDL, mg/dL	135.8±22.6	137.8±31.9	0.70
Triglyceride, mg/dL	137.8±31.9	139.5±41.6	0.81
HDL, mg/dL	31.7±4.8	31.9±4.7	0.82
Calcium, mg/dL	9.3±0.8	9.4±0.6	0.46
Sodium, mEq/L	140.5±1.4	140.7±1.2	0.42
Potassium, mEq/L	4.1±0.7	4.2±0.5	0.39
TSH, mIU/L	3.3±1.3	3.4±0.8	0.63

Table 2: ECG parameters of the obese group

Parameters	Baseline	Post-exercise	P value
QT	366.3 ± 19.7	332.1 ± 21.2	P <0,001
QTc	404.2 ± 25.8	409.8 ± 32.4	P = 0,107
QTd	27.7 ± 9.7	24.3 ± 9.2	P = 0,002
Tpe	91.7 ± 10.7	85.1 ± 7.8	P <0,001
Tpe/QT	0.23 ± 0.03	0.26 ± 0.09	P <0,001
Tpe/QTc	0.215 ± 0.023	0.213 ± 0.022	P = 0.453

Table 3: ECG parameters of the non-obese group

Parameters	Baseline	Post-exercise	P value
QT	356.3 ± 28.7	329.7 ± 22.1	P <0,001
QTc	390.8 ± 25.5	395.1 ± 23.7	P = 0,114
QTd	28.6 ± 8.8	26.8 ± 10.8	P = 0,124
Tpe	90.3 ± 9.8	83.4 ± 8.4	P < 0,001
Tpe/QT	0.22 ± 0.02	0.25 ± 0.02	P <0,001
Tpe/QTc	0.214 ± 0.023	0.211 ± 0.021	P = 0.218

OP-032 EFFECT OF PRONE POSITION ASPIRATION TO THE RESULT OF PERICARDIOCENTESIS

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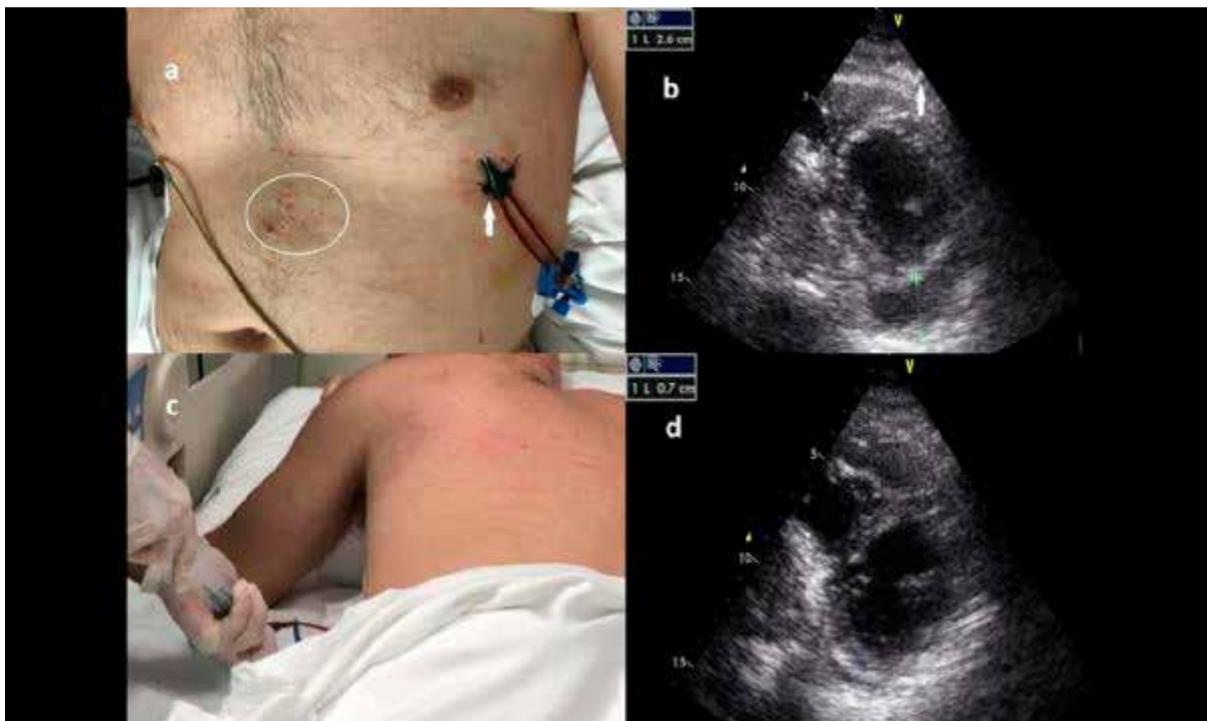
Objective: Complete drainage of pericardial effusions by pericardiocentesis cannot be always possible if much of the pericardial effusion is posteriorly located. Herein, we report the effect of prone position in improving drainage of posteriorly located residual pericardial effusion in a patient with cardiac tamponade which was successfully treated with apical pericardiocentesis after unsuccessful subxiphoid attempts.

Case: A 45-years-old man complaining of dyspnea and orthopnea due to cardiac tamponade was referred to our hospital after unsuccessful subxiphoid pericardiocentesis. He was on chemotherapy treatment for metastatic lung adenocarcinoma. Vital signs are stable except for tachycardia and tachypnea. Electrocardiogram showed sinus tachycardia and low voltage criteria. Physical examination revealed jugular venous distension and muffled heart sounds. Transthoracic echocardiography discovered massive but unequally distributed pericardial effusion leading to cardiac tamponade. Maximal accumulation of pericardial effusion was posterior and measured 36 mm. However, apical anterolateral pericardial effusion was measured 22 mm and suitable for pericardiocentesis with apical approach. Echocardiography guided apical pericardiocentesis was successfully done (Fig. 1a). 600 mL hemorrhagic effusion was drained. Catheter was left in place for 24 hours and additional 100 mL effusion was further drained. Control echocardiography revealed 26 mm residual posterior pericardial effusion and anteriorly located catheter (Fig. 1b). Patient repositioned in prone position and additional 90 mL was drained through anteriorly located catheter (Fig. 1c). Final echocardiography showed 7 mm pericardial effusion (Fig. 1d). At 6 weeks follow-up patient echocardiography showed 4 mm pericardial effusion.

Conclusion: Pericardiocentesis is a firstline treatment in cardiac tamponade and can be successfully performed as an echo-assisted or guided procedure.1-3 Our case demonstrated that residual posterior effusions encountered in case of unequally distributed pericardial effusions can be further drained with aspiration in prone position after pericardiocentesis with the help of gravity to minimize incomplete drainage and prevent the recurrence of cardiac tamponade.

Keywords: Echocardiography, pericardial effusion, pericardiocentesis

Figure 1



Arrow showed successful pericardiocentesis with apical approach. Circle showed previous unsuccessful multiple needle puncture sites (a). Echocardiography revealed 26 mm residual posterior pericardial effusion. Arrow pointing anteriorly located catheter (b). Prone position aspiration of residual pericardial effusion (c). After prone position aspiration final echocardiography showed 7 mm pericardial effusion (d).

OP-033 AN UNUSUAL CAUSE OF PERICARDIAL EFFUSION RESULTING TAMPONADE: ANGIOSARCOMA

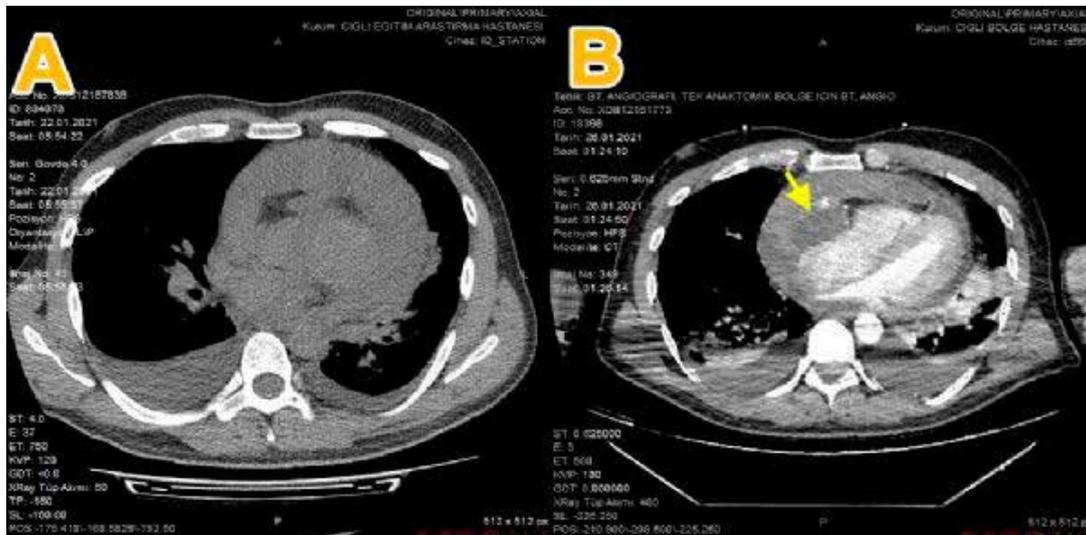
Zeynep Yapan Emren, Oktay Şenöz

İzmir Bakırçay University Çiğli Training and Research Hospital

A 40-year-old man was admitted to the hospital with three days of dyspnea and fainting. On his physical examination, he was anxious and feeling dumbness on his chest. Blood pressure was 90/60 mmHg, pulse rate was 110 per minute. Cardiac sounds were barely heard. He had a massive pericardial effusion on chest tomography (figure-1). Laboratory analyses; C-reactive protein (=200 mg/L) and D-dimer (>4400 ug/L) was high, other laboratory parameters were unremarkable. Therefore, he was referred to cardiology. Transthoracic echocardiographic (TTE) examination revealed that pericardial fluid impaired ventricle function which was consistent with cardiac tamponade. The patient was rushed to the intensive care unit. He underwent percutaneous pericardiocentesis. A 1200 cc fluid was removed from the pericardial sack. The fluid trait was exudate. After pericardiocentesis, TTE demonstrated trabecular mass around the right ventricle (figure-2), and the control chest tomography demonstrated mass around right atrium (figure-1). At the same time, the pericardial sheath was removed since the pericardial fluid was minimal. However pericardial fluid re-accumulated around the right ventricle which impeded right ventricle dilatation. Therefore, he underwent a surgical operation. Operation demonstrated serohemorrhagic fluid and hematoma on the right ventricle. And there was a mass behind the right atrium extending from the atrioventricular sulcus to the right ventricular base. Pathologic specimen showed grade 3 angiosarcoma. Chemotherapy treatment was commenced. At three months follow up he is alive.

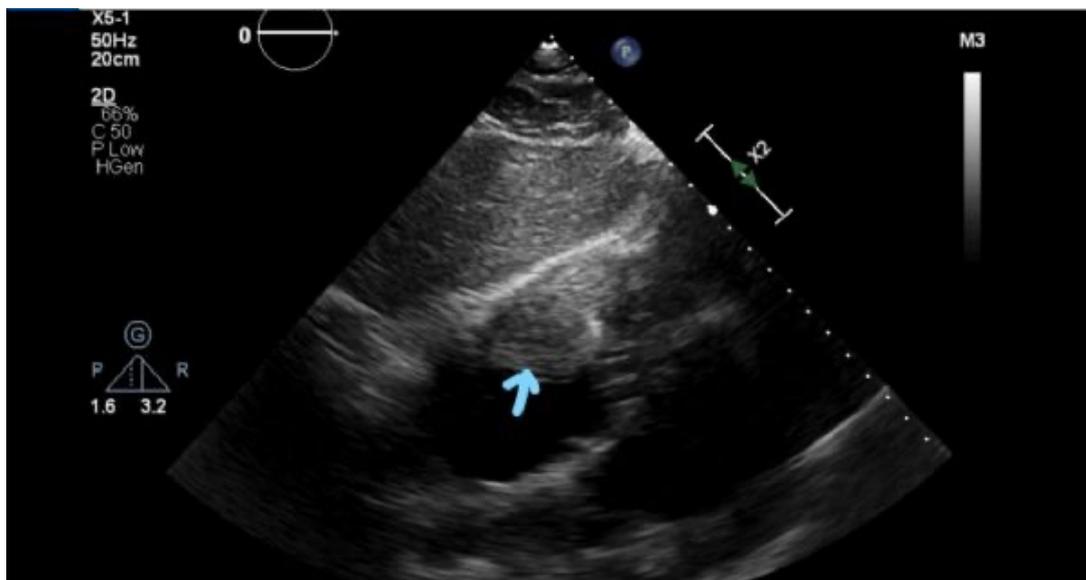
Keywords: Angiosarcoma, pericardial effusion, percutaneous pericardiocentesis

figure-1



The chest tomography showed massive pericardial effusion (A), after pericardiocentesis there was a mass located around right atrium (B)

Figure-2



After pericardiocentesis transthoracic echocardiography from subcostal view showed the mass around right atrium

OP-034 RARE BUT ALWAYS KEEP IN MIND: PURULENT PERICARDITIS

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INTRODUCTION: Purulent pericarditis has become a disease that we almost never encounter in our clinical practice modern cardiology lives. With this case, we reported the treatment management of our patient who was diagnosed with purulent pericarditis.

CASE: A 51-year-old male patient was urgently referred to our hospital due to dyspnea, hypotension, and tachycardia from the cardiology department of an external center. There is a history of primary direct stent implantation with a diagnosis of acute coronary syndrome 30 days ago. Laboratory findings from blood samples were as follows: Serum C- reactive protein 53 mg/l, white blood cell count 26380 /mm³, blood urea nitrogen 76 mg/dl, creatinine 3.7 mg/dl. Transthoracic echocardiography performed under emergency conditions revealed massive pericardial fluid compressing the right ventricle in diastole, and emergency pericardiocentesis was performed due to the serious clinical condition of the patient. 1000 cc of purulent material was drained from the patient and left for free drainage. In the analysis of the patient's pericardial fluid sample compatible with exudate (Figure I). The pigtail placed in the pericardial cavity of the patient was washed with saline in the following hours. In order not to disturb the patient's clinic, the pericardium was irrigated by aspirating back with saline, not exceeding 10 cc each time. The washing process was terminated until the material became clear. The irrigation process was completed with approximately 1500cc of SF. A similar procedure was continued for the following three days. Staphylococcus aureus growth was detected in the pericardial fluid sample culture sent on the first day of admission to our hospital and in all four blood cultures. The empirical treatment of vancomycin and meropenem was continued because the agent produced was sensitive to these agents. The treatment of the patient, whose antibiotic response was obtained and his toxicity regressed, was completed in 28 days. In the echocardiography performed before discharge, no additional pathology was found except minimal pericardial fluid (Figure II). The patient was discharged with full recovery.

DISCUSSION: Purulent pericarditis is a cardiological emergency that should always be kept in mind and known, with a mortality rate of 40% if it is not treated, and its transformation into constrictive pericarditis in the long term if treatment is not managed correctly. However, its prevalence could be increase in our country in the near future due to the increase in oncological treatments, the use of immunosuppressive drugs, the increase in the use of catheters. An effective and rapid removal of purulent material from the pericardial area, which is a closed space, is as important as the urgent initiation of effective antibiotic therapy in the treatment. The reason that there is limited data on this subject in the literature, the contribution of our case in this sense is important.

Keywords: Purulent pericarditis, fever, echocardiography

Figure I



: Exudative pericardial fluid sample of purulent nature taken from the patient

Figure II



In transthoracic echocardiography before discharge, residual minimal pericardial fluid in the posterior wall of the left ventricle, LV: Left ventricle, LA: Left atrium, Ao: Aorta, DA: Descending Aorta

OP-035 CORONARY SUBCLAVIAN STEAL SYNDROME PRESENTING WITH ACUTE CORONARY SYNDROME

Hande Seymen, Muhammed Emre Güleşir

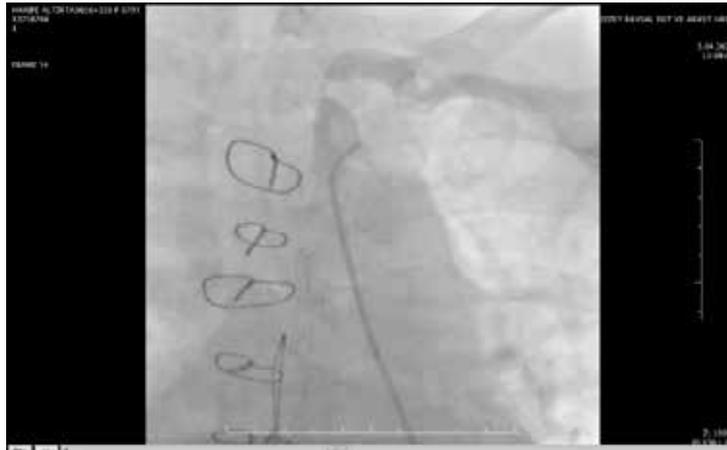
Abant İzzet Baysal Üniversitesi Eğitim ve Araştırma Hastanesi, Bolu, Türkiye

A 79-year-old woman was admitted to emergency department with acute onset chest pain. The patient had a medical history with diabetes mellitus, hypertension and coronary bypass surgery (LIMA to LAD and SVG to RCA). In echocardiographic assessment, LVEF was %55 with segmental wall motion abnormality. Cardiac troponin levels were high. An urgent coronary angiography was performed. Coronary angiography was done via femoral artery and showed %90 stenosis in the left subclavian artery (Figure 1) and good functioning grafts. An ad hoc PTA with 10x39 mm balloon-expandable stent enabled a successful revascularization of the left subclavian artery (Figure 2). At follow-up, angina had resolved. Dual antiplatelet therapy was recommended for 12 months and the patient was discharged uneventfully.

With contributions from my colleague Emre Güleşir, I'd like to present our Coronary Subclavian Steal Syndrome Case Presenting With Acute Coronary Syndrome. Our patient was 79-year old female she had heartburn for two days. She had diabetes, hypertension, coronary bypass surgery and percutaneous coronary intervention to RCA last year. She was using aspirin, clopidogrel, pantoprazole, rosuvastatin. Vital signs were normal. Echocardiographic assessment showed left atrial enlargement and inferior Wall motion abnormality. ECG was sinus rhythm with st depression in anterior precordial leads. Troponin level was elevated. Coronary angiography revealed %40-50 stenosis in distal LMCA and total occlusion in LAD at mid section, moderate stenosis in proximal Cx and total occlusion in distal part; moderate stenosis in RCA. No occlusion in saphenous graft to PDA. Left subclavian was subtotal occluded therefore LIMA perfusion was unsatisfactory. Via 6f destination long sheath subclavian stenosis crossed with 5f right Judkins catheter with a 0.035 wire and 10 x 39 mm balloon expandable stent was implanted at 10 atm and ostium was flared at 14 atm. After that LIMA-LAD graft was demonstrated to be open. In the aftermath patients complaints reduced to bare minimum.

Keywords: coronary subclavian steal syndrome, percutaneous transluminal angioplasty, subclavian stenosis, subclavian stenting

Figure 1



%90 stenosis in the left subclavian artery

Figure 2



The lesion was stented using an 10x39 mm balloon expandable stent successfully

OP-036 PERIMYOCARDITIS AS FIRST SIGN OF HIGH-GRADE B-CELL LYMPHOMA

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OBJECTIVE: Acute pericarditis is an inflammation of the pericardium that is most often caused by a viral infection and rarely can be seen with lymphomas. In this case, we describe a patient with high grade B cell lymphoma with cardiac involvement, whose first clinical finding was chest pain. We aim to emphasize the importance of close follow-up echocardiography in the cases of acute myocarditis.

CASE: A 21-year-old male patient, who had no known history of chronic disease or substance use, was admitted to hospital with the complaint of retrosternal, sharp, severe chest pain. When the patient was questioned in detail, it was found that he had fever and cough for two weeks. On admission, his vital signs were normal. On physical examination, rhythmic S1 and S2 on cardiac auscultation. Pericardial rub which is an audible medical sign used in the diagnosis of pericarditis was not detected. The investigations performed at the hospital were; ECG that showed a sinus tachycardia with a heart rate of 115 bpm, widespread 1 mm downsloping ST depression (Figure 1). LV chamber dimensions, functional parameters, interventricular septum and posterior wall thickness measured in the normal range by echocardiography. There were no valvular disease and pericardial effusion.

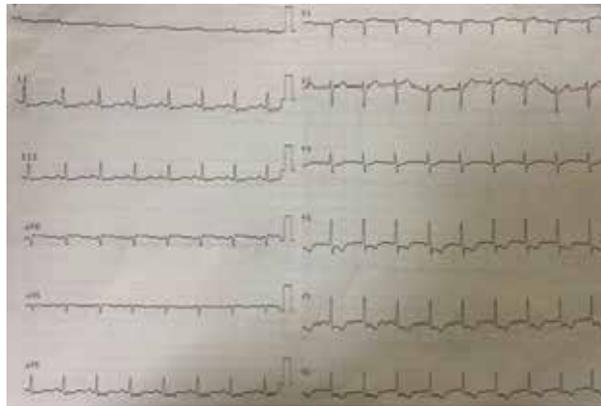
The patient was hospitalized with a prediagnosis of perimyocarditis. Angiography was not planned due to the normal LV function with no wall motion defect and serious thrombocytopenia. Because of the thrombocytopenia the hematology department was consulted with a peripheral blood smear. PBS examination confirmed leukoerythroblastic reaction. The bone marrow aspiration was performed. A control echocardiography was performed upon detection of an increase in ProBNP and Troponin I. The echo showed a marked wall thickening especially of the posterior septum (20 mm). Ef was normal and there was mild pericardial effusion without signs of tamponade (Figure 2). Because of acute increase in wall thickness and acute pericardial effusion, cardiac infiltration was considered in the patient. We started a therapy with intravenous methylprednisolone 80 mg per day. Bone marrow aspiration and biopsy confirmed High-Grade B-cell lymphoma.

High grade B-cell lymphoma chemotherapy was initiated and a decrease was found in troponin daily follow-up. The patient died after 20 days due to sepsis.

RESULTS AND CONCLUSION: Disseminated lymphoma with cardiac involvement can occur in up to 20% of patients with lymphoma. Our patient presented with chest pain and high troponin levels without the classic B symptoms of lymphoma. We couldn't determine pericardial effusion in the first echo, two days later we determine pericardial effusion and posterior wall hypertrophy due to cardiac metastasis. Therefore close follow-up with echo is very important in the cases of myocarditis with unknown cause. Pericardial effusion in patients with lymphoma might be due to direct involvement of pericardium and hematogenous spread.

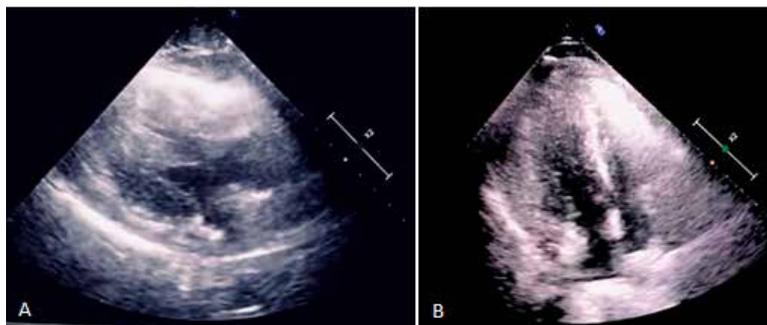
Keywords: Perimyocarditis, Non-Hodgkin's lymphoma, B-Cell lymphoma

ECG



A 12-lead ECG that showed a sinus tachycardia with a heart rate of 115 bpm, widespread 1 mm downsloping ST depression in leads I-II-III-aVF and V3-V6 (Figure 1)

echo



Parasternal long axis (A) and apical four chamber view (B); A transthoracic echocardiography showed a marked wall thickening especially of the posterior septum (20 mm). Ejection fraction was normal and there was mild pericardial effusion (10 mm) without signs of tamponade (Figure 2).

OP-039 A SYSTEMATIC REVIEW AND META-ANALYSIS ON PROGNOSTIC VALUE OF MEAN PLATELET VOLUME (MPV) IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Objective: We aimed to summarize current evidence on prognostic significance of MPV measured on admission in prediction of clinical endpoints in patients with acute coronary syndrome (ACS).

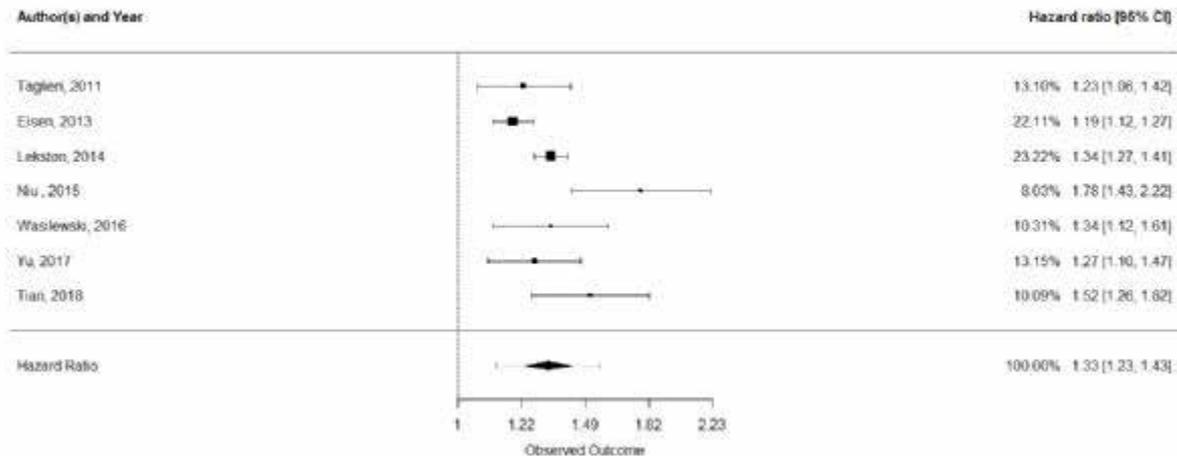
Method: The protocol of the meta-analysis was prospectively registered in PROSPERO (CRD42021230058). We searched PubMed, Scopus, Web of Science and additional databases to retrieve observational cohort studies and sub-analysis of randomised controlled trials that enrolled patients with all types of ACS. Main outcome measures were predefined as major adverse cardiovascular events (MACE) and mortality. We performed a random-effects model meta-analysis separately for studies that reported continuous and categorized endpoints. Additionally, we estimated prediction intervals to account for between-study variability while interpreting the results of the meta-analysis.

Results: The meta-analysis included 41 studies with 33443 participants. For categorized endpoints, patients with higher MPV were more likely to experience long-term MACE (hazard ratio (HR)=2.18, 95% confidence interval (CI): 1.47-3.23; odds ratio (OR) = 2.04, 95% CI: 1.53-2.73) and meet mortality endpoint (HR=2.45, 95% CI: 1.44-4.18; OR = 2.94, 95% CI: 1.70-5.09). Per-one femtoliter increase in MPV was also associated with long-term MACE (HR=1.21, 95% CI: 1.11-1.33) and mortality (HR=1.33, 95% CI: 1.23-1.43). For one-month outcomes, categorized MPV was reported to be predictive of MACE (OR = 1.85, 95% CI: 1.21-2.82) and mortality (OR = 2.23, 95% CI: 1.43-3.48). In addition, MPV as a continuous variable was also associated with one-month mortality (HR=1.31, 95% CI: 1.07-1.60). For in-hospital outcomes, categorized MPV was linked to both MACE (OR = 1.67, 95% CI: 1.24-2.26) and mortality (OR = 1.72, 95% CI: 1.36-2.16). Importantly, among all the reported effect estimates, prediction interval was only significant for continuous MPV as an independent predictor of long-term mortality (HR=1.33, 95% prediction interval: 1.13-1.57)

Conclusion: MPV as a continuous variable demonstrated its prognostic significance in prediction of long-term mortality. Further high-quality studies are of crucial importance to ascertain the value of MPV in prediction of other endpoints.

Keywords: Acute coronary syndrome, mean platelet volume, prognosis, meta-analysis, myocardial infarction

Forest-plot



Hazard ratio for long-term mortality. Mean platelet volume treated as a continuous variable

OP-040 EVALUATION OF INFLAMMATORY MARKERS IN PATIENTS WITH ISOLATED MYOCARDIAL BRIDGE: A CASE-CONTROL STUDY

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Objectives: Studies have shown that atherosclerosis (AS) is an inflammatory process and inflammatory markers are simple tools to assess inflammatory status of the patients. Myocardial bridge (MB) is commonly observed congenital heart disease that is characterized by a narrowing of a coronary vessel lumen during the systole. Because AS has been demonstrated to develop in the proximal and distal segments of most MB cases, this study aimed to investigate the well-known inflammatory markers, such as neutrophile-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-high density cholesterol ratio (MHR), in MB patients without significant coronary artery disease (CAD).

Methods: In this case-control study, consecutive patients who underwent coronary angiography (CAG) in a tertiary center were retrospectively screened. Patients who were diagnosed with acute coronary syndrome, who had a previous history of percutaneous coronary intervention or coronary artery bypass grafting, and heart failure were excluded from the study. Also, MB patients who had significant CAD (>%50) were not included. After applying exclusion criteria, the study population was consisted of 48 MB cases. The control group was consisted of age- and sex-matched 40 cases who had normal coronary arteries on CAG. MB was defined as a narrowing of the epicardial coronary artery during the systole and dilatation during the diastole without any evidence of vasospasm.

Results: Baseline characteristics, including hypertension, diabetes, hyperlipidemia, chronic obstructive pulmonary disease, smoking, and atrial fibrillation, were similar between the groups. Also, medical therapy, except Ca channel blocker treatment, were not different between the groups. There were no significant differences in two groups in terms of laboratory data. Remarkably, the inflammatory markers, including NLR, PLR, and MHR, were similar in both groups. Both univariable and multivariable logistic regression analysis showed that none inflammatory markers were predictors of MB.

Conclusion: The present investigation revealed that inflammatory markers, including NLR, PLR, and MHR, might not be used to predict MB in patients undergoing CAG

Keywords: Myocardial bridge, inflammatory markers, atherosclerosis

Table 1

Table 1 Comparison of demographic and clinical characteristics of patients according to presence of MB in coronary angiography

	Control group (n=40)	Patients with MB (n=48)	P value
Age, y	54 (42 – 61)	54 (45 – 68)	0.392
Male gender	18 (45.0%)	28 (58.3%)	0.212
Hypertension	13 (32.5%)	21 (43.8%)	0.279
Diabetes mellitus	5 (12.8%)	7 (14.6%)	0.812
Hyperlipidemia	3 (7.5%)	3 (6.3%)	1.000
COPD	2 (5.0%)	2 (4.2%)	1.000
Smoking	5 (12.5%)	10 (20.8%)	0.296
Atrial fibrillation	2 (5.0%)	3 (6.3%)	1.000
Medical treatment			
Acetylsalicylic acid	5 (12.5%)	8 (16.7%)	0.581
Beta blocker	8 (20.0%)	12 (25.0%)	0.576
Statin	3 (7.5%)	4 (8.3%)	1.000
Ca channel blocker	1 (2.5%)	37 (77.1%)	<0.001
Acc inh. /ARB	7 (17.5%)	8 (16.7%)	0.918

Abbreviations: MB, myocardial bridge; COPD, chronic obstructive pulmonary disease; ACE, angiotensinogen converting enzyme; ARB, angiotensinogen receptor blocker.

Table 2

Table 2 Comparison of laboratory variables of patients according to presence of MB in coronary angiography

	Control group (n=40)	Patients with MB (n=48)	P value
Laboratory variables at admission			
Hematocrit, %	40.9 (38.4 – 44.9)	41.0 (38.0 – 44.5)	0.693
Hemoglobin, g/dL	13.6 (12.5 – 14.7)	14.0 (13.0 – 15.0)	0.966
RDW, %	12.9 (12.4 – 13.9)	12.9 (12.0 – 13.3)	0.194
WBC count, cells/ μ L	6.9 (5.5 – 7.4)	6.5 (5.2 – 9.0)	0.977
Platelet count, mm ³	228 (190 – 279)	231 (202 – 285)	0.753
MPV, fL	10.1 (9.0 – 11.1)	10.0 (9.4 – 13.0)	0.116
PCT	0.25 (0.20 – 0.28)	0.20 (0.20 – 0.30)	0.755
Lymphocytes, mm ³	2.03 (1.59 – 2.49)	1.90 (1.70 – 2.50)	0.967
Neutrophils, mm ³	4.10 (3.27 – 5.14)	4.00 (3.42 – 5.40)	0.663
Monocytes, mm ³	0.40 (0.33 – 0.59)	0.40 (0.30 – 0.55)	0.778
Creatinine, mg/dL	0.9 (0.8 – 1.0)	0.9 (0.7 – 1.1)	0.455
Urea, mg/dL	27 (25 – 36)	29 (25 – 36)	0.675
AST, U/L	19 (16 – 25)	18 (14 – 26)	0.179
ALT, U/L	26 (18 – 33)	23 (15 – 32)	0.246
Glucose, mg/dL	100 (88 – 107)	96 (87 – 107)	0.852
Total cholesterol, mg/dL	198 (178 – 231)	193 (161 – 218)	0.246
Low density cholesterol, mg/dL	129 (114 – 150)	121 (102 – 142)	0.215
High density cholesterol, mg/dL	47 (37 – 53)	43 (37 – 55)	0.711
Triglycerides, mg/dL	126 (79 – 169)	109 (79 – 139)	0.253
NLR	2.1 (1.4 – 2.7)	2.0 (1.6 – 2.4)	0.870
PLR	113 (91 – 138)	110 (88 – 147)	0.841
MHR	9.2 (6.6 – 13.7)	1.0 (0.68 – 1.33)	0.744

Abbreviations: MB, myocardial bridge; RDW, red distribution width; WBC, white blood cell; MPV, mean platelet volume; PCT, plateletcrit; AST, aspartate amino transferase; ALT, alanine amino transferase; NLR, neutrophile-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; MHR, monocyte-to-high density cholesterol ratio.

OP-041 THE PREDICTIVE VALUE OF CRP / ALBUMIN RATIO ON THE DEVELOPMENT OF CONTRAST INDUCED NEPHROPATHY IN NON-ST-ELEVATED MYOCARDIAL INFARCTION PATIENTS WHO HAVE UNDERGONE PERCUTANEOUS CORONARY INTERVENTION

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Objective: With the development and widespread use of percutaneous coronary intervention (PCI) techniques in patients with coronary artery disease, CIN has become an important complication. There are many literature data indicating that inflammation has an important role in the pathogenesis of CIN. Biomarkers such as Crp, Crp / Albumin can help identify those at high risk in CIN patients. In our study, we aimed to examine the predictive value of CRP and albumin ratio (CAR), a newly defined inflammatory marker, in the development of CIN in NSTEMI patients who underwent PCI.

Methods: This study is a single-center, retrospective and observational study. 195 NSTEMI patients who had PCI within 24-72 hours between 2016 and 2020 were included in the study. Serum Crp, albumin and creatinine levels before coronary angiography and serum creatinine levels measured after 48-72 hours were noted. The diagnosis of CIN was accepted as a 0.5 mg / dL or > 25% increase in serum creatinine value 48-72 hours after the PCI compared to the pre-procedure. Patients were divided into two groups, with and without CIN, and their relationship with CAR levels was compared.

Statistical Analysis: Continuous variables were presented as means ± standard deviations if normally distributed and medians [interquartile ranges (IQRs)] if not normally distributed, while categorical variables were given as percentages. The chi-squared (χ^2) test was used to compare categorical variables between the groups, while the Kolmogorov-Smirnov test was employed to assess whether the variables were normally distributed. A Student's t-test or Mann-Whitney U test was used to compare the continuous variables between the groups according to whether they were normally distributed or not. In order to determine the independent predictors for development of CIN, variables found to be associated at a $p < 0.05$ level according to univariate analysis, were included in the multivariate logistic regression analysis with the results reported as the odds ratios (OR) and 95% confidence intervals (CI). The discriminatory ability of CAR, CRP and albumin in determining the development of CIN was analyzed by using the receiving operating characteristic (ROC) curve and area under the curve (AUC), accompanied by 95% confidence interval. The optimal cutoff value was also calculated from the point of maximal sensitivity and specificity by using Youden's index. To compare the discriminatory performance of the CAR, CRP and albumin, pairwise comparison of ROC curves by using DeLong et al. was also performed. The threshold of statistical significance was established at $p < 0.05$. All statistical analyses were performed using the Statistical Package for the Social Sciences version 24.0 software program (IBM Corp., Armonk, NY, USA). ROC curves of the models were compared using with MEDCALC software program (Software bvba 13, Ostend, Belgium).

Results: CIN development was found in 33 of the 195 patients included in the study. Patients who suffered CIN were older ($p = 0.009$) and had more diabetes ($p = 0.007$) and lower LVEF ($p = 0.029$). In addition, patients who developed CIN had higher CRP and CAR levels ($p < 0.001$ and $p < 0.001$ respectively), while they had lower albumin levels ($p < 0.003$). Advanced age (Odds ratio [OR] = 1.053, 95% CI = 1.010-1.098, $p = 0.015$), presence of diabetes (OR = 3.450, 95% CI 1.473-8.078, $p = 0.004$), high CRP in model 1 multivariate analysis level (OR = 1.091, 95% CI = 1.035-1.151, $p = 0.001$) and hypoalbuminemia (OR = 0.297, 95% CI = 0.114-0.777, $p = 0.013$) were independent predictors for the development of CIN. In Model 2 multivariate analysis, advanced age (1.060, 95% CI = 1.016-1.106, $p = 0.007$), diabetes (OR = 3.460, 95% CI 1.501-7.977, $p = 0.004$) and high CAR level (OR = 1.403, 95% CI 1.156-1.704, $p = 0.001$) were found to be independent predictors of CIN development. We used AUC value for diagnostic accuracies and discriminatory performances of the CAR (AUC=0.794; 95%CI=0.731-0.849; $p < 0.001$), CRP (AUC=0.770, 95% CI=0.704-0.827; $p < 0.001$) and albumin (AUC=0.666; 95%CI=0.595-0.731; $p < 0.001$) for detecting the development of CIN. The receiver operating characteristics curve analysis also revealed a cut off value of CAR greater than 2.46 predicts the development of CIN in NSTEMI patients who have done PCI with a 79.4 % sensitivity, and a 79.5 % specificity. Furthermore, a comparison of the ROC curves of CAR with those of CRP and albumin showed that CAR had a superior discriminative ability than either CRP or albumin alone for predicting the CIN development with a p value 0.011, and 0.036, respectively (Figure 1).

Conclusion: CAR not only shows the severity of inflammation but also predicts the development of CIN.

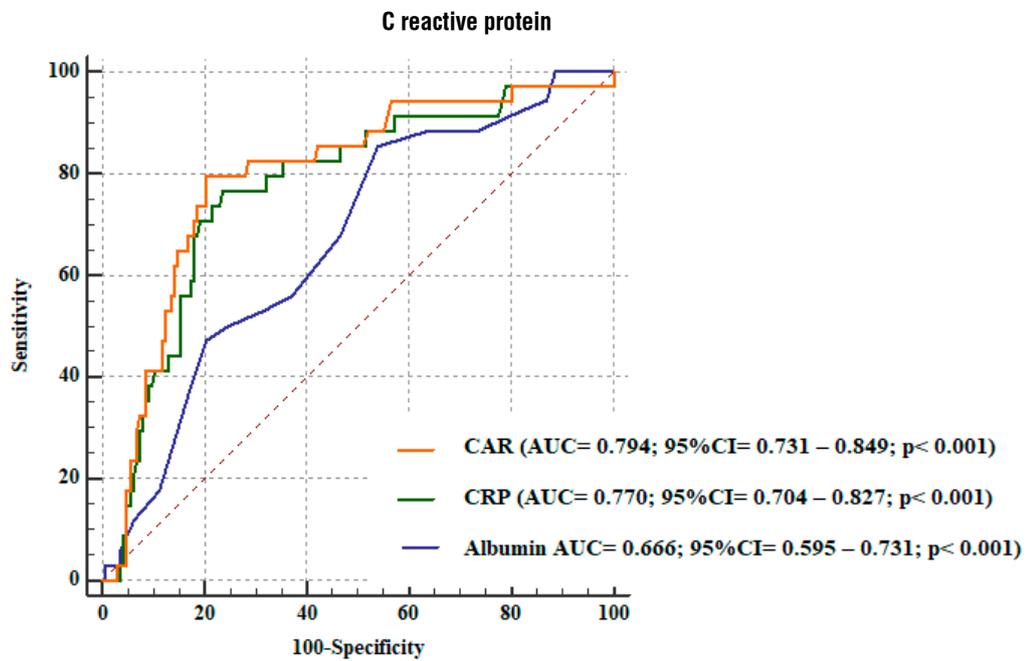
Table 1. Demographic, admission clinical and laboratory parameters of the study cohort†

Variables	All population (n= 195)	Group A (n= 162)	Group B (n= 33)	p
Male gender, n (%)	148 (75.9)	126 (77.8)	22 (66.7)	0.174
Age, year	65.1±10.3	64.2±10.2	69.3±9.4	0.009
BMI	28.7±4.9	28.5±4.6	29.8±6.3	0.143
Hypertension, n (%)	160 (82.1)	132 (81.5)	28 (84.8)	0.646
Diabetes Mellitus, n (%)	75 (38.5)	55 (34.2)	20 (58.8)	0.007
HL, n (%)	96 (49.2)	82 (50.6)	14 (42.4)	0.391
Current Smoking, n (%)	119 (61)	103 (63.6)	16 (46.5)	0.105
LVEF, (%)	48.7±10.4	48.7±10.2	44.2±12.4	0.029
Amount of contrast medium , mL, median, [IQR]	150 [120-250]	140 [100-185]	130 [120-250]	0.349
FBG, mg/dL, median, [IQR]	106 [93-131]	105.5 [92.8-130.2]	109 [92.5-136]	0.892
eGFR, mL/min/1.73m ² , median, [IQR]	65.2±20	66.0±20	66.3±18	0.740
eGFR, mL/min/1.73m ² , median, [IQR]	63.4±24	66.9±24	46.5±15	< 0.001
TC, mg/dL,	180±49	178±50	188±45	0.292
HDL-C, mg/dL	40.7±11.4	40.6±11.1	41.1±12.8	0.794

Variables	All population (n= 195)	Group A (n= 162)	Group B (n= 33)	p
LDL-C, mg/dL	107±38	106±39	113±34	0.349
Triglyceride, mg/dL, median, [IQR]	135 [99-196]	139 [96-199]	129 [112-166]	0.460
Htc, (%)	39.2±6.1	39.4±6.1	38.1±5.8	0.237
CRP, median, [IQR]	7.6 [4.9-11.3]	6.8 [4.5-9.7]	12.4 [9.7-16.9]	<0.001
Albumin	4.1±0.5	4.2±0.5	3.9±0.4	0.003
CAR, median, [IQR]	1.79 [1.22-2.85]	1.62 [1.10-2.25]	3.23 [2.48-4.30]	<0.001

†Continuous variables were presented as means ± standard deviations if normally distributed and medians [interquartile ranges (IQRs)] if not normally distributed, while categorical variables were given as percentages

Abbreviations: BMI, Body mass index; HL, Hyperlipidemia; LVEF, Left ventricular ejection fraction; FBG, Fasting blood glucose; eGFR, Glomerular filtration rate; TC, Total cholesterol; LDL, Low-density lipoprotein; HDL, High-density lipoprotein; Htc, hematocrit; CRP,



Variables	Difference between areas	95%CI	z statistic	p
CAR vs CRP	0.024	0.006 ?0.043	2.551	0.011
CAR vs Albumin	0.129	0.008 ?0.249	2.093	0.036
CRP vs Albumin	0.104	-0,027 ?0.236	1.555	0.120

Figure 1. Discriminatory performance of CAR is statistically superior to that of both CRP and albumin for CIN development.

Cut off value > 2.46 ; % 79.4 Sensitivity; % 79.5 specificity

OP-042 EVALUATION OF THE RELATIONSHIP BETWEEN CORONARY ARTERY ECTASIA AND MPV TO PLATELET COUNT RATIO AND SYSTEMIC IMMUNE-INFLAMMATION INDEX

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Objective: to evaluate the systemic immune-inflammation index (SII) and MPV to platelet count (MPV/platelet) ratio in patients with coronary artery ectasia (CAE).

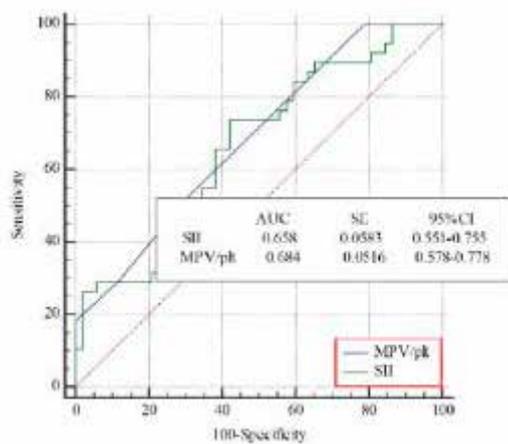
Method: 76 patients with CAE among patients with suspected coronary artery disease on non-invasive imaging tests and coronary angiography performed between June 1, 2020 and January 1, 2021 were included in the study as the study group, and 104 healthy individuals with normal coronary arteries were included as control group. Demographic characteristics of the patients included in the study were recorded, lipid parameters, routine biochemistry tests and complete blood counts were studied from fasting venous blood samples. The systemic immune-inflammation index was calculated by the platelet X neutrophil/lymphocyte formula and the MPV/platelet count ratio by dividing MPV to the platelet counts. CAE was classified according to the classification method determined by Markis et al. $p < 0.05$ was considered as statistically significant.

Results: The mean age of the patients was 60.6 ± 8.6 (48 female, 26.7%). The groups were similar in terms of gender, DM, HT, hyperlipidemia, smoking and family history of atherosclerosis ($p > 0.05$, all). Among the patients in CAE group, 54 had ectasia in LAD, 42 had in CX and 36 had in RCA. There were not any difference in laboratory parameters including complete blood counts between groups but SII and MPV/platelet ratio were both higher in CAE group and the difference was statistically significant ($p = 0.011$ and $p = 0.002$, respectively). Demographic properties and laboratory results of groups are summarized in Table 1. There was a moderate correlation between MARKIS classification and SII and MPV/platelet ratio ($r_s: 0.280$, $p = 0.008$ and $r_s: 0.320$, $p = 0.002$). The multivariate logistic regression analysis revealed that SII and MPV/platelet ratio are associated with CAE (OR: 1.004 [1.001-1.008], $p = 0.012$ and OR: 1.459 [1.208-1.762], $p = 0.007$). Pairwise comparison of Receiver operating characteristics (ROC) curve analysis revealed that MPV/platelet ratio was noninferior to SII for predicting CAE (AUC: 0.684 and 0.658, z statistics 0.314 and $p = 0.753$) (Figure 1).

Conclusion: SII and MPV/platelet ratio, which give information about inflammation and platelet activation, are associated with CAE. Our results need to be supported with prospective studies with larger populations so that these biomarkers can be used in diagnosis of CAE.

Keywords: coronary artery ectasia, immune-inflammation index, mean platelet volume

Figure 1- ROC analysis of MPV/platelet ratio and SII



ROC: Receiver Operating Characteristics; MPV/platelet: MPV to platelet count; SII: systemic immune-inflammation index

Table 1- Demographic properties and laboratory results of groups

	CAE group (n=76)	Control group (n=104)	p
Age, years	60.3±10.4	60.7±7.2	0.401
Female, n(%)	26(34)	22(21)	0.167
HT, n(%)	54(71)	56(53)	0.098
DM, n(%)	24(31)	26(25)	0.491
Smoking, n(%)	34(44)	28(27)	0.079
Family history, n(%)	8(10)	12(11)	0.880
Hyperlipidemia, n(%)	46(60)	78(75)	0.143
LVEF, %	59.6±4.3	60.4±5.7	0.494
WBC (103/mL)	8.3±2.1	8.7±2.3	0.337
HGB (g/dL)	13.6±1.8	13.6±1.6	0.999
PLT (103/mL)	256.7±63.2	273.2±53.1	0.183
Lymphocyte (103/L)	2.5±0.6	2.7±0.5	0.509
Neutrophil (103/L)	4.7±1.5	5.0±1.4	0.358
CRP (mg/dL)	2.9±3.1	2.1±1.5	0.120
Glucose (mg/dL)	135.7±39.1	138.7±49.3	0.758
Creatinine (mg/dL)	0.8±0.1	0.7±0.1	0.198
Urea (mg/dL)	5.54±2.81	4.15±1.27	0.539
MPV (fL)	8.6±0.9	8.5±0.9	0.702
HDL (mg/dL)	43.9±10.3	44.0±9.7	0.976
SII	554.2±281.7	415.1±127.3	0.011*
MPV/platelet ratio	0.04±0.02	0.03±0.01	0.002*

CRP: C-reactive protein, DM: diabetes mellitus, HGB: hemoglobin, HDL: high density lipoprotein cholesterol, HT: hypertension, LVEF: left ventricle ejection fraction, MPV: mean platelet volume, SII: systemic immune-inflammation index, WBC: White blood cell count

OP-043 C REACTIVE PROTEIN TO ALBUMIN RATIO IS PREDICTIVE OF ACUTE STENT THROMBOSIS AFTER PRIMARY PERCUTANEOUS PRIMARY INTERVENTION

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Introduction: C-reactive protein to albumin ratio (CAR) is a novel marker of inflammation. Previous studies showed the association of CAR and coronary artery disease severity, prognosis after acute coronary syndrome and saphenous vein graft disease. In the present study, value of CAR in prediction of acute stent thrombosis after primary angioplasty for ST-segment elevation myocardial infarction (STEMI) is investigated.

Material and methods: The study protocol was approved by the Institutional Ethics Committee. A total of 2403 consecutive patients who underwent primary PCI for STEMI were retrospectively analyzed. Stent thrombosis which occurred during first 24 hours after stent implantation was defined as acute stent thrombosis. Patients were divided into two groups as stent thrombosis and no stent thrombosis. Predictors of acute stent thrombosis were investigated by multivariate logistic regression analysis.

Results: Acute stent thrombosis was occurred in 58 patients. CAR was significantly higher in stent thrombosis group (0.142 ± 0.066 vs 0.117 ± 0.095 , $p < 0.001$) (Table 1). In multivariate logistic regression analysis, CAR was found as an independent predictor of acute stent thrombosis (OR 1.878, %95 CI 1.658-2.125, $p = 0.001$). Admission Killip Class >1 , stent length and stent diameter were other independent predictor of acute stent thrombosis (Table 2).

Conclusion: C-reactive protein to albumin ratio is independently associated with acute stent thrombosis after primary angioplasty for ST-segment elevation myocardial infarction.

Table 1

	No Stent Thrombosis (n=2345)	Stent Thrombosis (n=58)	p value
Age (mean \pm sd.)	55.92 \pm 10.80	55.71 \pm 11.07	0.522
Male Gender [n(%)]	1932 (82.3)	48 (82.7)	0.433
Hypertension [n(%)]	932 (39.7)	24 (41.3)	0.545
Diabetes Mellitus [n(%)]	1722 (73.4)	41 (70.6)	0.321
Smoking [n(%)]	1355 (57.7)	33 (56.9)	0.685
Hemoglobin	13.93 \pm 1.53	13.26 \pm 1.12	0.370
WBC (x 1,000)	11.87 \pm 3.51	12.04 \pm 3.24	0.511
Platelet (x 1,000)	267.39 \pm 54.11	269.55 \pm 59.97	0.117
Creatinine	1.01 \pm 0.51	1.03 \pm 0.42	0.329
Glucose	147.10 \pm 75.31	146.58 \pm 67.76	0.322
Total Cholesterol	183.10 \pm 36.07	186.40 \pm 53.47	0.221
LDL-C	112.25 \pm 37.50	114.65 \pm 44.37	0.465
HDL-C	40.47 \pm 8.25	41.12 \pm 7.60	0.276
Triglyceride	144.46 \pm 90.55	139.35 \pm 88.08	0.333
CK-MB	231.64 \pm 127.14	246.08 \pm 145.36	0.105
HbA1C	6.65 \pm 1.82	6.30 \pm 1.76	0.588
Killip Class >1	225 (9.6)	8 (13.7)	0.003
hsCRP (mg/L)	5.2 \pm 2.6	5.8 \pm 3.1	0.090
Albumin (g/L)	44.4 \pm 12.6	40.7 \pm 13.5	0.078
CRP/Albumin Ratio	0.117 \pm 0.095	0.142 \pm 0.066	<0.001

Table 2

Variables	Univariable		Multivariable	
	OR (95 % CI)	p value	OR (95 % CI)	p value
Age	0.977 (0.956-1.022)	0.866	-	-
Male Gender	1.225 (0.590-2.876)	0.545	-	-
Hypertension	1.016 (0.633-1.567)	0.890	-	-
Diabetes Mellitus	0.912 (0.345-1.987)	0.690	-	-
Smoking	1.255 (0.567-2.445)	0.455	-	-
CRP/Albumin Ratio	1.990 (1.788-2.243)	<0.001	1.878 (1.658-2.125)	0.001
Killip Class >1	1.845 (1.188-2.776)	0.01	1.655 (1.089-2.211)	0.02
Creatinine	1.188 (0.852-1.726)	0.388	-	-
CK-MB	1.009 (1.001-1.012)	0.088	1.007 (1.000-1.003)	0.065
LDL-C	1.016 (0.995-1.023)	0.212	-	-
HDL-C	1.007 (0.988-1.027)	0.206	-	-
Triglyceride	0.988(0.975-1.012)	0.322	-	-
Glucose	0.999 (0.996-1.004)	0.325	-	-
Stent length	1.244 (1.108-1.371)	0.031	1.134 (1.089-1.481)	0.045
Stent diameter	0.554(0.222-0.888)	0.044	0.633 (0.237-0.875)	0.028
Tirofiban	0.789 (0.512-1.355)	0.677	-	-

OP-044 INCREASED DE RITIS RATIO MAY INDICATE CARDIOVASCULAR RISK IN PCOS PATIENTS

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Objectives: Polycystic ovary syndrome (PCOS) is a disease characterized by chronic anovulation and hyperandrogenism, and is the most common endocrine disorder in women of reproductive age. It causes hirsutism, menstrual irregularities, metabolic disorders as well as an increase in the risk of cardiovascular disease. Previous studies have reported endothelial dysfunction, signs of subclinical atherosclerosis, and increased risk of cerebrovascular disease due to PCOS. It has been shown in previous studies that insulin resistance, dyslipidemia, various biomarkers, and carotid-intima media thickness and vascular calcification, which are indicators of atherosclerosis, show an increase in cardiovascular risk in PCOS patients. In the previous studies, as a routine noninvasive laboratory test, the AST/ALT ratio (De Ritis ratio) has been determined as a useful marker to assess cardiovascular risk. The relationship between De Ritis ratio and PCOS, which is useful in the assessment of cardiovascular mortality and risk, has not been a subject of research in previous studies. In this study, we aimed to investigate the De Ritis ratio between PCOS and the control groups and to reveal the relationship between this parameter and the cardiovascular risk in PCOS patients.

Methods: In this study, we enrolled 39 women with PCOS and 40 age and body mass index-matched healthy women. PCOS was diagnosed using the Rotterdam criteria. Patients with a prior history of any liver damage or diseases were excluded from this study. The QRISK2 risk algorithm was used to assess cardiovascular risk in the study population.

Results: A total of 79 participants (39 patients in the PCOS group and 40 in the control group) were included in the present study. Baseline demographic, clinical, and laboratory characteristics of the study groups are presented in Table 1. There were no statistically significant differences between the two groups with respect to the clinical, demographic, and laboratory findings except for follicle-stimulating hormone (FSH), luteinizing hormone (LH), testosterone levels, De Ritis ratio, and QRISK2 score. Patients with PCOS had significantly higher serum testosterone, LH levels, De Ritis ratio value, and QRISK2 score than the control subjects. However, serum FSH levels were significantly lower in PCOS patients compared to the control group. In the univariate correlation analysis, a positive correlation was found between De Ritis ratio and QRISK2 score ($r=0.344$, $p=0.002$) (Figure 1).

Conclusion: PCOS has been associated with possible cardiovascular risk. De Ritis ratio obtained from transaminases, which is a simple blood test, can be used as a useful marker during the assessment of cardiovascular risk in PCOS patients.

Keywords: cardiovascular risk, De Ritis ratio, PCOS

Figure 1

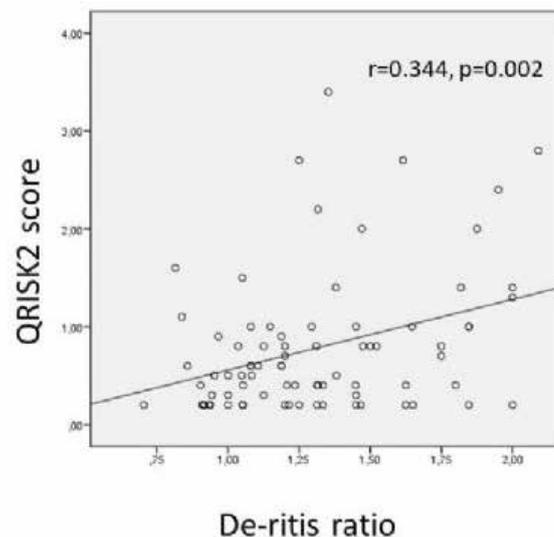


Table 1

	Control (n:40)	PCOS (n:39)	p value
Age (y)	28.5±4.3	27.7±4.6	0.453
Smoking history (n)	8 (20%)	12 (31%)	0.200
Family history (n)	5 (13%)	10 (26%)	0.114
Body mass index (kg/m ²)	28.2±0.5	29.2±0.7	0.525
Systolic blood pressure (mmHg)	105.1±13.6	105.4±11.9	0.893
Diastolic blood pressure (mmHg)	67.6±8.5	67.3±9.6	0.873
Follicle stimulating hormone (IU/mL)	7.4 [5.9-9.4]	6.1 [4.9-6.7]	0.001
Luteinizing hormone (IU/mL)	4.9 [3.7-6.2]	7.5 [5.2-10.3]	<0.001

	Control (n:40)	PCOS (n:39)	p value
Age (y)	28.5±4.3	27.7±4.6	0.453
Estradiol (pg/ml)	40.2 [30.1-56.6]	54.9 [35.2-71.1]	0.095
DHEA-S (mg/dl)	148.3 [103.1-300.1]	184.3 [11.6-236.8]	0.661
Prolactin (ng/ml)	9.8 [7.1-14.7]	8.3 [5.2-12.8]	0.149
Testosterone	30.3 [23.3-36.7]	42.1 [31.7-72.6]	<0.001
LDL (mg/dl)	85.6±29.5	91.7±26.2	0.344
HDL (mg/dl)	51.1±8.3	46.7±13	0.079
Triglyceride (mg/dl)	141.7±80.1	151±78.8	0.574
Hemoglobin (g/dl)	12.5±1.5	13.1±1.9	0.078
Glucose (mg/dl)	92.1±11.5	99.7±11.4	0.348
AST (IU/L)	22.5±6.6	34.3±16.2	<0.001
ALT (IU/L)	20.2±7.6	25.3±10.8	0.074
De Ritis ratio	1.16±0.26	1.46±0.33	<0.001
QRISK2 score	0.3 [0.2-0.5]	1 [0.6-1.4]	<0.001

OP-045 SINGLE STENTING VERSUS DOUBLE STENTING TECHNIQUE IN TRUE BIFURCATION CORONARY LESIONS

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Objective: The ideal treatment technique for coronary bifurcation lesions remains unknown. Although single-stenting strategy has been recommended by default, little evidence exists regarding clinical outcomes between single vs double-stenting in current practice. The purpose of this study was to compare procedural details and clinical outcomes between single vs double-stenting techniques in true bifurcation coronary lesions.

Method: Retrospective study of all patients (pts) referred for percutaneous coronary intervention (PCI) of true bifurcation lesions between June 2018 and June 2020. Only Medina X,X,1 lesions were included. Pts were split in 2 groups: group 1 (single-stenting) and group 2 (double-stenting). Procedural details and clinical outcomes were assessed. Acute and long-term adverse events included procedural complications (a composite outcome of side branch occlusion, coronary iatrogenic dissection and type 4 acute myocardial infarction (AMI)) and a composite of cardiovascular death, AMI, stroke, re-restenosis and reintervention, respectively.

Results: A total of 118 pts were included, 74,6% male, mean age of 66,4 ±11 years.

Ninety-five pts (80,5%) were treated with single-stenting (G1) and 23 pts (19,5%) with double-stenting technique (G2). Both groups were well matched regarding baseline characteristics and clinical presentation. T and protrusion (TAP) and minicrush were the most frequent double-stenting techniques (43,5% and 21,7%).

G2 lesions mainly involved the left main (LM) and proximal left anterior descendent artery (LAD) (52,2%) and in G1 mid LAD (34,7%). LM lesions were more common in G2 (26,1% vs 8,4%; p=0,030). G1 had more lesions Medina 1,1,1 (75,8% vs 52,2%; p=0,025) and less Medina 0,1,1 (9,5% vs 30,4%; p=0,015). Proximal optimization technique and kissing balloon occurred more in G2 (p<0,05). G2 had more intravascular ultrasound guided PCI (p=0,046). Femoral access, heparin, contrast and radiation dose, and fluoroscopy time were higher in G2 (p<0,05). Acute adverse composite outcome was similar in both groups (G1 13% vs G2 14,3%; p=0,855).

Median follow-up was similar (G1 16,8 ±7,9 and G2 19,7 ±8,8 months; p=0,127). G1 had less occurrence of long-term adverse composite outcome (7,4% vs 26,3%; p=0,032). Excluding interventions in LM, G2 had more significant incidence of acute events (20% vs 2,6%; p=0,028), and higher rate of long-term adverse events (20% vs 4,2%; p=0,056). In interventions of the LM only, no differences were noticed in acute and long-term composite events between groups.

Conclusion: Double-stenting techniques in true coronary bifurcation lesions included more often LM lesions and were complex procedures requiring frequently intracoronary imaging. Although acute adverse events were similar to those of single-stenting, long-term adverse outcomes were more frequent in double-stent group, except for LM lesions.

Keywords: coronary bifurcation lesions, percutaneous coronary intervention, double-stenting, single-stenting

OP-046 IMPELLA FOR CARIOGENIC SHOCK AND HIGH-RISK PERCUTANEOUS CORONARY INTERVENTION: A SINGLE-CENTER EXPERIENCE

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Background: The Impella is a percutaneous ventricular assist device that unloads the left ventricle by ejecting blood to the aorta. Its use in cases of cardiogenic shock (CS) and high-risk percutaneous coronary intervention (HR-PCI) is increasing.

Objective: To report clinical outcomes with the Impella device in the settings of CS and HR-PCI.

Methods: Single-center retrospective study including consecutive patients (2007-2019) implanted with Impella for CS treatment or hemodynamic support of HR-PCI.

Results: 22 patients were included: 12 were treated for CS and 10 underwent Impella-supported PCI. Impella 2.5 (7) and Impella CP (15) were used.

In the CS group (75.9% male, mean age 50.4 ± 18.9 , median duration of support 19 ± 24 hours), CS etiologies were myocardial infarction (41.7%), acute myocarditis (25.0%) and acute decompensated heart failure (33.3%). All patients presented with multiorgan dysfunction and were in stage D or E of the SCAI classification of CS. Most patients (83.3%) had severe left ventricular (LV) dysfunction and half also had right ventricular impairment. In 5 cases, combined support of Impella and venoarterial extracorporeal membrane oxygenation (ECMO) was used: in 2 patients, Impella was implanted for LV venting; 3 patients needed escalation to ECMO due to refractory CS. Hemolysis was the most frequent device-related complication (63.7%). Three patients had BARC type 3 vascular complications. Three patients were transferred to a transplantation center, but none survived to transplant. In-hospital, cumulative 30-day and 1-year mortality were 58.3%, 66.6% and 83.3%, respectively.

In the HR-PCI group (all male, mean age 73.7 ± 9.1 years, 50% diabetic, mean left ventricular ejection fraction 39.4 ± 13.6) all patients had multives- sel, highly complex, disease (mean baseline SYNTAX I score 44.1 ± 13.7); six had a last remaining conduit. All patients were considered ineligible for surgery by the Heart Team. Half of the patients underwent PCI in the setting of an acute coronary syndrome. Median number of vessels treated was 2 ± 1 . Seven patients underwent unprotected left main PCI. Impella was immediately explanted after PCI in all cases. There were no intraprocedural or device-related deaths. In-hospital and 30-day mortality were 10%; 1-year cumulative mortality was 30% (all deaths were of cardiovascular causes).

Conclusions: In the CS group, in-hospital and 30-day outcomes were poor, in line with the existing evidence, illustrating the severity, complexity and heterogeneity of this clinical scenario. Acceptable rates of major device-related complications were observed. In the HR-PCI cohort, the use of Impella to provide hemodynamic support was feasible and safe. Long-term results express the severity of the underlying disease and the patients' complexity. With the expanding use of the device, tools to identify the most suitable candidates for Impella support are warranted.

Keywords: Cardiogenic shock, Coronary disease, Impella, Mechanical circulatory support

OP-047 PERCUTANEOUS CORONARY INTERVENTIONS TO NATIVE VS. GRAFT VESSELS IN ACUTE MYOCARDIAL INFARCTION PATIENTS WITH HISTORY OF CORONARY ARTERY BYPASS GRAFTING

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Introduction: Coronary artery disease (CAD) is a common cause of death. Medical treatments, percutaneous coronary interventions (PCI) and coronary artery bypass grafting (CABG) are used in the treatment of CAD. After these treatments, thrombosis or restenosis can be seen in both stents and grafts. Accordingly, patients need repetitive interventional procedures. In patients with a history of CABG, responsible lesions may be grafts, as well as native vessels may be responsible for the clinical situation. In terms of the choice of the procedure, the suitability of the vessels for percutaneous intervention is decisive. Interventions in the graft vessels are often worse in terms of the success of the procedure. For this reason, we observed the percutaneous interventions performed in patients presenting with acute myocardial infarction (AMI) with a history of CABG in terms of both short-term safety and long-term efficacy.

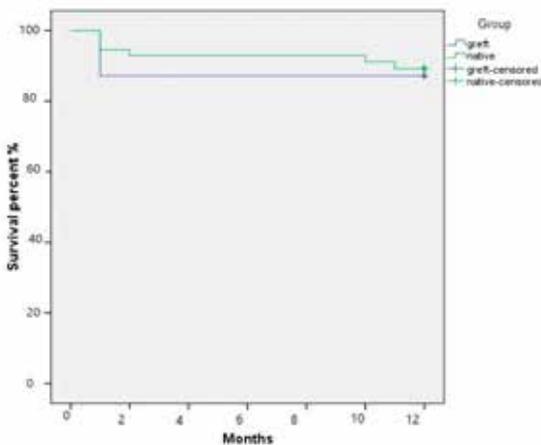
Methods: The study was conducted retrospectively and in a single center. Patients with a history of CABG, who were admitted to our hospital with AMI and treated with PCI were included in the study. In-hospital death and death, stroke, acute stent thrombosis events of the patients within the first 30 days were recorded as major adverse cardiac events (MACE) for 30 days. Recurrent need for vascular intervention to the same vessel within first year; the target vessel revascularization (TVR) rate was recorded and evaluated as the effectiveness point.

Results: A total of 95 patients meeting the criteria were included in the study. While PCI was performed in the graft vessels in 39 (41.1%) patients and in the native vessels in 56 (58.9%) patients. In-hospital deaths has occurred 4 (4.2%) patients, 30 days MACE has occurred in 8 (8.4%) patients and 6 (6.3%) patients has first year TVR. There was no significant difference in inhospital death, 30-days MACE, and 1-year TVR rates among the patient groups who had PCI in the graft vessel or native vessel. In the regression analysis, diabetes was observed as the predictor of both 30-day MACE and 1-year TVR (respectively; OR: 10.73, 95% CI: 2.00-57.56, p: 0.006; OR: 17.25, 95% CI: 1.90-156.30, p: 0.011). The results are summarized in Table-1. In survival analysis, no significant difference was observed in terms of graft and native vessel intervention (p: 0.677). Survival analysis is shown in Figure-1.

Conclusions: Successful percutaneous interventions to graft vessels give similar results to interventions of native vessels. Diabetes mellitus seems to be a prognostic predictor after recurrent acute myocardial infarction and successful percutaneous coronary intervention in patients with a history of coronary artery bypass grafting.

Keywords: Acute myocardial infarction, Coronary artery bypass grafting, Percutaneous coronary interventions

Figure-1



Survival analysis of groups Graft: OR: 10,590 %95 CI: 9,436-11,744 vs. Native: OR: 11,179 %95 CI: 10,453-11,905 p:0,677

Table-1

Parameters	Graft PCI	Native PCI	P value
Number of patients	39 (41.1%)	56 (58.9%)	
Age	69.33±9.97	66.66±8.30	0.159
Gender (Male %)	31 (79.5%)	42 (75%)	0.400
In-hospital deaths	2 (5.1%)	2 (3.6%)	0.545
30-days MACE	5 (12.8%)	3 (5.4%)	0.180
First year TVR	2 (5.1%)	3 (7.1%)	0.522
Hypertension	38 (97.4%)	54 (96.4%)	0.634
Diabetes Mellitus	13 (33.3%)	12 (21.4%)	0.145
Pulmonary diseases	3 (7.7%)	6 (10.7%)	0.452
Heart Failure	5 (12.8%)	5 (8.9%)	0.389
Atrial Fibrillation	2 (5.1%)	2 (3.6%)	0.545
Renal Failure	1 (2.6%)	3 (5.4%)	0.455
DES stent rate	33 (84.6%)	52 (92.9%)	0.171
Klopidogrel rate	30 (76.9%)	46 (82.1%)	0.355

Comparisons of Groups

OP-048 EVALUATION OF INFLAMMATION AND ATHEROGENIC INDICES IN CORONARY ARTERY ECTASIA

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Objective: Coronary artery ectasia (CAE) is characterized by the enlargement of a coronary artery 1.5-fold or greater than the normal coronary artery segment. The pathophysiology of CAE has not been fully elucidated. But, emerging data increasingly support that ectasia formation are considered as the result of atherosclerosis and inflammatory processes. Therefore, we aimed to investigate systemic immune-inflammation index (SII) and atherogenic indices in patients with CAE.

Methods: 95 patients with CAE who had undergone diagnostic coronary angiography between January 2019 and February 2020 at affiliated university hospital were included in this retrospective study. 68 age-and- gender matched subject having normal coronary flow were selected as controls. SII is calculated by $(N \times P) / L$ (N, P and L represent neutrophil counts, platelet counts and lymphocyte counts, respectively). Neutrophil to lymphocyte ratio (NLR) was calculated by dividing the N by L. Lipid profiles (levels of total cholesterol [TC], triglycerides [TG], high-density lipoprotein cholesterol [HDL-C], and low-density lipoprotein cholesterol [LDL-C]) were recorded. Then, atherogenic index of plasma (AIP; TG/HDL-C), and atherogenic coefficient (AC; Non-HDL-C/HDL-C) were analyzed.

Results: AIP, NLR and SII levels were similarly between groups ($p > 0.05$). Compared to the control group, HDL-C (42.3 ± 11.9 vs. 46.2 ± 8.8 , $p = 0.024$) was lower while AC (4.0 ± 1.5 vs. 3.4 ± 1.0 , $p = 0.004$) was significantly higher in CAE patients. Receiver operating characteristic analysis showed that the optimal cutoff value to predict the occurrence of CAE was 3.4 for AC (sensitivity, 58%; specificity, 58%; the area under curve, 0.610; 95% confidence interval [CI], 0.524–0.696; $p < 0.017$). Binary logistic regression analysis showed that AC ($\beta = 2.077$; 95% CI, 1.230–3.506; $p = 0.006$) was an independent predictor of CAE.

Conclusion: Inflammation indices such as SSI and NLR were similar in patients with CAE and healthy subjects. Compared to the control group, HDL-C was lower while AC was significantly higher in CAE patients. AC was independent predictor of CAE.

Keywords: Coronary artery ectasia, inflammation, systemic immune-inflammation index, atherogenic index of plasma, atherogenic coefficient

Demographic and laboratory findings of the study population

Variables	CAE group (n=95)	Control group (n=68)	p
Gender (female), n (%)	51 (53)	29 (43)	0.150
Age, years	48.9±10.7	51.7±6.9	0.063
Smoking, n (%)	25 (26)	19 (28)	0.478
Hypertension, n (%)	29 (31)	29 (43)	0.077
Diabetes mellitus, n (%)	20 (21)	19 (28)	0.203
Dyslipidemia, n (%)	38 (40)	32 (47)	0.230
White blood cell count, ($\times 10^3/\mu\text{L}$)	7.8±2.0	8.3±2.3	0.166
Neutrophil cell count, ($\times 10^3/\mu\text{L}$)	4.5 [6.0-3.4]	4.6 [5.7-3.7]	0.801
Lymphocyte cell count, ($\times 10^3/\mu\text{L}$)	2.4±0.9	2.5±0.9	0.501
NLR	2.1 [2.9-1.5]	1.9 [2.6-1.5]	0.537
Platelet ($10^3/\mu\text{L}$)	245 [297-192]	257 [296-215]	0.397
SII	480 [738-350]	495 [611-380]	0.901
Fasting glucose, mg/dl	100 [118-90]	109 [125-90]	0.217
Creatinine, mg/dl	0.74±0.15	0.79±0.16	0.064
LV ejection fraction (%)	57.1±3.6	57.1±2.5	0.728
Total cholesterol, mg/dl	198.4±36.8	198.3±39.2	0.978
HDL cholesterol, mg/dl	42.3±11.9	46.2±8.8	0.024
LDL cholesterol, mg/dl	118.7±27.8	127.5±34.8	0.064
Triglyceride, mg/dl	154 [234-112]	150 [182-113]	0.615
Non-HDL cholesterol, mg/dl	156.2±35.9	152.1±36.7	0.482
Atherogenic index of plasma	0.56 [0.81-0.34]	0.51 [0.67-0.36]	0.174
Atherogenic coefficient	4.0 ±1.5	3.4±1.0	0.004
1. Atherogenic coefficient: non-HDL-C /HDL-C; Atherogenic index of plasma: log TG/HDL-C; CAE; Coronary artery ectasia; HDL; High-density lipoprotein; LDL: Low-density lipoprotein; LV: left ventricular; NLR: neutrophil lymphocyte ratio; Non-HDLc: TC-HDL-C; SII: systemic immune-inflammation index.			

OP-049 ASSOCIATION BETWEEN NONTRADITIONAL LIPID PROFILES AND ISOLATED CORONARY ARTERY ECTASIA

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Background: Coronary artery ectasia (CAE) is the aneurysmal dilatation of the coronary artery, recognized as a special clinical form of coronary stenosis besides atherosclerosis. The mechanisms, however, responsible for CAE formation during the atherosclerotic process and the exact clinical significance are not well known. Dyslipidemia is considered an independent risk factor for cardiovascular disease (CVD). Moreover, few studies have focused on the relationship between dyslipidemia and CAE. We aimed analyze the relationship between serum traditional lipid profiles and nontraditional lipid profiles to find the risk factors for CAE.

Patients and methods: We conducted a prospective cohort study on patients admitted because of typical or atypical chest discomfort suggestive of angina in Istanbul Medeniyet University and Goztepe Prof.Dr Suleyman Yalcin City hospital from January 2017 to June 2020. We included 75 consecutive patients with CAE; and 60 consecutive patient with Coronary artery disease and 100 consecutive patients with no coronary atherosclerosis and no ectasia/ proved to have normal coronary angiograms. We recorded and compared the general data, cardiovascular risk factors, blood examination index, and coronary angiography data between the all study groups. Serum levels of total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) were performed using standard techniques, and we computed four nontraditional lipid variables (atherogenic dyslipidemia index, non HDL-C, atherogenic coefficient, cardiac risk ratios 1 and 2).

Results: Serum Creatinin, Fasting Glucose, triglyceride, atherogenic dyslipidemia index, non HDL-C, atherogenic coefficient, and cardiac risk ratios 1 and 2 were significantly greater in the patients isolated CAE and patients with than in the patients with normal coronary artery (all $p < 0.001$, respectively). HDL-C levels. High-density lipoprotein cholesterol levels were significantly lower in CAD group than in the normal coronary artery group ($P < 0.001$). Total cholesterol and LDL cholesterol levels were similiary between all groups ($p > 0.05$) The multivariate logistic regression models revealed that atherogenic dyslipidemia index (TG/HDL-C) was found to be independent factor predicting isolated CAE ($p < 0.001$, Odds ratio (OR) = 1.27, 95% Confidence interval (C.I.) = 1.11– 1.45). In ROC curve analyses, an TG/HDL-C of 2.85 was determined as an effective cut-off point in CAE with a sensitivity of 80% a specificity of 78%, respectively (AUC=0.91 $p=0.001$; 95% CI (0.867-0.955)). In receiver operating characteristic curve analyses, an TG/HDL-C of 2.75 was determined as an effective cut-off point in CAD with a sensitivity of 76% a specificity of 76%, respectively (AUC=0.83 $p=0.03$; 95% CI (0.768-0.910)).

Key words: Nontraditional lipid profiles, lipid ratios, atherogenic dyslipidemia index, Coronary artery ectasia; coronary artery disease

Introduction: Isolated coronary artery ectasia (CAE) is characterized as a localized or diffuse, non-occlusive dilatation of epicardial coronary arteries in the diameter of a coronary artery segment of 1,5-fold normal size, considering as such the adjacent non-dilated segments [1,2]. Coronary artery ectasia may be associated with atherosclerosis and other diseases such as kawasaki disease, scleroderma, polyarteritis nodosa, syphilitic aortitis, ehler-Danlos disease, bacterial infections. [3-5]. CAE incidence is reported between 0.3% and 4.9% in patients undergoing coronary angiography [6,7]. Coronary artery ectasia may be asymptomatic but it can cause serious cardiac events, such as angina pectoris, myocardial infarction, malignant arrhythmia and even sudden death [4, 8]. Autopsy cases have revealed the histological alterations of ICAE were equivalent with observed findings in atherosclerotic plaques, such as diffuse intimal and medial degeneration and hyalinization of the media of the vessel wall [2]. Unfortunately, there is still limited information on the underlying biological process [9]. Risk factors for CAE are not well defined as those for stenotic atherosclerosis. Correlation of CAE with hypertension [5], diabetes mellitus (DM), dyslipidemia, smoking and family history of coronary artery disease is still controversial in literature [7, 10].

Dyslipidemia is among the best-understood CAD risk factors [11]. Traditional lipid measurement indices (including Total Cholesterol (TC), Low-Density Lipoprotein Cholesterol (LDL-C), and Triglyceride (TG)) have been proven to be related to CAD onset. Many studies have demonstrated that the reduction in LDL-C significantly reduces cardiovascular event incidence rates. However, even after the recommended level of LDL-C is achieved, the residual cardiovascular risk remains at approximately 50%, highlighting the need to identify new predictors of CAD [12]. Nontraditional lipid profiles including non-HDL-C (TC minus HDL-C), atherogenic dyslipidemia index (TG/HDL-C), atherogenic coefficient (non HDLC/HDL-C), cardiac risk ratio 1 (TC/HDL-C) and cardiac risk ratio 2 (LDL-C/HDL-C) [13]. Recent studies have been placed in the clinical implications of nontraditional lipid profiles as powerful and independent predictors of cardiovascular disease than single lipid parameters. [14-16].

We hypothesized that evaluating the influence of nontraditional lipid profiles on the probability of subclinical atherosclerosis is helpful to understand the pathogenesis of CAE related to dyslipidemia. In order to analyze the above issues, our study investigated the association between CAE and non-traditional lipids profiles and compare CAD and NCA patients.

Materials and Methods

Study design and population

Study population was collected prospectively from pool of patients referred for elective cardiac catheterization at Istanbul Medeniyet University and Goztepe Prof.Dr Suleyman Yalcin City hospital in the period extending from from January 2017 to June 2020. Patients were referred for elective coronary angiography after being interviewed and physically examined by a cardiologist (HA) due to a variety of reasons: stable angina and positive non-invasive evaluation by treadmill stress test or myocardial perfusion scintigraphy. The Study included 75 consecutive patients with CAE; and 60 consecutive patient with Coronary artery disease and 100 consecutive patients with no coronary atherosclerosis and no ectasia/ proved to have normal coronary angiograms. Te exclusion criteria were as follows: acute coronary syndrome, coronary spasm, History of myocardial infarction, percutaneous coronary intervention, and coronary bypass grafting, congenital heart disease, valvular heart diseases, arrhythmia, left ventricular ejection fraction < 55%, chronic renal failure, malignancy and acute/chronic infection, immune disease, connective tissue disorders, and receiving lipidlowering therapy, hepatic or renal insuficiency acute/chronic infection, immune disease, rheumatic disease, chronic systemic illness, alcohol abuse were the exclusion criteria. Demographic features such as sex, age, family history, smoking were recorded. Body mass index (BMI) was calculated by the World Health Organization criteria [17]. Patients who were under antihypertensive treatment or had blood pressure higher than 140 mm Hg systolic or 90 mm Hg diastolic measured from any arms were considered as hypertensive. Patients with known diabetes, taking antidiabetic medication, or

who had fasting plasma glucose levels >126 mg/dL were considered as diabetic. The local ethics committee approved the study protocol. This study was accord with the declaration of Helsinki. Written informed consent was obtained prior to enrolment.

Laboratory tests: Venous blood samples were drawn from each participant after an overnight (12-h) fasting. All biochemical and hematological parameters including fasting blood glucose, creatinine, urea, uric acid, aspartate aminotransferase (AST), alanine aminotransferase (ALT), TC, TG, HDL-C, and LDL-C were analyzed on the day of sample collection using a commercial autoanalyzer (c8000i; Abbott Diagnostics GmbH, Germany). For samples with a TG level of <400 mg/dL, the LDL-C was calculated using the Friedewald formula [(LDL-C = TC – HDL-C – (TG/5)] (23). Hematological analyses were carried out using the BC-6800 Hematology Analyzer (Shenzhen Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China). Non-HDL-C was calculated as HDL-C subtracted from TC. The TC/HDL-C, TG/HDL-C, and LDL-C/HDL-C ratios were calculated as TC, TG, and LDL-C divided by HDL-C, respectively.

Coronary Angiography: Coronary angiography was performed using femoral approach by the standard Judkins method. Angiographic images were evaluated by two independent researchers. Patients were allocated into three groups: patients with CAD, those with isolated CAE and subjects with normal coronary angiograms. Isolated CAE was defined as localized or diffuse dilatation of the coronary artery by 1.5 times or more compared to the adjacent normal segments or the diameter of the patient's largest coronary artery without significant stenotic lesion [3,18]. According to the Markis classification, the severity of isolated CAE was classified as: type I, diffuse ectasia at least 2 vessels; type II, diffuse ectasia in one vessel and localized ectasia in another vessel; type III, diffuse ectasia of only 1 vessel; and type IV, segmental localized ectasia [2]. NCA were defined as the absence of angiographic atherosclerosis during routine coronary angiography; >50% stenosis in at least one epicardial coronary artery was defined as CAD. / Significant CAD was defined as >50% stenosis in at least 1 coronary arte

Statistical analysis: Statistical analysis was performed using the SPSS version 26.0 software (IBM Corp., Armonk, NY, USA). Continuous data were expressed in mean \pm SD or median (interquartile range [IQR]), while categorical data were expressed in number and percentage. The normality assumption was checked using the Kolmogorov-Smirnov test. One-way analysis of variance (ANOVA) with post-hoc Tukey test, independent samples t-test, Kruskal-Wallis and Mann-Whitney U tests were used to examine independent quantitative variables. The paired samples t-test and Wilcoxon test were performed to analyze dependent quantitative variables. Independent qualitative variables were examined using the chi-square test or Fisher's exact test, when the chi-square assumptions were not met. Multivariate logistic regression analysis and receiver operating characteristic (ROC) curve were to assess the independent risk factors for CAE and the predictive value. P value less than 0.05 was considered statistically significant.

Results: Seventy five consecutive CAE patients (54(%72) men, mean age: 56. \pm 8 years), 60 consecutive CAD patients (43 (%71) men, mean age: 57.8 \pm 7 years), and 75 patients with normal coronary arteries (NCA; 45(%60) men, mean age: 54.7 \pm 8.4 years) were included. The demographic, clinical, and angiographic data of the study population are summarized in Tables-1. No differences in age, gender, body mass index were observed among the 3 groups (all P >0.05). The 3 groups had similar traditional cardiovascular risk factors with regards to presence of hypertension, diabetes and smoking with the exception of having a family history of CAD. The family history of CAD in the CAD group were significantly higher than those of the CAE group and the NCA group (p<0.007). The Markis classification and distribution and frequency of the ectatic coronary arteries are shown in Table-1. Accordingly, ectasia frequency was highest in the left anterior descending (LAD) artery (76%), followed by the right coronary artery (RCA) (64%), circumflex artery (44%). In the CAD group, the average SYNTAX score was 8.9+ 6.1. Laboratory measurements from individuals with isolated CAE and those with CAD and normal controls were presented in Table-2. Hemoglobin ,white blood cel, Alanine transaminase (ALT) and aspartate transaminase (AST) levels were similar in all groups. Serum creatinine level was within normal limits in all patients, however creatinine values were statistically lower in the NCA group (p <0.001) compared with patients with obstructive CAD as well as CAE group. The data indicated that patients with CAE and CAD showed higher fasting glucose levels compared with patients with NCA. The data indicated that patients with CAE and CAD showed higher fasting glucose levels compared with patients with NCA. Compared with the participants' traditional lipid profiles; serum TC, LDL-C levels were similar in both groups. HDL-C and TG levels were significantly different among the groups. HDL cholesterol levels were lowest in the CAD patients and highest in the NCA group, and this difference was statistically significant (p <0.001). TG levels were found to be higher in the CAD and CAE groups, compared to the NCA group (p<0.001). There was no significant difference TG the CAD and CAE groups. Compared with the participants' non-traditional lipid profiles ; Participants with CAD and CAE were showed a worse lipid profile (high levels of TC/HDL-C, TG/HDL-C, LDL-C/HDL-C, and non-HDL-C, non-HDL –C /HDL-C). Table-3 shows the Results of receiver-operating curve (ROC) analyses and cutoff points for lipid indexes for the prediction of CAE with their corresponding specificity and sensitivity. Cutoff points for TG/HDL-C, LDL-C/HDL-C, TC/HDL-C and non-HDL –C /HDL-C to predict CAE were 2.85, 2.74, 4.6 and 3.6 respectively. ROC curves, atherogenic dyslipidemia index (TG/HDL-C ratio) had the highest predictivity, with a sensitivity of 80% and a specificity of 78% than other lipid parameters(Table-4). In ROC curve analyses, an TG/HDL-C of 2.85 was determined as an effective cut-off point in CAE with a sensitivity of 80% a specificity of 78%, respectively (AUC=0.91 p=0.001; 95% CI (0.867-0.955), Figure-2 a). In ROC curve analyses, an TG/HDL-C of 2.75 was determined as an effective cut-off point in CAD with a sensitivity of 76% a specificity of 76%, respectively (AUC=0.83 p=0.03; 95% CI (0.768-0.910), Figure-2 b). The multivariate logistic regression models revealed atherogenic dyslipidemia index (TG/HDL-C) was found to be independent factors predicting isolated CAE (Table-5)

Table-1 Demographic, Clinical, and Angiographic Characteristics of the Study Population

	NCA (n = 75)	CAE (n = 75)	CAD (n = 60)	p Value
Age (years)	54.7 ± 8.4	56.7 ± 8.0	57.8 ± 7.0	0.07
BMI (kg/m ²)	28.6 ± 3.6	28.9 ± 3.6	28.7 ± 4.1	0.91
Male (n,%)	45(%60)	54(%72)	43(%71)	0.21
Smoker (n,%)	23(%30)	27(%36)	20(%33)	0.78
Hypertension (n,%)	37(%49)	48(%64)	34(%56)	0.19
Diabetes mellitus(n,%)	16(%21)	19(%25)	21(%35)	0.19
Hyperlipidemia (n,%)	19(%25)	26(%35)	22(%37)	0.30
Family Histof of CAD (n,%)	19(%25)	16(%21)	27(%45)	0,007**
Angiographic Characteristics				
Marcis classification		21 (%28)		
Type I				
Type II		18 (%24)		
Type III		20 (%27)		
Type IV		16 (%21)		
CAE distribution				
LAD		57 (%76)		
LCX		33 (%44)		
RCA		48 (%64)		
SYNTAX score			8.9±6.1	

BMI, Body mass index; CAD, Coronary artery disease; CAE, Coronary artery ectasia; NCA, normal coronary artery, LCx, left circumflex artery; LAD, left anterior descending artery; LMCA, left main coronary artery; RCA, right coronary artery. * Control vs. other groups. ** CAE group vs. CAD group.

Table-2 Laboratory Findings of the Study Population.

	NCA (n=75)	CAE (n = 75)	CAD (n = 60)	P Value
Fasting Glucose (mg/dl)	96.3 ± 11.8	100.1 ± 11.2	108 ± 23.3*	<0.001
Creatinine (mg/dl)	0.7 ± 0.1	0.9 ± 0.2*	0.9 ± 0.2*	<0.001
AST (U/l)	21 ± 8.5	22 ± 5.4	23.1 ± 8.9	0.27
ALT (U/l)	19.9 ± 5.6	20.5 ± 7.7	20.3 ± 5.6	0.06
Hs CRP (mg/L)	1.0 ± 0.8	3.1 ± 2.3*	0.8 ± 0.7*	<0.001
WBC (10 ³ /mm ³)	7.5 ± 1.7	7.1 ± 2.6	8.1 ± 1.9	0.05
Hemoglobin (g/dL)	13.3 ± 1.6	13.8 ± 1.3	13.4 ± 1.5	0.07
Traditional lipid profiles				
TC (mg/dl)	194.7 ± 41.9	205.1 ± 40.3	193.1 ± 38.2	0.15
HDL-C (mg/dl)	49.5 ± 12.1	41.5 ± 10.2*	37.3 ± 7.0**	<0.001
TG (mg/dl)	108.6 ± 26.5	188.1 ± 71.3*	180.6 ± 100.2*	<0.001
Nontraditional lipid profiles				
LDL-C (mg/dl)	118.5 ± 37	125.2 ± 34.1	118.9 ± 39.5	0.47
Atherogenic Dyslipidemia Index (TG/HDL-C)	2.2(1.8-2.7)	4.8 ± 2.3*	5.1 ± 3.1*	<0.001
Cardiac Risk Ratio 2 (LDL-C/HDL-C)	2.4(2.0-3.3)	2.9(2.5-3.6) *	3.3 ± 1.3*	<0.001
Atherogenic Coefficient (non-HDL -C /HDL-C)	2.9(2.4-4.0)	4.0(3.3-4.7) *	4.4 ± 1.5*	<0.001
Cardiac Risk Ratio 1 (TC/HDL-C)	3.98 (3.4-5.0)	5.0 (4.3-5.7)*	5.4 ± 1.5*	<0.001
Non-HDL -C (TC-HDL)	145.2 ± 43.7	163.6 ± 35.8	155.8 ± 39.6	0.02

ALT, alanine transaminase; AST, aspartate transaminase; HDL-C, High density lipoprotein cholesterol; LDL-C, Low density lipoprotein cholesterol; TC, Total cholesterol; TG, Triglyceride, WBC, White blood cell. * Control vs. other groups. ** CAE group vs. CAD group.

Table-3 The comparison of Lipid profiles according to the type of coronary ectasia in CAE group

	Type1	Type 2	Type 3	Type 4	p	1 vessel	2 vessel	3 vessel	4 vessel	p
Traditional lipid profiles										
TC (mg/dl)	212,3±44,5	202,7±35,0	199,0±40,1	207,3±43,1	0,75	212,4±45,1	198,2±34,3	203,8±41,7	207,2±39,0	0,68
HDL-C (mg/dl)	41,1±9,3	42,4±12,5	43,0±10,0	38,9±9,1	0,65	43,5±12,3	40,7±8,3	40,0±9,2	42,2±11,3	0,66
LDL (mg/dl)	130,7±36,0	123,4±25,8	119,0±34,4	129,1±41,0	0,69	127,8±40,9	121,4±26,2	125,7±34,6	129,4±33,4	0,92
TG (mg/dl)	204,0±68,7	182,6±43,5	184,9±86,1	178,1±81,0	0,71	198,1±94,7	175,7±53,6	190,6±61,2	183,2±35,1	0,75
Nontraditional lipid profiles										
Non-HDL-C	165,5±24,7	166,1±44,4	152,8±31,9	164,5±26,5	0,61	155,5±34,5	178,7±43,3	159,3±27,7	161,0±25,9	0,13
TG/HDL-C	5,2±2,1	4,5±1,3	4,6±2,7	5,0±3,0	0,75	5,0±3,1	4,4±1,5	5,0±2,0	4,8±2,2	0,84
LDL-C/HDL-C	3,2±0,6	3,1±1,1	2,8±0,7	3,4±1,4	0,28	3,1±1,2	3,1±0,9	3,2±0,9	3,2±0,8	0,94
Non HDL-C/HDL-C	4,2±0,8	4,0±1,3	3,7±0,9	4,6±1,8	0,21	4,1±1,5	4,0±1,0	4,2±1,1	4,1±1,0	0,94
TC/HDL-C	5,2±0,8	5,0±1,3	4,7±0,9	5,6±1,8	0,21	5,1±1,5	5,0±1,0	5,2±1,1	5,1±1,0	0,94

HDL-C, High density lipoprotein cholesterol; LDL-C, Low density lipoprotein cholesterol; HDL-C, Non-HDL-C ; non- High density lipoprotein cholesterol; TC, Total cholesterol. TG, Triglyceride,

Table -4 ROC curve for predicting coronary artery ectasia and cutoff points for maximum of sensitivity and specificity

	ROC (95 % CI)	Cut point	Sensitivity (%)	Specificity (%)
TG/HDL-C	0.910(0.867-0.955)	2.85	80	78
LDL-C/HDL-C	0.558 (0.462-0.655)	2.74	56	52
TC/HDL	0.596 (0.500-0.691)	4.6	61	55
Non-HDL-C /HDL-C	0.596 (0.500-0.691)	3.6	61	55

AUC, Area under the curve ; CI, confidence interval; HDL-C, High density lipoprotein cholesterol; LDL-C, Low density lipoprotein cholesterol; HDL-C, Non-HDL-C ; non- High density lipoprotein cholesterol; ROC, receiver operating characteristic curve ;TC, Total cholesterol; TG, Triglyceride.

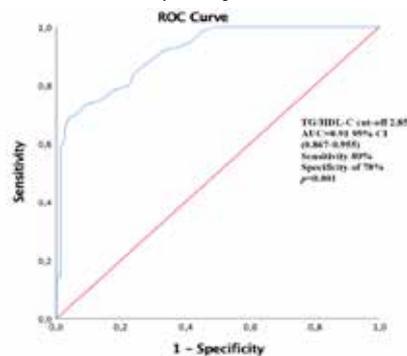


Figure-2a. In ROC curve analyses, an TG/HDL-C of 2.85 was determined as an effective cut-off point in CAE with a sensitivity of 80% a specificity of 78%, respectively (AUC=0.91 p=0.001; 95% CI (0.867-0.955).

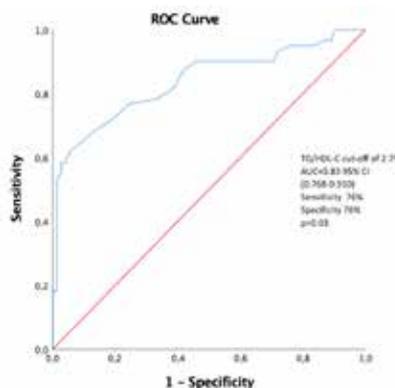


Figure-2 b. In ROC curve analyses, an TG/HDL-C of 2.75 was determined as an effective cut-off point in CAD with a sensitivity of 76% a specificity of 76%, respectively (AUC=0.83 p=0.03; 95% CI (0.768-0.910).

Table 5. Univariate and Multivariate Logistic Regression Analysis Showing Predictors of the CAE

	Univariate Model					Multivariate Model				
	OR	%95 CI			p	OR	%95 CI			p value
Age, years	0.99	0.96	-	1.03	0.823					
Gender (male)	2.15	1.18	-	3.92	0.042					
BMI	1.01	0.94	-	0.1.09	0.814					
Hypertension	1.64	0.93	-	2.90	0.089					
Diabetes	0.64	0.34	-	1.18	0.157					
Family History of CAD	0.46	0.24	-	0.87	0.017					
Smoking	1.2	0.66	-	2.14	0.549					
Kreatinin	1.0	0.96	-	1.05	0.896					
Glucose	1.0	0.99	-	1.01	0.615					
TG/HDL-C	1.28	1.13	-	1.44	<0.001	1.27	1.11	-	1.45	<0.001
WBC	0.91	0.81	-	1.02	0.093					

BMI, Body mass index; CAD, Coronary artery disease; HDL-C, High density lipoprotein cholesterol; TG, Triglyceride; WBC, White blood cell.

Discussion: In the present study, all conventional serum lipid profiles, non-traditional serum lipid ratios were investigated and TG, non-HDL-C TG/HDL-C, TC/HDL-C, LDL-C/ HDL-C and non-HDL-c/HDL-C was significantly higher in both CAE and CAD groups than in the NCA group. TG/HDL-c has a good predictive value for both of CAE and obstructive CAD. The positive relationship between atherogenic dyslipidemia and the presence of CAE may support that atherosclerosis may play a role in the etiology of CAE. The histopathological characteristics of patients with CAE were similar to those with CAD. Some studies claim that CAE is a variant of atherosclerosis [3]. There is still a debate on the risk factors for CAE. Some studies revealed that coronary ectasia was associated with the classic cardiovascular risk factors [19, 20].

Dyslipidemia is a major contributor to atherosclerosis, which is characterized by the deposition of lipids and fibrous elements in the arteries and further formation of atheroma [11]. Our study focused on the relationship between CAE and lipid parameters [19-21]. Previous studies have shown that lipid metabolism is correlated with CAE. It was discovered that patients with familial hypercholesterolemia had significantly higher ectasia prevalence. The same study showed that ectasia was closely associated with higher LDL cholesterol and a lower HDL-C [21]. It was reported that hyperlipidemia is a strong predictor of long-term mortality in patients with CAE [20]. Fang et al. found that dyslipidemia was greater than 2 fold in patients with CAE compared with that in the general population [19].

Nontraditional lipid parameters and atherogenic indices (atherogenic dyslipidemia index, non HDL-C atherogenic coefficient, cardiac risk ratios 1 and 2) relationship between atherogenic dyslipidemia and CVD is well known [14, 15]. Many clinical studies make effort to introduce a better marker of that atherogenic indices can predict the risk of CVD. It has been shown that atherogenic dyslipidemia index (or atherogenic index of plasma) [TG/HDL-C] is a strong marker to predict the risk of atherosclerosis and coronary heart disease (CHD) [22-24]. Qin et al. found that TG levels and Cardiac Risk Ratio-2 ratio were significantly higher in the CAE patients and that they are both independent risk factors and have a fine predictive value for CAE [25]. Our Study. Similarly, in our study, triglyceride and Cardiac Risk Ratio -2 (LDL/HDL-C) were significantly higher in the CAE and CAD groups than in the NCA group. Dogdus et al. evaluated the relationship between the atherogenic dyslipidemia and isolated CAE. Atherogenic indices (atherogenic dyslipidemia index(TG/HDL-C ratio , non HDL-C, cardiac risk ratios 1 and 2, atherogenic coefficient,) were significantly greater in the isolated CAE patients than in the control and multivariate logistic regression models revealed that atherogenic dyslipidemia index was found to be independent factor predicting isolated CAE [26]. Atherogenic dyslipidemia index (TG/HDL-C ratio , non HDL-C, cardiac risk ratios 1 and 2, atherogenic coefficient) were significantly greater in the isolated CAE patients and CAD patients than in patients with NCA. Atherogenic dyslipidemia index (TG/HDL-C ratio) was found to be independent factors predicting isolated CAE. Atherogenic dyslipidemia index was good predictor both of CAD and CAE thus indicating that dyslipidemia plays an important role in CAE dyslipidemia plays an important role in CAE and thus may be to some extent related to the mechanism of CAE. Although the specific pathophysiological mechanism is still unclear, we can take the triglyceride/HDL ratio as a reference to evaluate the possibility of CAE.

There are some limitations to this study. First, it has a single-center design with relatively small sample size. Second, the TG/HDL ratio was not assessed as a parameter of the metabolic syndrome. Although patients using lipid-lowering drugs were excluded from the study, other drug information was not included in our study. The effects of these treatments, diet and exercise on lipid parameters were not evaluated. This study was a cross-sectional study, which limits the ability to conclude a cause-effect association between lipid and lipid ratios.

In conclusion, Increased atherogenic indices (atherogenic dyslipidemia index, non HDLC, atherogenic coefficient, cardiac risk ratios 1 and 2) may be involved in the pathogenesis of the isolated CAE. There may be association between hyperlipidemia and the prognosis of CAE, and lipid-lowering drugs can decrease the prevalence of CAE. Further prospective, randomized-controlled large-scale studies are needed to confirm these findings.

OP-051 THE PROGNOSTIC PERFORMANCE OF PLATELET TO LYMPHOCYTE RATIO AMONG PATIENTS WITH ACUTE DECOMPENSATED HEART FAILURE

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OBJECTIVE: There are various inflammatory mediators in the pro-inflammatory pathway that can cause myocardial apoptosis, hypertrophy and fibrosis, as well as lead to adverse cardiac remodeling. This study aims to evaluate the role of circulating platelet to lymphocyte ratio (PLR) in predicting six-month mortality in patients with heart failure (HF).

METHOD: All patients included in the study had the following inclusion criteria: progressive dyspnea associated with clinical evidence of pulmonary congestion and decreased cardiac output requiring hospitalization. From February 2019 to April 2020, a total of 103 patients met the inclusion criteria. Univariate and multivariate Cox proportional-hazard regressions were performed to determine the association of PLR with survival.

RESULTS: A total of 103 patients with HF were included in the study. BMI (p=0.028), NYHA class (p=0.001), PLR (p<0.001), serum creatinine (p<0.001), and LVEF (P=0.042) showed significant differences between surviving and deceased patients. Univariate analysis identified high serum creatinine, old age, high PLR, low LVEF, smoking habit, and high platelet count as poor prognostic factors for survival.

CONCLUSION: In this study of HF patients, a significant relationship was found between PLR and six-month mortality. A higher PLR in HF patients has been associated with poor clinical outcomes and PLR may be useful in the future as a new marker in the management of HF patients.

Keywords: Heart failure, platelet to lymphocyte ratio, survival

Figure 1

	Survivors (n= 85)	Non-survivors n= (18)	P value
Demographics			
Age (years)	70.5 ± 8.6	71.3 ± 7.4	0.635
Male gender	47 (55.3%)	12 (66.7%)	0.932
Smoking	30 (35.3%)	7 (38.9%)	0.353
BMI (kg/m ²)	24.9 ± 3.9	23.6 ± 2.9	0.028
Vital signs			
Systolic BP (mmHg)	129.2 ± 12.2	133.1 ± 13.1	0.094
Diastolic BP (mmHg)	77.1 ± 7.3	78.5 ± 7.4	0.292
Heart rate (/min)	80.1 ± 11.8	83.0 ± 14.8	0.207
Co-morbidities			
Hypertension	57 (67.1%)	9 (50.0%)	0.554
Diabetes	33 (38.8%)	6 (33.3%)	0.757
NYHA class			
NYHA III	51 (60.0%)	7 (38.9%)	0.001
NYHA IV	34 (40.0%)	11 (61.1%)	
Biochemical parameters			
Hemoglobin (g/dL)	11.1 ± 1.2	11.1 ± 1.4	0.158
PLR	121.04 ± 41.59	162.74 ± 49.37	<0.001
Serum sodium (mEq/L)	136.7 ± 3.3	138.8 ± 3.2	0.930
Potassium (mEq/L)	4.5 ± 0.5	4.4 ± 0.5	0.476
Serum creatinine (mg/dL)	1.1 ± 0.2	1.3 ± 0.1	<0.001
TG (mg/dL)	104.0 (46.2-312.3)	118.0 (48.0-312.0)	0.714
LDL-C (mg/dL)	120.4 ± 42.4	123.6 ± 43.4	0.269
Echocardiography			
LVEF (%)	47.0 ± 7.2	36.4 ± 5.7	0.041
LVDd (mm)	52.5 ± 6.2	53.4 ± 7.5	0.312

Patient characteristics at baseline according to mortality for the entire cohort (n = 103)

Figure 2

Baseline variable	Univariate analysis		Multivariate analysis	
	Risk ratio (95% CI)	p-value	Risk ratio (95% CI)	p-value
Serum creatin	9.91 (3.660-26.838)	<0.001	4.594 (1.589-13.282)	0.005
LVEF	1.150 (1.093-1.211)	<0.001	1.059 (1.003-1.119)	0.037
PLR	1.361 (1.258-1.473)	<0.001	1.276 (1.166-1.396)	<0.001
Smoking	1.083 (1.059-1.107)	<0.001	1.051 (1.020-1.083)	<0.001
Platelet	2.903 (1.529-5.510)	0.001	2.536 (1.087-5.109)	0.030
NYHA class IV	1.426 (0.712-2.855)	0.317		NS
Age>80	0.788 (0.348-1.780)	0.566		NS

Results of univariate Cox proportionalhazards regression analysis regarding survival in patients with acute heart failure.

OP-052 SEX DIFFERENCES IN PATIENTS WITH MID-RANGE HEART FAILURE

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Objective: Mid-range heart failure forms a special group of heart failure. We aimed to investigate gender based differences in patients with mid-range heart failure.

Method: A total of 203 patients with mid-range heart failure were classified into two groups by gender. Clinical, echocardiographic and biochemical parameters were compared between two groups.

Results: Compared with men, women were older (67.5±11.4 vs. 71.5±11.9 years, p=0.019), had higher body mass index [27.8 (36-30) kg/m² vs. 31.2 (26.2-35.4) kg/m², p<0.001]. Women had higher prevalence of atrial fibrillation [34 (%26.4) vs. 33 (%44.6), p=0.006]. Also men have higher prevalence of coronary artery disease [93 (%72.1) vs. 35 (%47.3), p<0.001]. In echocardiographic parameters; E/E' ratio was higher in females [6 (5-7) vs. 7 (5-8), p=0.044], B-blockers [68 (%52.7) vs. 29 (%39.2), p=0.043], acetyl salicylic acid [60 (%46.5) vs. 18 (%24.3), p=0.001] and statins [62 (%48.1) vs. 20 (%20.7), p=0.002] were used more in men. Whereas, oral anticoagulants [30 (%23.3) vs. 32 (%43.2), p=0.003] were used more in women. In biochemical parameters; eGFR (74.2±21.2 vs 62.2±21.3, p=0.001), Hb [13.7 (11.8-14.9) vs. 12 (11.1-13), p>0.001], Htc (40.1±5.6 vs. 36.9±4.1, p<0.001) was lower in women.

Conclusion: Compared with men in patients with mid-range heart failure, women were older, had higher prevalence of atrial fibrillation, body mass index, E/E' ratio and had lower eGFR. Whereas men have higher prevalence of coronary artery disease. Awareness of gender differences in patients with mid-range heart failure is essential for more effective and individualised clinical management.

Keywords: mid range heart failure, sex, differences

Table 1: Baseline characteristics of patients with mid range heart failure

Characteristic	Male (n=129)	Female (n=74)	P-value
Age (years)	67.5±11.4	71.5±11.9	0.019
Body mass index (kg/m ²)	27.8 (36-30)	31.2 (26.2-35.4)	<0.001
Coronary Artery disease	93 (%72.1)	35 (%47.3)	<0.001
Hypertension	87 (%67.4)	49 (%66.2)	0.489
Diabetes mellitus	44 (%34.1)	25 (%33.8)	0.544
Atrial Fibrillation	34 (%26.4)	33 (%44.6)	0.006
Anemia	49 (%38)	33 (%44.6)	0.255
Ejection fraction (%)	49 (45-49)	49 (45-49)	0.445
LVEDD (mm)	50 (48-54)	50 (48-52)	0.306
Left atrium diameter (mm)	38.8±5.5	39.3±6.3	0.570
Right ventricular diameter (mm)	24 (22-25)	23 (21-24)	0.070
sPAP (mmHg)	25 (20-35)	23 (21-24)	0.084
E/E' ratio	6 (5-7)	7 (5-8)	0.044
E/A ratio	0.77 (0.67-1.14)	0.88 (0.65-1.27)	0.388
ACE-I/ARB	61 (%47.3)	39 (%52.7)	0.275
Beta- blocker	68 (%52.7)	29 (%39.2)	0.043
Spirolactone	19 (%14.7)	9 (%12.2)	0.388
Loop diuretic	31 (%24)	24 (%32.4)	0.129
Oral anticoagulant	30 (%23.3)	32 (%43.2)	0.003
Acetyl salicylic acid	60 (%46.5)	18 (%24.3)	0.001
Statin	62 (%48.1)	20 (%20.7)	0.002
Glucose (mg/dl)	109 (97-146)	112 (96-162)	0.880
HbA1c (%)	6 (5.7-6.9)	6.2 (5.7-8)	0.620
Hemoglobin (g/dl)	13.7 (11.8-14.9)	12 (11.1-13)	<0.001
Hematocrit (%)	40.1±5.6	36.9±4.1	<0.001
WBC/mm ³	7.2±2.2	7.9±5.7	0.055
eGFR (ml/min)	74.2±21.2	62.2±21.3	0.001
Urea (mg/dl)	40 (31-50)	43 (34-54)	0.189
AST (U/L)	22±8	25±16	0.140
ALT (U/L)	20 ±13	23±27	0.262

Baseline characteristics of patients with mid range heart failure

OP-053 THE RELATIONSHIP CONTROLLING NUTRITIONAL STATUS SCORE AND HOSPITAL MORTALITY IN DECOMPENSATED HEART FAILURE PATIENTS

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Background and Aim; Chronic Heart Failure (HF) is sometimes complicated by malnutrition, which can lead to muscle weakness, cognitive impairment, dysphagia and cardiac cachexia. However, the clinical importance of nutritional status in acute HF patients requires further investigation. The Controlling Nutritional Status (CONUT) score, which is calculated from serum albumin and cholesterol levels, has been shown to predict adverse outcomes at different diseases. The aim of this study is to investigate the relationship between the controlling of nutritional status score (CONUT) and hospital mortality in patients with decompensated heart failure (DHF).

Methods: A total of 332 patients who hospitalized due to DHF between 2018-2019 in Pamukkale University Cardiology Department were analyzed retrospectively. Patients divided two groups as; discharge and deceased patients. CONUT score was used for evaluating nutritional status patients with DHF. This score uses three parameters; the serum albumin level (g/dl), total cholesterol level (mg/dl), total lymphocyte count (count/ml). In this way, it provides an evaluation of protein reserves, calorie depletion and immune defense score values were assigned to different ranges of laboratory measurements as follows: serum albumin ≥ 3.5 g / dL, 0 points; 3.49-3, 2 points; 2.99-2.5, 4 points; and < 2.5 , 6 points; lymphocytes ≥ 1600 μ L – 1.0 point; 1200–1599, 1 point; 800–1199, 2 points; and < 800 , 3 points; and total cholesterol ≥ 180 mg / dL, 0 point; 140-179, 1 point; 100–139, 2 points; and < 100 , 3 points. INA score of 0-1 was defined normal, while 2-4 was defined as mild CONUT, 5-8 as medium CONUT and ≥ 9 was severe- CONUT. A higher CONUT score indicates a worse nutritional status. Logistic Regression Analysis was used for determining which variables affects hospital mortality in DHF patients.

Results: Table 1 revealed that baseline characteristics and CONUT score of the study population. Table 2 shows that clinical features and laboratory findings of the study population. Systolic (106 ± 21.9 ; 117.4 ± 24.1 , $p < 0.001$) and diastolic blood pressure (mmhg) (65 ± 10.9 ; 68.6 ± 11.7 , $p = 0.003$) at admission, lymphocyte (K/uL) (1.2 ± 0.8 ; 1.3 ± 0.7 , $p = 0.016$), albumin (g/L) (3.4 ± 0.6 ; 3.6 ± 0.6 , $p = 0.007$), sodium (mmol/L) (135.2 ± 7.7 ; 136.9 ± 5.6 , $p = 0.02$), cholesterol (mg/dL) (125.8 ± 33.4 ; 143.3 ± 42.7 , $p = 0.001$) and GFR (mg/dL) (18.1 ± 13.5 ; 60.4 ± 23.7 , $p < 0.001$) levels were significantly lower in deceased patient group (respectively deceased patient, discharge patient). Red cell distribution width (%) (19.4 ± 17.8 ; 17.6 ± 15.3 , $p < 0.001$), urea (mg/dL) (82.5 ± 44.6 ; 69.2 ± 39.3 , $p = 0.012$) creatinine (mg/dL) (1.7 ± 0.9 ; 1.4 ± 0.7 , $p < 0.001$), c-reactive protein values (mg/L) (5.3 ± 5.8 ; 4.6 ± 6.5 , $p = 0.017$) and duration of hospital stay (day) (23.1 ± 10 ; 5.7 ± 3.6 , $p < 0.001$) were significantly higher in deceased patient group (respectively deceased patient, discharge patient). CONUT score was found to be statistically significantly higher in the deceased patient group ($p < 0.001$) and according to multiple logistic regression analysis (table 3), the moderate and severe CONUT score increases the in-hospital mortality 2.8 times compared to low or normal (O.R; 2.8, $p = 0.038$).

Conclusion: Moderate and severe CONUT score was independently associated with hospital mortality in DHF patients. Malnutrition increases in-hospital mortality at patients who hospitalized for DHF.

Sex n, (%)	Deceased Patient	Discharge Patient	p value
Female	29 (24)	92(76)	0.8
Male	48 (22.7)	163(77)	
Concomitant diseases n, (%)			
HT	39(49.4)	127(49.8)	0.944
DM	29(37.7)	123(49.8)	0.103
HL	12(15.6)	30 (11.8)	0.491
CAD	43(55.8)	153 (60)	0.516
CRF	19(24.7)	52(20.4)	0.519
Stroke	3(3.9)	10 (3.9)	1.000
COPD	15(19.5)	42(16.5)	0.659
Drug Using n, (%)			
ASA	30(39)	130(51.8)	0.049
P2Y12 inhibitors	13(16.9)	45(17.9)	0.968
Beta blockers	51(66.2)	175(69.7)	0.563
ACE/ARB	23 (29.9)	100(39.8)	0.148
Spironolactone	24 (31.2)	82(32.7)	0.915
Anticoagulant	31(41.3)	83(33.2)	0.256
Diuretic	84(70.1)	153(61)	0.185
CCB	18(23.4)	50(19.9)	0.621
Digoxin	15(19.5)	45 (17.9)	0.889

Table 1: Baseline Characteristics and CONUT score of the Study Population

Type of HF n, (%)			
REF-HF	44(24.4)	136(75.6)	0.530
MEF-HF	14(18.4)	62(81.6)	
PEF-HF	19(25)	57(75)	
CONUT scores			
Normal- mild	14 (8.1)	158(91.9)	<0.001*
Moderate-Severe	63(39.4)	97(60.6)	

HT; hypertension, DM; diabetes mellitus, HL; hyperlipidemia CAD; coronary artery disease CRF; chronic renal failure COPD; chronic obstructive pulmonary disease, ASA; acetylsalicylic acid ACE; angiotensin converting inhibitor ARB; angiotensin receptor blocker CCB; calcium channel blocker HF; heart failure, REF; reduced ejection fraction, MEF; moderate ejection fraction, PEF; preserved ejection fraction, CONUT; controlling nutritional status score

Table 2: Clinical Features and Laboratory Findings of the Study Population

	Deceased Patient (median± SS)	Discharge Patient (median± SS)	P value
Age	70.8± 10.2	68.5± 11.8	0.244
LVEF	36.6± 12.3	38 ± 11.7	0.378
Heart rate	94.1± 24.2	90.9 ±24.2	0.284
SBP at admission	106 ± 21.9	117.4 ±24.1	<0.001*
DBP at admission	65 ±10.9	68.6 ±11.7	0.003*
WBC	11±5.3	9.9 ± 3.7	0.128
Hemoglobin	12.9±10.9	23±121.2	0.617
Platelet	237.1± 113.6	234± 87.8	0.748
RDW	19.4± 17.8	17.6± 15.3	<0.001*
Glucose	153.1± 81.2	156.7± 85.6	0.701
Urea	82.5± 44.6	69.2± 39.3	0.012*
Creatinine	1.7±0.9	1.4± 0.7	<0.001*
Sodium	135.2± 7.7	136.9±5.6	0.02*
Potassium	4.6± 0.7	6.3±27.9	0.282
AST	83± 203	69.8± 195.4	0.318
CRP	5.3±5.8	4.6±6.5	0.017*
Magnesium	2±0.4	2±0.4	0.122
Uric acid	9.2±3.7	8.4±3.3	0.076
Lymphocyte	1.2±0.8	1.3±0.7	0.016*
Albumin	3.4±0.6	3.6±0.6	0.007*
Cholesterol	125.8±33.4	143.3±42.7	0.001*
GFR	18.1±13.5	60.4±23.7	<0.001*
Duration of hospital stay	23.1±10	5.7±3.6	<0.001*

LVEF; left ventricular ejection fraction, SBP; systolic blood pressure, DBP; diastolic blood pressure, WBC; white blood cell, RDW; red cell distribution width, AST; aspartate amino transferase, CRP; c reactive protein, GFR; glomerular filtration rate

Table 3: Multivariate logistic regression analysis for variables affecting hospital mortality

	O.R.	P value	95% C.I. for O.R.	
			Lower	Upper
Creatinine (mg/dl)	0.533	0.056	0.28	1.015
Duration of hospital stay	1.384	<0.001*	1.277	1.499
Moderate and severe CONUT score	2.805	0.038*	1.061	7.416

Model included; age, sex, concomitant diseases, systolic blood pressure at admission, red cell distribution width, creatinine, duration of hospital stay, CONUT (controlling nutritional status) score
*p< 0.05

OP-054 EVALUATION OF SLEEP QUALITY IN PATIENTS WITH REDUCED EJECTION FRACTION HEART FAILURE USING ANGIOTENSIN RECEPTOR NEPRILYSIN INHIBITOR

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Objectives: It is known that chronic heart failure (CHF) reduces sleep quality by causing sleep problems. In recent years, it has been observed that sacubitril-valsartan, which is an angiotensin receptor neprilysin inhibitor (ARNI), reduces mortality and hospitalization in CHF patients. The aim of our study is to examine whether sacubitril-valsartan has an effect on sleep quality in CHF patients apart from these benefits.

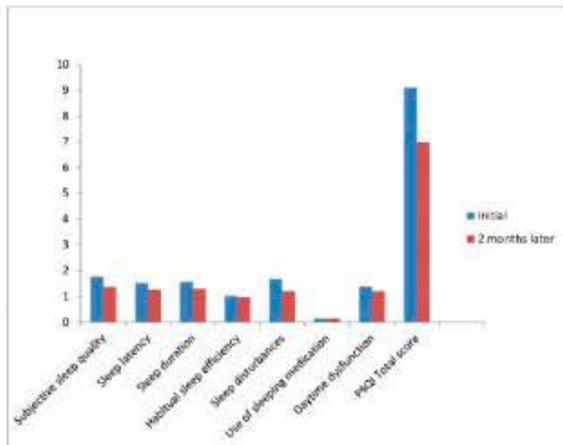
Method: In our study, 44 patients with a history of heart failure with reduced ejection fraction (HFrEF, EF<= 40%) who applied to our cardiology outpatient clinic of Gazi Yaşargil Training and Research Hospital were included. Demographic, clinical, laboratory, electrocardiography and echocardiographic parameters of these patients were examined. Sacubitril-valsartan treatment was initiated in each patient. Dose titration was performed in patients who could tolerate the treatment. Pittsburgh sleep quality index (PSQI) questionnaire was performed in all patients before treatment and at the end of the second month.

Results: The median age of the study population was 61.5 (47.2–70.7, IQR) years and 30 (68.2%) of them were male. There were 30 (72.7%) ischemic HF patients and 14 (27.3%) non-ischemic HF patients. After 2 months of ARNI treatment, there was a significant decrease in the total PSQI score of patients compared to the baseline [9.0 (7.0–2.0) vs 7.0 (5.0–9.0), p < 0.001].

Conclusion: Sleep problems in chronic heart failure patients are one of the unfavorable clinical conditions. According to our study, it can be said that sacubitril-valsartan treatment improves sleep quality in patients with HFrEF.

Keywords: ARNI, heart failure, sleep quality

Comparison of the total PSQI score consisting of seven components at initial and after treatment.



Frequency distribution of dimensions of sleep quality

	Initial	2 months later	p value
Subjective sleep quality			
Very good, n (%)	12(27.2)	26(59.1)	< 0.001
Good, n (%)	21(47.7)	11(25.0)	0.078
Bad, n (%)	8(18.2)	3(6.8)	0.125
Very bad, n (%)			
Sleep latency			
Never, n (%)	2(4.5)	2(4.5)	-
<once/week, n (%)	19(43.2)	30(68.2)	0.003
1-2times/week, n (%)	21(47.7)	10(22.7)	0.013
>=3times/week, n (%)	2(4.5)	2(4.5)	-
Sleep duration			
>7hours, n (%)	4(9.1)	8(18.2)	0.125
6-7hours, n (%)	18(40.1)	22(50.0)	0.388
5-6hours, n (%)	15(34.1)	9(20.5)	0.109
<5hours, n (%)	7(15.9)	5(11.3)	0.500
Habitual sleep efficiency			
>85%, n (%)	7(15.9)	9(20.5)	0.500
75-84%, n (%)	29(65.9)	29(65.9)	-
65-74%, n (%)	8(18.2)	6(13.6)	0.500
<64%, n (%)	0(0)	0(0)	-
Use of sleeping medication			
Never, n (%)	41(93.2)	41(93.2)	-
<once/week, n (%)	1(2.3)	1(2.3)	-
1-2times/week, n (%)	2(4.5)	2(4.5)	-
>=3times/week, n (%)	0(0)	0(0)	-
PSQI Total score >= 6, n (%)	36(81.2)	32(72.3)	0.125

PSQI: Pittsburgh sleep quality index

OP-055 IS THERE A DIFFERENT MECHANISM ON THE BASIS OF HEART FAILURE EMERGING IN SLEEP APNEA?: CORRELATION OF MYOCARDIAL EFFICIENCY INDEX WITH NT-PRO BNP

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Objective: We aim to evaluate the association between myocardial mechanoenergetics index (MEEi) and NT-Pro BNP in the early stages of obstructive sleep apnea (OSA).

Methods: In our cohort study, we included 81 eligible patients without cardiovascular disease (CV) newly diagnosed with OSA in a tertiary outpatient clinic between January 2013 and January 2014. We recorded the demographic data of the patients, blood test results, clinical characteristics, blood pressures, heart rates, conventional echocardiography, and NT-Pro BNP measurements. OSA is diagnosed and categorized into mild, moderate and severe OSA groups according to Apnea-Hypopnea Index (AHI). MEEi could be stated as the ideal amount of blood pumped with one heartbeat in 1 sn in 1 gram myocardium, simply calculated as stroke volume (SV)/heart rate (HR) ratio and normalized by left ventricular (LV) mass.

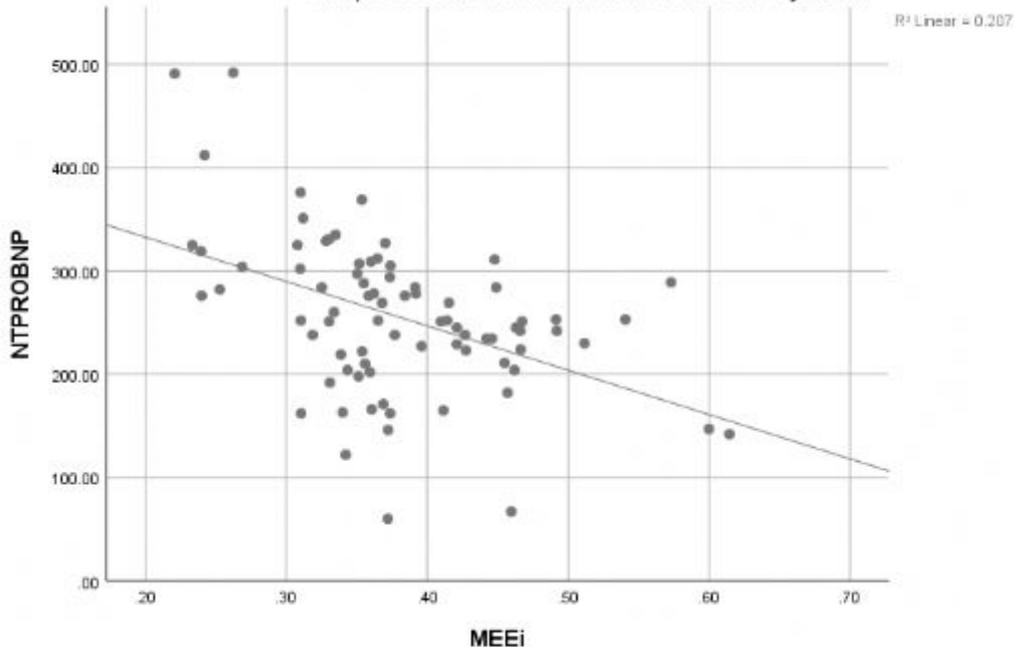
Results: The mean age was 49.4±12.6, and the male/female ratio was 50/31. Demographic, clinical variables of the study population could be seen in Table 1. Hypertension (p=0.046) and systolic(p=0.003) and diastolic blood pressures(p<0.001) were more frequent in the OSA group. And, OSA patients interventricular(p=0.001) and posterior(p<0.001) wall dimensions were more hypertrophic. MEEi was lower in OSA patients compared with non-OSA patients (0.41±0.06 vs. 0.36±0.08). However, it does not reach statistical significance (p=0.384). MEEi was correlated with NT-Pro BNP (r:-0.455,p<0.001).

Conclusions: For the first time, MEEi negative correlation with NT-Pro BNP showed in newly diagnosed OSA patients. Moreover, MEEi may reflect a disturbance in production and energy use in the hypertrophied myocardium. Therefore, MEEi and NT-Pro BNP association may explain the mechanism before apparent heart failure development in patients with OSA.

Keywords: Myocardial Energetics, Myocardial Mechanoenergetic Efficiency Index, NT-Pro BNP, Obstructive Sleep Apnea

Demographic, clinical variables of the study population

Simple Scatter with Fit Line of NTPROBNP by MEEi



Abbreviations: MEEi: Myocardial mechanoenergetic efficiency index, NT-Pro BNP: N- terminal pro brain natriuretic peptide

Demographic, clinical variables of the study population

	Non-OSA (n=22)	OSA (n=59)	p-value
Age (year)	47.1 ±12.6	50.3±12.6	0.836
Sex (M/F)	14/8	39/20	0.829
Smoking (%)	8(36)	29(49)	0.076*
DM (%)	7(31)	20(34)	0.423*
HT (%)	9(41)	41(69)	0.046*
Obesity (%)	10(45)	33(56)	0.459*
BMI (kg/m2)	29.77±7.14	32.01±6.09	0.347
WCir(cm)	98.4±16.88	99.95±23.14	0.067
HR(beat per minute)	73.22±9.28	74.32±6.46	0.083
SBP(mmHg)	123.90±7.21	128.83±8.39	0.003
DBP(mmHg)	80.86±3.35	85.71±5.71	<0.001
AHI	1.4±1.76	34.26±21.80	<0,001
NT-ProBNP	217.890 29.148	266.874 84.315	0.001
LVD	47.87±3.10	47.91±3.48	0.912
LVS	28.28±3.16	28.55±3.96	0.341
IVS	10.97±1.34	11.22±1.34	0.001
PW	9.87±0.92	10.35±1.22	<0.001
Ao	29.57±4.61	30.00±4.14	0.385
LA	38.46±4.02	38.07±3.65	0.842
LV EF(%)	65.00±4.12	63.45±3.73	0.116
Stroke Volume	90.54±8.27	80.05±10.09	0.001
Stroke Work	150.80±18.40	161.27±28.86	0.007
LVM	174.22±25.02	204.67±43.06	0.003
MEE	70.87±11.33	72.84±12.23	0.427
MEEi	0.41±0,06	0.36±0.08	0.384

Abbreviations: OSA: Obstructive sleep apnea M/F: Men/Female, DM: Diabetes mellitus, HT: Hypertension, MetS: Metabolic syndrome, BMI: Body mass index, WCir: Waist circumference, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, AHI: Apnea-hypopnea index, NT-Pro BNP: N-Terminal Pro Brain Natriuretic Peptide, LVD: Left ventricular diastolic dimension, LVS: Left ventricular systolic dimension/IVS: Interventricular septum, PW: Posterior wall, Ao: Aortic dimension, LA: Left atrial dimension, LV EF: Left ventricular ejection fraction/LVM: Left ventricular mass, MEE: Myocardial mechanoenergetic efficiency, MEEi: Myocardial mechanoenergetic efficiency index, OSA: Obstructive sleep apnea. *= Tested with Chi-square and Fisher exact tests.

OP-057 PERICARDIAL EFFUSION AND CARDIAC TAMPONADE AFTER COVID-19 VACCINE: A RARE CASE

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Introduction: Mounting evidence is now supporting that COVID-19 affects the cardiovascular system with acute cardiac injury, high risk of thrombosis including stroke, pulmonary embolism, and acute coronary syndrome. Conversely, very few attention has been paid to pericardial effusion (PE).

There is no case report of pericardial effusion and tamponade developing after covid-19 vaccine (pfizer-biontech) in the literature.

Case Presentation: A 44-year-old female patient with no known history of chronic disease was admitted to the emergency room with tachycardia and shortness of breath after covid-19 vaccine (Pfizer Biontech). The blood pressure of the patient who was consulted to cardiology: 80/40 mmHg. Electrocardiography (ECG) findings were consistent with sinus tachycardia, heart rate of 102 beats/min and low voltage (Figure 1). In the patient's laboratory parameters; ALT 65.5 U/L (reference:0-33 U/L), AST 57 U/L (reference:0-32 U/L), CRP 28.44 mg/L (reference: 0-5 mg/L) was detected. Troponin and other blood values of the patient were within normal reference ranges. Echocardiography (ECHO): Ejection Fraction was 65%, pericardial effusion (PE) was present at its widest point, 5 cm encircling the heart, collapsing the right heart cavities in diastole, forming the clinic of tamponade (Figure 2). Emergency pericardiocentesis was applied to the patient. Pericardial fluid content of the patient was serous. The patient, whose clinical stability was stabilized after pericardiocentesis, was followed in the coronary intensive care unit. No pericardial effusion was observed in the control echo during follow-up. The patient's PCR results were negative. The patient was discharged with cardiology outpatient control.

Discussion: Although the pathophysiology is not completely understood, current literature

attributes the development of pericardial effusion in COVID-19 patients to the systemic inflammatory response and subsequent cytotoxic and immune-mediated effects related to SARS-COV-2. There is no case report of pericardial effusion and tamponade developing after covid-19 vaccine (pfizer-biontech) in the literature. Whether these vaccines, which were approved for immediate use due to the pandemic, have such effects requires further research and similar case examples.

Conclusions: Presumably, There is a higher incidence of COVID-19-related cardiac diseases such as pericarditis that can manifest from minimal PE to cardiac tamponade. Cardiologists and emergency physicians should be aware and extensively look for PE at the time of the COVID-19 outbreak. Whether these vaccines, which were approved for immediate use due to the pandemic, have such effects requires further research and similar case examples. However, it should not be forgotten that the only and effective way to prevent the pandemic is still vaccines, even though there are these and similar side effects.

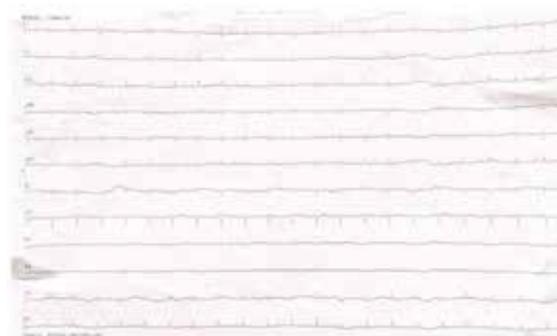
Keywords: Covid-19, Pericardial effusion, Vaccine, Cardiac tamponade

Echocardiography



Echocardiography (ECHO): Ejection Fraction was 65%, pericardial effusion (PE) was present at its widest point, 5 cm encircling the heart, collapsing the right heart cavities in diastole, forming the clinic of tamponade

Electrocardiography (ECG)



Electrocardiography (ECG) findings were consistent with sinus tachycardia, heart rate of 102 beats/min and low voltage

OP-058 FREE-FLOATING RIGHT ATRIAL THROMBUS WITH ACUTE PULMONARY EMBOLISM TREATED WITH HEPARIN INFUSION

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Abstract With increasing age, the risk of pulmonary embolism and mortality increases. A free-floating right atrial thrombus is usually associated with pulmonary embolism. The diagnosis of pulmonary embolism is difficult and frequently missed because elderly patients may attribute nonspecific symptoms to an underlying cardiopulmonary disease. The presence of a right atrial thrombus with pulmonary embolism is a critical medical emergency; treatment options include surgical thrombectomy, thrombolysis and anticoagulation. Anticoagulation alone can be successfully used in elderly patients who are at high risk for bleeding due to thrombolytic therapy. We present the case of an 89-year-old woman whose echocardiography showed a free-floating right atrial thrombus. The condition was managed successfully with anticoagulant therapy using heparin infusion.

Introduction The presence of a right atrial thrombus with pulmonary embolism (PE) is a critical medical emergency. The finding of a free-floating right atrial thrombus is rare, and its prevalence in the context of PE ranges from 7% to 18%. In patients with a right atrial thrombus, a snake-like, nonuniform, bulky mass that mimics myxoma and moves from the right atrium to the right ventricle is generally observed. Echocardiography helps detect the thrombus and evaluate right ventricle function; thus, it can help in selecting the appropriate treatment. Treatment options include surgical thrombectomy, thrombolysis and anticoagulation. This case report highlights the importance of using echocardiography for the diagnosis of right atrial thrombus with PE and including heparin infusion in its treatment.

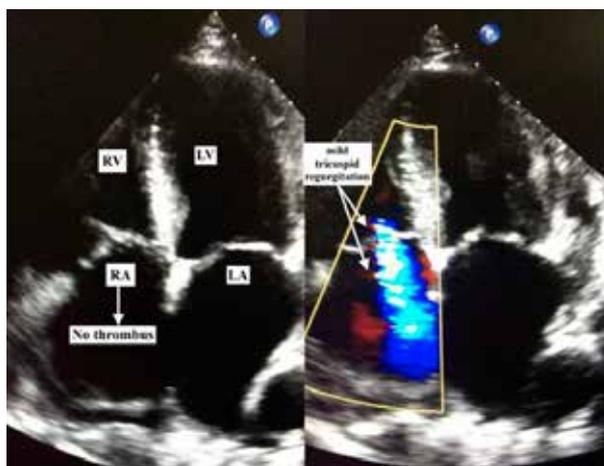
Report of the Case We report the case of an 89-year-old woman with a history of hypertension. She presented to the emergency department with palpitation and acute dyspnoea that had been increasing progressively over the previous 5 days. On physical examination, it was observed that the pulse rate was arrhythmic (155 bpm), respiration rate was 28 breaths per minute, oxygen saturation was 85% on room air and arterial blood pressure was 100/70 mmHg; 3/6 systolic murmur was noted in the tricuspid area during cardiac auscultation. Further examination of her respiratory system revealed a decreased respiratory sound in both lungs. Electrocardiography showed atrial fibrillation with a high ventricular response and left bundle branch block. No pathological appearance was detected on chest X-ray except mild cardiomegaly. Biochemical examination revealed high D-dimer levels of 4,860 µg/L (reference range: 0–500 µg/L) and hs-troponin-I levels of 622.7 ng/L (reference range: 0–11.6 ng/L). Transthoracic echocardiography (TTE) was performed using a Philips iE33 echocardiography machine and X5 transducer (Philips Healthcare, Andover, MA, USA). This revealed a free-floating, snake-like thrombus in the right atrium, with moderate systolic right ventricular dysfunction, a coaptation defect on the tricuspid valve and pulmonary hypertension (pulmonary artery systolic pressure, 85 mmHg) (Figure 1, Video 1 and 2). The patient had a normal left ventricular function (ejection fraction of 55%). Venous Doppler imaging of the lower extremities showed the presence of a floating thrombus in the left common femoral vein extending up to 4 cm to the left superficial femoral vein and saphenofemoral junction. Angiographic computed tomography (CT) suggested signs of PE at the level of the segmental branches, with extension to the subsegmental arteries. The patient was transferred to the coronary intensive care unit for treatment. The cardiovascular surgery department was consulted regarding the possibility of the right atrial thrombus resection. Her haemodynamics were stable; hence, medical follow-up was primarily planned in the coronary intensive care unit, without using a thrombolytic agent. She was administered a bolus dose of unfractionated heparin (80 IU/kg), followed by continuous intravenous heparin infusion (18 IU/kg/h). The dosage was adjusted by controlling the activated partial thromboplastin time (aPTT) to be 1.5–2.5 times every 6 hours. Forty-eight hours after heparin infusion, repeated TTE revealed a complete regression of the right atrial thrombus, decreasing tricuspid regurgitation and improved right systolic dysfunction and pulmonary hypertension (pulmonary artery systolic pressure, 40 mmHg) (Figure 2, Video 3 and 4). Subcutaneous enoxaparin 2 × 0.8 mL was initiated after stopping heparin infusion. The general condition of the patient improved after 1 week, and she was discharged with a prescription of rivaroxaban because of her self-reported inability to adhere to the international normalised ratio follow-up. A whole-body CT scan did not reveal the presence of any underlying malignancy. Screening for thrombophilia also showed negative results. The patient was discharged on day 7. Written informed consent was obtained from the patient for this case report.

Discussion Intracavitary masses such as right heart thrombus, tumours and vegetation are rare and are often detected incidentally. These are generally seen as an irregular, often thin and long, highly moving, serpentine masses that move from the right atrium to the right ventricle and mimic a myxoma. These may occur locally in the right atrium and are also called as ‘emboli-in-transit’ due to their progression from the deep veins to the right atrium via embolisation. The tricuspid valve in the right atrium, the Eustachian valve, patent foramen ovale and the interatrial septum form the basis for the attachment of the thrombus to the right atrium. Pulmonary thromboembolism is usually caused by deep vein thrombosis in the lower extremities. The presence of a right atrial thrombus with acute PE is a critical medical emergency and is associated with a higher mortality rate than acute PE alone. The resulting mortality rate has been reported to be ranging from 27% to 100% without treatment, with the prevalence rate ranging from 7% to 18%. TTE is a noninvasive procedure and can be used at the bedside for the diagnosis of PE and can aid in the prediction of patient prognosis. In our patient, TTE revealed a free-floating, snake-like thrombus in the right atrium, with moderate systolic right ventricular dysfunction and a coaptation defect on the tricuspid valve. The optimal management of right atrial thrombus remains controversial; however, treatment should be initiated immediately. Treatment options include thrombolytic therapy, surgical embolectomy and anticoagulation. There is no clear consensus on the ideal treatment option, and mortality rates vary according to the treatment method used. In some studies, similar mortality rates have been reported with all three treatment options. According to the available guidelines from the European Society of Cardiology and the American Society of Thoracic Surgery, patients with both right heart thrombus and massive PE, regardless of haemodynamic instability, are classified to be at high risk, with a recommendation for urgent treatment. Thrombolytic therapy is a readily available treatment option and is indicated for use in patients with haemodynamic dysfunction, hypotension and shock symptoms as well as general condition disorders due to PE. Nkoke et al. described a case of right atrial thrombus and bilateral massive PE that was treated successfully with tenecteplase. A study comparing thrombolytic therapy with heparin in patients with stable haemodynamics showed same overall mortality rate with both treatments. Heparin infusion was planned for our patient because she had stable haemodynamics. Lohrmann et al. reported the case of a right atrial thrombus with bilateral proximal PE that was treated with thrombectomy and bilateral pulmonary endarterectomy. With surgical embolectomy, mortality rates range between 20% and 50% even in centres with highly experienced professionals. Thus, surgical embolectomy may be a suitable treatment option in cases where medical therapy is contraindicated. Anticoagulation should be used in all cases, including those of right atrial thrombus and PE, after thrombolytic therapy or surgical embolectomy. Anticoagulation alone has been successfully used in elderly patients who are considered at a high risk for bleeding with thrombolytic therapy. Temtanakitpaisan et al. reported the case of a 92-year-old male having a right atrial thrombus with PE who was successfully treated with heparin alone. In our case as well, the elderly patient at a high risk of bleeding was treated with heparin only. Treatment of patients with free-floating right atrial thrombus should be started as soon as the diagnosis is made. The role of TTE is very important in the diagnosis, determining prognosis and treatment planning of these patients. TTE showing thrombus in the right atrium is a sign of a poor prognosis. As shown in our case, heparin infusion in the right atrial thrombus can be considered as an alternative approach to thrombolytic therapy and surgical embolectomy in haemodynamically stable elderly patients at a high risk of bleeding.

Figure 1, Right Atrial Thrombus and Severe Tricuspid Regurgitation



Figure 2, 48 Hours After Heparin Infusion



OP-060 A CASE OF ACUTE UPPER LIMB ISCHEMIA IN A PATIENT WITH COVID-19

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The Coronavirus disease 2019 (COVID-19) is an emerging condition that affects the whole world and causes life-threatening clinical characteristics ranging from asymptomatic cases to severe symptoms. Although initially it is assumed to be a respiratory disease, it has been shown to cause many different manifestations. One of these different manifestations is hypercoagulopathy. Herein, we report a case of limb ischemia developed in a patient who has been treated in the intensive care unit due to COVID-19 pneumonia. A 72-year-old female patient with a history of diabetes mellitus presented to the emergency department with complaints of shortness of breath and cough. Chest computed tomography demonstrated bilateral ground-glass opacities and consolidation areas characteristic of viral pneumonia (Figure 1). Then, a nasopharyngeal swab was taken from the patient, and COVID-19 was diagnosed based on rapid polymerase chain reaction testing. On hospital day 6, her D-dimer was noted to be elevated to 19.177 ng/mL on her routine daily labs. Physical examination demonstrated that her left arm was cold with palpation and purple in appearance with a well-demarcation line (Figure 2). The fingers were necrotic. her clinical signs suggested the diagnosis of acute limb ischemia. This case report highlights that it is crucial to monitor for thromboembolic events in the extremities of critically ill patients, especially in cases with significantly increased levels of D-dimer.

Keywords: limb ischemia, COVID-19, hypercoagulopathy

Figure 1

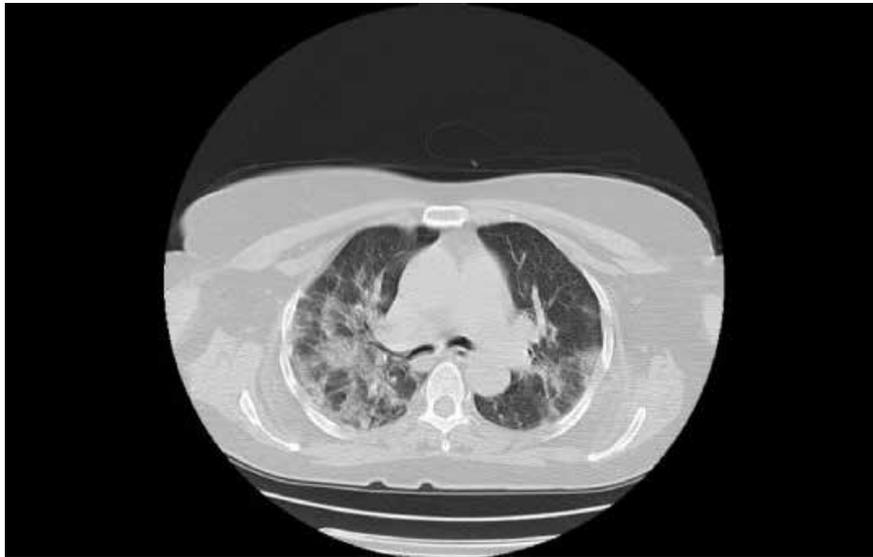


Figure 2



OP-061 A DIFFERENT APPROACH IN THE DIFFERENTIAL DIAGNOSIS OF THE PATIENT WITH CHRONIC DIARRHEA, TTR AMILOIDOSIS

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INTRODUCTION: Amyloidosis is a disease with severe progressive organ dysfunction characterized by the storage of amyloid fibrils in various organs resulting from improper folding of proteins in the body. (1-2) 28 different proteins it has been shown that it can be stored as amyloid. The types are determined accordingly (AL, TTR, AA etc.) (1-2) Diagnosis can be made by imaging methods (echocardiography-MRI-bone scintigraphy), tissue biopsy, and genetic analysis. (1) Here in we present a patient with accompanied of chronic diarrhea, who was diagnosed with an amyloidosis with cardiac involvement after examining many years for different etiologies

CASE REPORT: A 42-year-old male patient was admitted to outpatient clinic of internal medicine with a complaint of diarrhea. In his history he was also admitted various clinic due to low blood pressure, dizziness and fainting twice, about four years ago. He had no previously known chronic illness otherwise 20 packet years of smoking. He had no obvious neurologic reason for syncope as it was evaluated by neurologist. According to hospital records, patient was also admitted to urology and gastroenterology clinic due to erectile dysfunction, and chronic diarrhea respectively. While colonoscopy revealed unremarkable findings, endoscopy demonstrated erythematous pangastritis. Patient was administrated Helicobacter Pylori eradication treatment. During the follow-up, the patient had still have a complain of diarrhea, and hospitalized internal medicine. He was consulted to the cardiology clinic as he was previously diagnosed with hypertrophic cardiomyopathy. His Electrocardiogram (ECG) was in sinus rhythm with hypovoltage. Laboratory parameters were unremarkable. In the past he was diagnosed with hypertrophic cardiomyopathy without obstruction. No significant arrhythmia was observed on rhythm Holter monitorization. Control echocardiography revealed ejection fraction of 70%, severe left ventricle hypertrophy (interventricular septum thickness: 24 mm, posterior wall thickness:16 mm), right ventricle hypertrophy, increased thickness of mitral valve and interatrial septum and grade 2 diastolic dysfunction. (image 1-2). Global longitudinal strain was 18.5% with cheery on the top pattern. Based on these findings and suspicion of amyloidosis. A rectal biopsy resulted in favor of amyloidosis. Grade 2-3 cardiac involvement was observed in technetium whole body bone scintigraphy. Monoclonal gammaglobinopathy was ruled out as a result of immune electrophoresis in the patient's serum and urine. As a result of the genetic analysis of the patient, a heterozygous mutation c.200G>A (p.Gly67Glurs121918090) was detected in the TTR gene exon 1. The patient began to be treated with 80 mg of TTR stabilizing agent Tafamidis "Vyndaquel" per day. He was partially respond to medical treatment.

DISCUSSION: Difficulties in the diagnostic process and limitations in specific treatment for amyloid are the most important obstacles to the clinician's course of diagnosis. However, amyloidosis is a disease of increasing diagnostic awareness. In amyloidosis, where cardiac involvement is present, the prognosis is poor but it has been shown that the prognosis can be significantly corrected with early diagnosis and treatment approaches. For this, it is believed that chemotherapy combinations that reduce free light chain proteins in AL-amyloidosis and anti-fibrillar treatments that act through different mechanisms can be used in TTR-amyloidosis. (1-3)

RESULT: In patients with wall thickening (especially involving the valves) and advanced stage of diastolic dysfunction in echocardiography, Infiltrative cardiomyopathies should be kept in mind as a differential diagnosis. Right ventricular hypertrophy, pericardial fluid, thickening of the interatrial septum and sparkling pattern are red flag echocardiographic findings supporting amyloidosis.

Keywords: Amiloidosis, Cardiac Amiloidosis, Chronic diarrhea, TTR Amiloidosis

SOURCE:

1. Cardiac amyloidosis: Recent advances in the diagnosis and therapy Yüksel Çavuşoğlu, Ebru Özpelit, Ahmet Çelik, Barış İkitimur, Meral Kayıkçioğlu, Lale Tokgözoğlu, Omaç Tüfekçioğlu, Mehmet Birhan Yılmaz. Cardiac amyloidosis: Recent advances in the diagnosis and therapy. Turk Kardiyol Dern Ars. 2019; 47(2): 1-34
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Image-1: IVS-PW thickening in PLAX view, thickening in accompanying valves



Hypertrophic IVS and PW in parasternal long axle image in transthoracic echocardiography, thickening of the accompanying mitral valves

Image-2: Thickening of IAS in the PSAX view



In transthoracic echocardiography, thickening of interatrial septum and tricuspid valves in Parasternal short axle image

Image-3: Right ventricular hypertrophy in SC view



In transthoracic echocardiography, right ventricular hypertrophy and pericardial fluid in subcostal view

OP-062 A RARE COMPLICATION OF SURGICAL ATRIAL SEPTAL DEFECT REPAIR: IATROGENIC DIVERSION OF THE INFERIOR VENA CAVA INTO THE LEFT ATRIUM

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Introduction: Atrial septal defect (ASD) accounts for 7-10% of all congenital heart diseases in children and It is also responsible for 30-33% of defects in adults with congenital heart diseases. Until the definition of percutaneous ASD closure in 1970 in the treatment of secundum ASD, Surgical ASD closure has been the mainstay of treatment for decades. Although transcatheter therapy is at the forefront in the treatment of secundum ASD today, for non-secundum ASD types and in the presence of additional pathologies or in anatomies not suitable for percutaneous closure, the surgical option is the only choice.

In here we will present a hemodynamic unstable state and hypoxia due to the suturing of the inferior vena cava during the minimally invasive surgical procedure of an ASD.

Case: In a 51-year-old woman suffering from dyspnea. On the detection of dilated right ventricle(RV)/ right atrium (RA) and an aneurysm on interatrialseptum(IAS) on transthoracic echocardiography (TTE), transesophageal echocardiography (TEE) performed and two different ASD-compatible defects are observed Surgery is recommended to the patient because the rims are insufficient and the septum is aneurysmatic.

After the minimally invasive surgical procedure, the patient is taken to the intensive care unit as intubated, but hypoxia and hypotension are observed in the follow-ups, inotropic support cannot be discontinued. TEE is performed because IVC cannot be clearly selected in the TTE.

On TEE, IVC diameter was 25 mm. IVC drains into left atrium (LA). On the bicaval image, there was a poor doppler current between RA and inferior vena cava (IVC). It was confirmed by contrast echocardiography that all microbubbles drains to LA from IVC.

In the current situation, it was understood that the IVC was sutured due to the closeness poor hemodynamic and hypoxic picture improved.

Discussion: Iatrogenic IVC to the left atrium during surgical closure of an ASD is a very rare complication but situations such as minimally invasive procedure from a limited area and the area between the ASD an IVC may predispose of this problem.

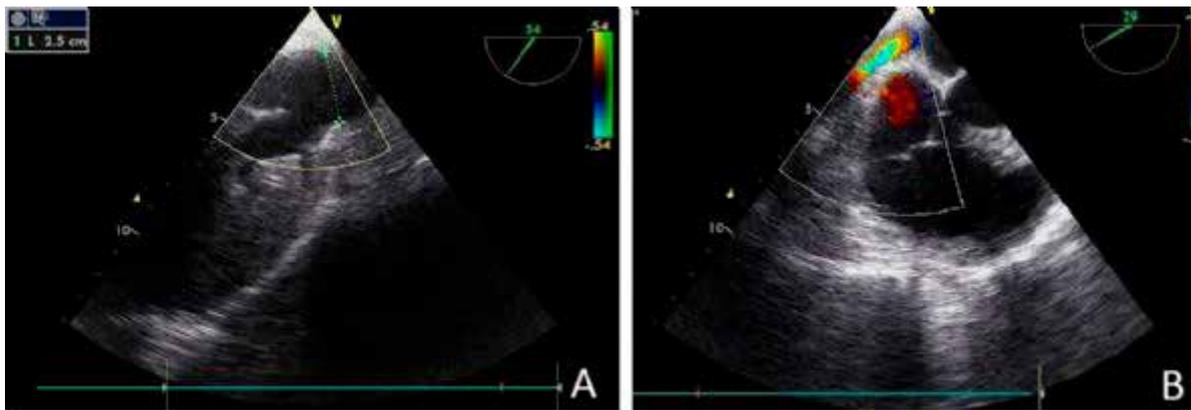
Multiple case reports have described this iatrogenic diversion of IVC flow to the LA in the repair of a low-lying ASD and also with inferior vena caval type ASD repair.

Depending on the amount of blood flow reaching the right atrium, hypoxia, cyanosis, hypotension, even paradoxical embolism, and Eisenmeger's syndrome may be seen in delayed cases.

In our patient, it was noticed and corrected in the early period. Performing the procedure with imaging in selected patients will help prevent such complications.

Keywords: atrial septal defect, complication, diversion

Figure



On the left figure, dilated inferior vena cava can be observed (A), also on the right figure on doppler image, the main drainage was to left atrium with a poor doppler current to right atrium can be observed(B)

Table 1. Demographic laboratory and echocardiographic parameters of the patients

	Total(n=72)	Congenital myopathy(n=24)	Control Group(n=48)	P value
Age (year)	26 (19-35)	30(21-39)	25(19-35)	0.126
Gender (female)	40(55 %)	15(62%)	28(58 %)	0.834
Body surface area (m2)	1.73(1.55-1.93)	1.74 (1.55-1.96)	1.72(1.54-1.91)	0.646
Body mass index (kg/m2)	23.1(21.2-25.7)	23.2(21.7-28.2)	23.1(20.9-25.2)	0.221
Creatinine(mg/dL)	0.69(0.60-0.83)	0.62(0.51-0.69)	0.79(0.65-0.92)	<0.001
Creatine kinase (U/L)	119(88-238)	309(162-712)	104(72-123)	<0.001
Hemoglobin(gr/dL)	14.0(13.1-15.8)	14.4(13.5-15.8)	14.0(12.8-16.0)	0.659
White blood cell (103/uL)	7.80(6.43-9.30)	7.84(6.62-9.30)	7.66(6.23-9.33)	0.865
LV diastolic diameter (mm)	45(42-48)	45(41-48)	46(42-48)	0.529
LV systolic diameter (mm)	27(26-29)	27(25-28)	27(26-29)	0.904
Septum thickness (mm)	9(8-10)	10(8-11)	9(8-10)	0.151
LV posterior Wall thickness (mm)	9(8-10)	9(8-10)	9(8-10)	0.759
LV diastolic volume index (ml/m2)	55(50-61)	51(48-61)	57(52-62)	0.109
LV systolic volume index (ml/m2)	20(18-23)	21(17-24)	20(18-22)	0.424
Biplane LV Ejection Fraction %	64(62-66)	62 (59-65)	64(63-66)	0.002
Apical 4 chamber LS %	-19.7(-19.3, -21.1)	-19.5(-17.6,20.6)	-19.9(-18.9, -21.2)	0.152
Apical 2 chamber LS %	-20.2(-19.3, -22.0)	-19.6(-18.2, -20.8)	-20.6(-19.4, -22.6)	0.011
Apical 3 chamber LS %	-20.0(-18.6, -21.7)	-19.3(-18.2, -20.4)	-20.3(-18.9, -21.9)	0.022
LV GLS %	-20.2(-19.1, -21.2)	-19.8(-18.4, -20.6)	-20.5(-19.3, -21.9)	0.022
LV GLS-endocardium %	-23.2(-22.0, -24.3)	-22.4(-21.1, -23.9)	-23.4(-22.5, -25.0)	0.027
LV GLS-epicardium %	-17.5(-16.6, -18.5)	-17.4(-15.4, -18.1)	-17.6(-16.8, -18.9)	0.062
Global Circumferential Strain %	-21.6(-19.9, -22.5)	-21.1(-18.9, -22.1)	-21.7(-20.4, -22.9)	0.116
Global Radial Strain %	45(39-49)	43(37-47)	46(40-49)	0.098
RV GLS total %	-23.4(-21.0, -25.1)	-21.2(-19.4, -24.7)	-23.9(-22.4, -25.6)	0.005
RV free wall longitudinal strain %	-27.9(-25.3, -29.9)	-26.6(-22.4, -29.2)	-28.4(-26.1, -30.2)	0.035
Heart rate (beat/min)	87(76-94)	85(75-98)	88(78-94)	0.603

Continuous variables are presented given as mean±SD or median (interquartile range) and categorical variables were expressed as number (%). LV, Left ventricle; GLS, Global Longitudinal Strain; LS, Longitudinal Strain; RV, Right Ventricle

OP-064 DECREASED INTRACARDIAC VOLUME RESERVE AND INCREASED SYMPATHETIC RESPONSE IN THE MECHANISM OF VASOVAGAL SYNCOPE: A COMPREHENSIVE ECHOCARDIOGRAPHIC AND HOLTER ECG MONITORING STUDY

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Background and aim: Despite the prevalence and associated morbidity of syncope, its mechanisms are not well understood and as a result, effective treatments have not been developed. We investigated the relationship between intracardiac volumes, deformation parameters and autonomic response with syncope in tilt table positive patients.

Methods: The study included 48 patients who underwent tilt table test due to syncope and diagnosed with vasovagal syncope (VVS) in Istanbul University Istanbul Faculty of Medicine Cardiology outpatient clinic, and 43 healthy controls whose age, sex, body-mass index (BMI) and body surface area (BSA) were matched. All subjects underwent 2D transthoracic and speckle tracking strain echocardiography using Philips IE33 ultrasound system. 24-hour Holter ECG monitoring was performed for heart rate variability (HRV) analysis.

Results: The mean age in the VVS group was 32.9 ± 9.3 years and female gender predominant ($n=37$, 77%). Age, gender, BMI, BSA and smoking were similar between VVS and control groups. Left ventricular (LV) end-diastolic volume index (EDVi), end-systolic volume index (ESVi), stroke volume index (SVi) were significantly lower in VVS group compared to controls (72.3 ± 10.7 vs. 72.3 ± 10.7 ml/m², $p<0.001$; 27.6 ± 5.3 vs. 31.2 ± 6.3 ml/m², $p = 0.004$; 44.5 ± 9.9 vs. 51.6 ± 9.1 ml/m², $p = 0.001$; respectively), while LV ejection fraction (EF), end-diastolic and end-systolic diameters were similar. Left atrium (LA) diameter and LA volume index (LAVI) were lower in VVS group compared to controls (30.2 ± 3.7 vs. 32.8 ± 3.9 mm, $p = 0.002$; 18.5 ± 4.5 vs. 24.6 ± 8.5 ml/m², $p<0.001$; respectively). Mitral A wave velocity was lower and septal e' velocity was higher in VVS group compared to controls (43.1 ± 13.1 vs. 43.1 ± 13.1 cm/sn, $p<0.001$; 10.4 ± 2.9 vs. 8.9 ± 2.5 cm/sn, $p = 0.016$; respectively). LV global longitudinal strain (GLS) and LA GLS were better in VVS group compared to controls (-19.6 ± 1.9 vs. $-18.6 \pm 2.5\%$, $p = 0.037$; 49.0 ± 15.8 vs. $43.7 \pm 6.2\%$, $p = 0.042$; respectively). Mean heart rate (HR), SDNN-24h, SDNN-index and pNN50 were higher in VVS group compared to controls (80.9 ± 9.9 vs. 73.3 ± 8.9 /min, $p<0.001$; 155.9 ± 33.4 vs. 129.7 ± 29.1 msn, $p<0.001$; 58.7 ± 20.6 vs. 43.3 ± 13.7 msn, $p<0.001$; 8.8 ± 8.7 vs. $5.5 \pm 4.7\%$, $p = 0.028$). In ROC analysis LAVI < 18.1 ml/m² predicts VVS with 72% sensitivity and 61% specificity (AUC=0.724, 95% CI: 0.616-0.831, $p<0.001$). LV EDVi < 73.5 ml/m² predicts VVS with 88% sensitivity and 65% specificity (AUC=0.776, 95% CI: 0.678-0.875, $p<0.001$).

Conclusion: We demonstrated small LV volumes and LAVI are predictors of VVS. In this study, increased mean HR, HRV, and myocardial contractility indicate high sympathetic stimulation in patients with VVS which is necessary to maintain cardiac output in patients with small LA volume and low intracardiac volume reserve. The use of β -blockers given to reduce myocardial contractility and activation of intracardiac mechanoreceptors is often not beneficial. Because methods to increase cardiac volume in these patients; for example, regular isometric exercise such as endurance training can prevent syncope. This findings may guide the management of patients with VVS.

Key words: Vasovagal syncope, left atrial volume index, left ventricular volume, heart rate

OP-065 SUBCLINICAL MYOCARDIAL SYSTOLIC FUNCTION IN PATIENTS WITH ARTERIOVENOUS FISTULAS AFTER RENAL TRANSPLANTATION

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Aims: Arteriovenous fistulas (AVFs) cause significant impairment on cardiac functions. We aimed to determine myocardial systolic function using speckle tracking echocardiography (STE) based strain (S) analysis and real time three dimensional echocardiography (3DE), in patients with AVFs after renal transplantation (Tx).

Subjects and Methods: We studied 20 patients (55±14 years, 57% male) and 20 age and sex matched healthy controls. Two dimensional STE based strain analysis was performed for left ventricle (LV), right ventricle (RV) and left atrium (LA). Reservoir phase strain, conduit (SR-C), and contractile phase (SR-A) strain rates for LA were obtained.

Results: There was no difference between the patients and the control group, regarding conventional echocardiographic parameters including LV ejection fraction (EF), LV end diastolic and end systolic volumes by 3DE. LV mass index was increased in patient group (123.31±15.04 g/m²; 95.2±0.01 g/m², p=0.0001) Tricuspidal annular systolic excursion (TAPSE)(21.68±2.54 mm; 23±0.05 mm, p=0.02) and RV peak systolic velocity (Sa) (0.12±0.02 m/s; 0.14±0.01 m/s, p=0.0001) were significantly reduced. RV global longitudinal strain was also decreased in patients (17.87±3.45 % to 24.07±0.05 %; p=0.0001). LA SR-C and SR-A were significantly increased (4.57±0.44 s⁻¹ to 2.59±0.51 s⁻¹; p= 0.0001 and 3.47±0.35 s⁻¹ to 2.64±0.50 s⁻¹; p=0.0001), compared to healthy controls.

Conclusions: Patients with AVFs have impaired myocardial systolic function regarding both RV and LV, while LA functions are enhanced. Real time 3DE and STE based strain imaging are detailed techniques which provide additive data about cardiac mechanics.

Keywords: speckle tracking echocardiography, myocardial dysfunction, av fistula

OP-066 CHA2DS2–VASC SCORE IS HIGHER IN PATIENTS WITH LEFT ATRIAL APPENDAGE THROMBUS ON TRANSESOPHAGEAL ECHOCARDIOGRAPHY

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Objective: The CHA2DS2–VASC score is a very useful clinical score in determining the risk of embolic stroke in patients with nonvalvular atrial fibrillation and in the indication of anticoagulant therapy. When we scan the literature, although there are studies on CHADS2 score; there are insufficient data to evaluate the relationship between left atrial appendage (LAA) thrombus and CHA2DS2–VASC score. The aim of our study is to evaluate this relationship.

Method: 100 patients were included in this retrospective study, 50 patients without thrombus in the LAA (group 1) and 50 patients with thrombus in the LAA (group 2) detected by transesophageal echocardiography (TEE). All TEE examinations were made due to non valvular atrial fibrillation. Demographic datas, vital signs, electrocardiography datas, echocardiography datas and laboratory findings were obtained retrospectively from patient files. Collected datas of both groups were compared statistically.

Results: The majority of main demographic and laboratory findings were similar among two groups. From the risk factors; the frequency of hypertension and diabetes were significantly high in group 2. From the echocardiography parameters; the LAA flow velocity is significantly high in group 2. Also; CHA2DS2–VASC score is found to be significantly higher in thrombus group (Table 1).

Conclusion: The main finding of our study was that left atrial appendage thrombus is more common in patients with high CHA2DS2–VASC score. In the light of our study, it is necessary to be very careful when evaluating patients with high CHA2DS2–VASC score in terms of LAA thrombus with TEE. However, prospective multicenter studies with a large number of patients and advanced statistical analyzes are needed in order to reach more precise results in this regard.

Keywords: CHA2DS2–VASC score, transesophageal echocardiography, left atrial appendage thrombus

Table 1. Demographic, laboratory clinical and echocardiography findings of the study population

Variable	Without thrombus (Group 1) n:50	With thrombus (Group 2) n:50	p
Age (years)	52 ± 14.7	54 ± 15.9	0.121
Gender (female), n (%)	23 (46)	25 (50)	0.497
Body mass index, kg/m ²	26.9 ± 3.0	26.7 ± 1.8	0.741
Hypertension, n (%)	13 (26)	20 (40)	0.004
Diabetes mellitus, n (%)	11(22)	17 (34)	0.003
Hyperlipidemia, n (%)	8(16)	9 (18)	0.654
Smoking, n (%)	15 (30)	16 (32)	0.705
Left ventricle ejection fraction, %	57.2 ± 5.83	52.1 ± 6.41	0.071
Left atrium diameter, mm	37.3 ± 4.72	43.5 ± 4.96	0.023
Plasma glucose, mg/dl	133 ± 43.8	141 ± 45.7	0.068
Creatinin, mg/dl	0.81 ± 0.63	0.84 ± 0.73	0.446
Hemoglobin, g/dl	13.5 ± 1.8	13.3 ± 1.9	0.536
White blood cell, 103/mm ³	9.3 ± 1.8	9.5 ± 1.8	0.656
Platelet, 103/mm ³	278.4 ± 51.5	295.1 ± 48.4	0.159
Heart rate, pulse/minute	93.5 ± 27.8	99.4 ± 30.4	0.086
Systolic blood pressure, mmHg	129.7 ± 26.7	134.8 ± 30.1	0.233
Diastolic blood pressure, mmHg	87.4 ± 12.6	91.2 ± 15.3	0.285
Left atrial appendage flow velocity, cm/s	27.4 ± 3.9	17.6 ± 3,4	<0.001
CHA2DS2–VASC score	1.2 ± 1.1	1.9 ± 1.4	<0.001

Demographic, laboratory clinical and echocardiography findings of the study population

OP-067 ASSESMENT OF AORTIC BIOPROTHESIS VALVE THROMBOSIS: TWO AND THREE DIMENSIONAL TRANSESOPHAGEAL ECHOCARDIOGRAPHY STUDY

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Case Report

A 66 years old male patient with a history of transcatheter aortic valve replacement (TAVR) 8 months earlier, type 2 diabetes mellitus and hypertension, had admitted the emergency department with New York Heart Association Class III symptoms. On admission, cardiac auscultation revealed pathologic 4/6 systolic murmurs, and electrocardiography demonstrates sinus tachycardia. Bedside transthoracic echocardiography (TTE) indicated increased bioprosthetic aortic valve gradient (Max gr: 74 mm-Hg Mean Gr 52 mm hg), the normal ejection fraction of left ventricle, moderate mitral regurgitation, and mild tricuspid regurgitation. Further investigation with two-dimensional (2D) and real-time three-dimensional (RT-3D) transesophageal echocardiography (TEE) revealed thrombus like image and obstruction, on mid-esophageal long-axis view at the bioprosthetic aortic valve (Fig 1,2 and Movie 1,2). However, bioprosthetic valve thrombosis (BVT) thought to be uncommon beyond three months of implantation.(1) Thus, the pathology had to be brightened. Subsequently, on the deep trans-gastric long-axis view (TG LAX), the aortic valve was assessed distinctly. We achieved precious information with TG LAX 2D (Fig 3 and Movie 3) and RT-3D (Fig 4,5,6 and Movie 4,5) images and ensured that the pathology was the obstructive thrombus. After the diagnosis the patient was treated with systemic anticoagulation therapy with vitamin K antagonist with a target international normalized ratio (INR) of between 2 and 3, Follow-up TEE performed after 10 weeks of anticoagulation revealed significant improvement in AV gradients, and the thrombus formation resolved.

Valvular heart disease affects more than 100 million patients worldwide. Since the last decade, there has been a shift from mechanical to bioprosthetic valve replacement(2). This could be explained by the mechanical heart valve's restricted indications. Prosthetic valve thrombosis, long term warfarin usage, and its complications are important ones. Despite less thrombogenic, bioprosthetic valve thrombosis (BVT) can also lead to bioprosthetic valve dysfunction(3).

BVT incidence may vary depending on the valve type(4). It might be underestimated because valve imaging is not performed post-valve implantation and the limitations of current imaging techniques. TEE and computed tomography (CT) are both good for BVT because the image quality is generally good with fewer artifacts in comparison with the mechanical heart valve(5).

However, except tertiary referral hospitals, cardiac CT experts cannot be reached easily. Hence, in daily practical trans-gastric TEE plans should be used more frequently to provide precious data about BVT. The case presented above emphasizes the importance of 2D and RT-3D trans-gastric TEE plans to test bioprosthetic aortic valve thrombosis.

Keywords: transesophageal, echocardiography, bioprosthetic, valve, thrombosis

fig a



fig b



OP-069 MEAN PLATELET VOLUME TO LYMPHOCYTE RATIO IN THE PREDICTION OF SYNTAX SCORE IN PATIENTS WITH NON-ST-ELEVATION MYOCARDIAL INFARCTION

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Objective: Mean platelet volume to lymphocyte ratio (MPVLR) is associated with major adverse cardiovascular events in patients with non-ST elevation myocardial infarction (NSTEMI). We aimed to compare MPVLR between NSTEMI patients and matched-controls and to evaluate its predictive value on SYNTAX score.

Method: Total 236 patients with NSTEMI and 114 age and co-morbidity matched subjects were included in our study. MPVLR was compared between the patient and control groups. The patient group was further divided into 3 tertiles according to SYNTAX scores as follows: low SYNTAX score tertile (score ≤ 22 , 150 patients); intermediate SYNTAX score tertile (score between 23 and 32, 48 patients); and, high SYNTAX score tertile (score ≥ 33 , 38 patients). MPVLR was further assessed among the tertiles.

Results: MPVLR was significantly greater in the patient group compared to the control group (4.20 ± 2.25 and 3.10 ± 1.42 , respectively, $p < 0.001$). MPVLR among low, intermediate and high score tertiles were calculated to be 3.23 ± 1.32 , 4.17 ± 1.58 and 7.15 ± 2.70 , respectively ($p < 0.001$). A strong positive correlation was found between MPVLR and SYNTAX score ($r = 0.591$; $p < 0.001$). In receiver operating characteristics (ROC) analysis, MPVLR ≤ 5.08 predicted a SYNTAX score ≤ 22 with 97% sensitivity and 57.7% specificity (AUC: 0.790; 95% CI: 0.701 - 0.879; Youden index: 0.54; $p < 0.0001$) and a MPVLR ≥ 5.84 predicted a SYNTAX score ≥ 33 with 81.8% sensitivity and 95.8% specificity (AUC: 0.887; 95% CI: 0.784 - 0.991; Youden index: 0.77; $p < 0.0001$).

Conclusion: MPVLR was significantly higher in NSTEMI patients, compared to controls. Higher MPVLR was associated with greater SYNTAX score in patients with NSTEMI. MPVLR may be used to predict severity of the CAD in patients with NSTEMI.

Keywords: Mean platelet volume to lymphocyte ratio, Non-ST elevation myocardial infarction, SYNTAX score

Figure 1

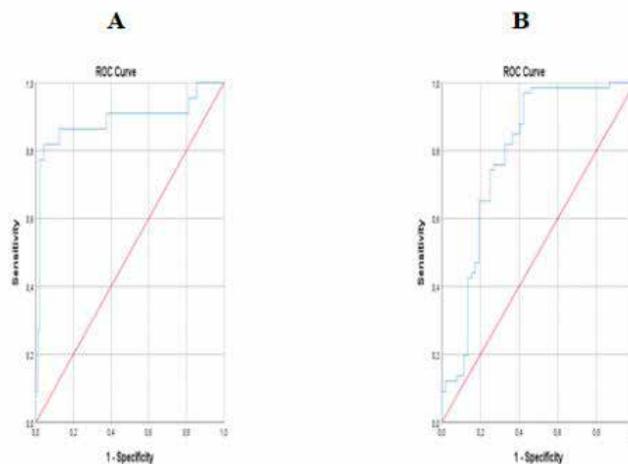


Figure 1: A) ROC curve of MPVLR variable for predicting SYNTAX score ≥ 33 . B) ROC curve of MPVLR variable for predicting SYNTAX score ≤ 22 .

Figure 1: A) ROC curve of MPVLR variable for predicting SYNTAX score ≥ 33 . B) ROC curve of MPVLR variable for predicting SYNTAX score ≤ 22 .

Table 2: ANOVA analysis of MPVLR in SYNTAX score tertiles

Variable	SYNTAX ≤ 22 (n = 150)	SYNTAX 23-32 (n = 48)	SYNTAX ≥ 33 (n = 38)	P-value
MPVLR	3.23 ± 1.32	4.17 ± 1.58	7.15 ± 2.70	< 0.001

Table 2: ANOVA analysis of MPVLR in SYNTAX score tertiles

OP-070 RELATIONSHIP BETWEEN LYMPHOCYTE TO C REACTIVE PROTEIN RATIO AND THROMBUS BURDEN IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Increased coronary thrombus burden is known to be a strong predictor of adverse cardiovascular (CV) outcomes. Lymphocyte to C-reactive protein ratio (LCR) can be used as a surrogate marker of pro-inflammation which is closely related to prothrombotic state. We aimed to evaluate the association between LCR and coronary thrombus burden in patients who presented with acute coronary syndrome (ACS). Patients who presented with ACS and treated with percutaneous coronary intervention (PCI) were included in the study.

Method: 205 ACS (NSTEMI-ACS 48.7% and STEMI 51.3%) patients who underwent PCI between June 2016 and June 2020 were included in the study. Angiographic coronary thrombus burden determined according to the 'thrombolysis in myocardial infarction (TIMI) classification. The patients were divided into two groups as low thrombus burden (LTB, TIMI grade 0-1-2) and high thrombus burden (HTB, TIMI grade 3-4-5).(Table-1)

Results: The lymphocyte / CRP ratio was significantly lower in those with HTB than those with LTB (171.8 ± 66.1, 75.4 ± 35.7, p <0.001). In the multivariate logistic regression analysis, a risk factor independent of thrombus load pain was found with a high lymphocyte / CRP ratio (odds ratio: 1.389, 95% CI: 1.017-1.399; p <0.05). In ROC analysis, a cut-off value of 88.5 for LCR had an 76.7% sensitivity and 76% specificity for predicting high coronary thrombus burden in ACS patients with PCI [(AUC): 81.4 p < 0.001](Table-2, Figure-1).

Conclusion: As a result, the decreased lymphocyte / CRP ratio is an independent parameter that predicts high coronary thrombus burden in ACS patients who were treated with PCI.

Keywords: Acute coronary syndrome, Lymphocyte to C-reactive protein ratio, Thrombus burden

Figure-1

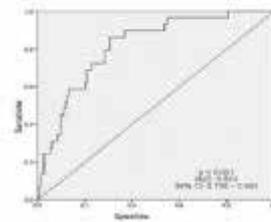
Table 1. Demographic and laboratory findings

Variable	LTB (n=81)	HTB (n=124)	p value
Age	38.8 ± 12.3	46.8 ± 12.8	0.026
Gender, Male, n (%)	51	58	<0.001
BTK, n	45	48	0.411
DM, n	21	28	0.083
Hyperlipidemia, n	14	15	0.549
Smoking, n	42	48	0.413
WBC, 10 ⁹ /L	8.3 ± 3.2	8.9 ± 3.6	0.152
Serum total cholesterol, 10 ³ mg/dL	4.2 ± 2.4	4.8 ± 2.8	0.124
Low-density lipoprotein cholesterol, 10 ³ mg/dL	1.8 ± 0.9	2.2 ± 1.0	0.400
High-density lipoprotein cholesterol, 10 ³ mg/dL	0.8 ± 0.3	0.8 ± 0.3	0.758
Fasting glucose, 10 ³ mg/dL	243 ± 83	233 ± 71	0.517
Hemoglobin, g/dL	12.8 ± 1.7	12.7 ± 1.5	0.254
Serum urea, mg/dL	69.0 ± 9.3	66.2 ± 12.2	0.266
Aspartate aminotransferase, IU/L	28.8 ± 9.6	24.7 ± 8.9	0.335
Alanine aminotransferase, IU/L	28.3 ± 9.4	22.2 ± 15.1	0.278
Creatinin, mg/dL	0.81 ± 0.23	0.89 ± 0.22	0.873
LDL cholesterol, mg/dL	81.8 ± 28.1	75.3 ± 32.7	0.283
CRP, mg/L	4.4 ± 3.3	15.8 ± 2.8	<0.001
Lymphocyte / CRP ratio	171.8 ± 66.1	75.4 ± 35.7	<0.001

DM: Diabetes Mellitus; BTK: Bypass; LDL: Low-density lipoprotein; CRP: C-reactive protein

Table 2. Multivariate logistic regression analysis detecting independent thrombus burden predictors variables

Variable	Odds Ratio (95% CI)	HTB (n=124)	p value
Age	1.022	(0.998-1.047)	0.178
Hypertension	1.069	(0.878-1.315)	0.128
Diabetes Mellitus	1.039	(0.613-1.382)	0.096
Lymphocyte/CRP ratio	1.388	(1.017-1.398)	0.008
LDL, mg/dL	1.008	(0.989-1.018)	0.133
WBC, 10 ⁹ /L	1.017	(0.984-1.050)	0.080
Platelet(10 ⁹ /L)	1.008	(0.987-1.054)	0.008



OP-072 RELATION OF TOTAL CHOLESTEROL TO HIGH DENSITY CHOLESTEROL RATIO ON PREDICTING CORONARY ARTERY ATHEROSCLEROSIS

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Atherosclerotic cardiovascular disease is still the major cause of mortality. Patient's risk factor assessment and demonstration of coronary atherosclerotic plaques is the key step in the primary prevention of cardiovascular events. The purpose of this study was to evaluate the relation of total cholesterol (TC) to high-density lipoprotein cholesterol (HDL-C) ratio on predicting atherosclerotic plaques and coronary artery calcification (CAC).

Methods. We analyzed 667 consecutive patients without any cardiac symptoms and events who underwent computerized tomography (CT) coronary artery calcium scoring. Of these, 148 had a score of 0 according to Agatston score; the remaining 519 patients had calcifications on coronary arteries with low, moderate or high risk. A cutoff cholesterol ratio was prespecified 3.5 which is known to be the ideal maximum value. Multivariate logistic regression were employed to evaluate the association between TC/HDL-C ratio of 3.5 and CAC.

Results. Increased TC/HDL-C was associated with an increased risk of CAC. A TC/HDL-C ratio under 3.5 was significantly correlated with no CAC in asymptomatic patients.

Conclusions. The findings of this study indicated that TC/HDL-C ratio plays an important role in predicting atherosclerotic plaque formation and calcification in asymptomatic patients. This finding needs to be supported by large scale studies for integration of dyslipidemia in new risk scorings for predicting atherosclerosis and beginning primary prevention strategies. Furthermore, new studies can be designed to demonstrate relation of TC/HDL ratio to severity of coronary artery disease.

Keywords: hyperlipidemia, atherosclerosis, prevention, imaging

OP-074 ATHEROGENIC INDEXES VERSUS HEMATOLOGIC INFLAMMATORY INDEXES: WHAT IS MOST USEFUL PREDICTOR OF CORONARY SLOW FLOW?

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Abstract

Aim: Previous studies reported that inflammation and atherosclerosis are linked coronary slow flow (CSF). The predominant pathological mechanism has not been elucidated yet. Hence, we aimed to compare hematologic inflammatory and atherogenic indexes simultaneously between patients with normal coronary flow (NCF) and CSF.

Material and Method: In a single-center retrospective analysis, 91 consecutive NCF patients and 90 consecutive CSF patients constituted two groups according to Thrombolysis in Myocardial Infarction frame count (TFC). Hematological indexes consist of the neutrophil- lymphocyte ratio (NLR), the lymphocyte to monocyte ratio (LMR), and the platelet-lymphocyte ratio (PLR), and the atherogenic indexes consist of an atherogenic index of plasma (AIP), atherogenic coefficient (AC), and Castelli's risk index (CRI). Baseline clinical parameters were compared beside the indexes.

Results: NLR, LMR, PLR were similar in groups. AIP, AC and CRI were significantly higher in the CSF group ($p < 0.05$). In correlation analysis, only CRI has a significantly positive correlation with mean TFC ($r: 0.419$ $p < 0.001$). In multivariate regression analysis, CRI was found as independently predictor of CSF (Odds ratio = 2.74, 95% CI= 1.21-6.207; $p=0.016$). Discussion: An elevated CRI may be an independent predictor for the presence of CSF. Additionally, it can be said that the inflammatory activity in CSF is transformed into atherosclerotic structures.

Keywords: Coronary Circulation; Inflammation; Coronary Atherosclerosis; Dyslipidemias

Introduction

Coronary slow flow (CSF), classified as heart syndrome Y or non-obstructive coronary artery disease (INOCA), is an angiographic phenomenon characterized by late dye transfer to the distal coronary vascular bed during angiography in patients without obstructive coronary lesions [1,2]. CSF is a considerable clinical entity because it causes acute cardiac ischemic effects as well as complaints reminiscent of myocardial ischemia in general [3]. CSF has well-established mechanisms based on inflammation, atherosclerosis, endothelial dysfunction, and microvascular resistance [4]. However, the proportion of this relationship between the factors involved in the pathological mechanism is still unclear.

Hematological inflammatory indexes consisting of neutrophil-to-lymphocyte ratio (NLR), lymphocyte-monocyte ratio (LMR), and platelet-lymphocyte ratio (PLR), associated with inflammation in cardiovascular diseases have been previously specified [5,6]. Studies have shown that there is a significant association between decreased density lipoprotein cholesterol (HDL-c) level, increased low-density lipoprotein cholesterol (LDL-c) level, and the incidence of cardiovascular diseases and adverse events [7,8]. Recently, researchers have defined atherogenic indexes, consisting of the Atherogenic plasma index (AIP), the Atherogenic coefficient (AC), and Castelli's risk index (CRI) to better define cardiovascular diseases based on atherosclerosis [9, 21-23].

Although many studies have been conducted on patients with CSF, as far as we know, no studies have been conducted comparing these hematologic inflammatories and atherogenic indexes in slow coronary flow. We aimed to compare the possible roles of atherogenic and hematological indexes in CSF, which includes both inflammation and atherosclerosis in its basic pathology.

Material and Methods

Patient selection

The present study was a retrospective and single-center study. Patients who admitted to cardiology clinic for stable angina pectoris between January 2015 and December 2018, who underwent coronary angiography and had angiographically normal or close to normal coronary arteries were retrospectively analyzed [4]. At the beginning of our study, 129 patients with coronary slow flow (CSF) were detected. Exclusion criteria are as follows: left ventricular ejection fraction $< 50\%$, history of the previous revascularization, acute coronary syndrome, obstructive coronary lesion ($> 20\%$), myocardial bridging, coronary ectasia, coronary spasm, infection, current anti-inflammatory and lipid-lowering medication usage, autoimmune disease, renal failure, anemia, hematological disease, and malignancy. After exclusion criteria, 181 patients were included in the final analysis, 90 consecutive patients with slow flow in either of their coronary arteries, versus 91 consecutive patients with normal coronary flow (NCF). Hypertension was defined as diastolic blood pressure ≥ 90 mm Hg or systolic blood pressure ≥ 140 mm Hg or with the use of any reported antihypertensive therapy. Diabetes mellitus (DM) was defined as the use of any antidiabetic agent. The study was in compliance with the principles in the Declaration of Helsinki and approved by the Local Institutional Ethics Committee (Ethics committee approval code numbered 2020-16/07 was obtained on 17.06.2020).

Angiographic Data and Determination of Slow Coronary Flow

Coronary angiography of all patients included in the study was performed using the Judkins technique and 6-French catheters from the femoral or radial artery. All angiographic images were recorded in Philips Allura Xper Percutaneous Coronary Intervention digital system at our cardiology clinic. Iopromide contrast (Omnipaque; GE Healthcare) was used in all study patients. Coronary flow of Angiograms was measured according to Thrombolysis in Myocardial Infarction (TFC) described by two cardiologists who were previously blinded to details of the study [10]. The first frame count was determined as the anterograde movement of the contrast material touching both borders of the coronary lumen [10]. The final frame count was determined as the first branch of posterolateral for right coronary artery (RCA), distal bifurcation for left anterior descending (LAD), and circumflex (CX) [10]. The obtained frame results were doubled since the angiographic filming speed was 15 frames/second. Since the LAD artery is naturally long as described previously, the corrected TFC was obtained by dividing it by 1.7 [10]. The mean TFC (mTFC) was calculated by summing the TFCs for the corrected LAD, CX, and RCA, and then dividing the sum by three [10]. Patients with TFC greater than two standard deviations (SD) from the published normal range for any of the three vessels were considered patients with CSF (36.2 ± 2.6 frames for LAD, 22.2 ± 4.1 frames for CX, and

20.4 ± 3 frames for the RCA) [10].

Laboratory Measurements and Definition of Indexes

Blood samples were taken in the morning after 8-12 hours of overnight fast then examined within one hour of arrival at the central laboratory. Complete blood cell counts in blood samples were analyzed using a Beckman Coulter automated hematology device (Beckman Coulter Brea, CA, USA). Serum levels of total cholesterol, triglyceride (TG), and HDL-c were measured with standard enzymatic methods (Abbot GmbH Co, Germany) with using a fully automated analyzer (Abbott Architect c16000) with original reagents. LDL-c concentrations were by the Friedewald method [11]. Body mass index (BMI) was calculated by dividing weight by the square of the height (kg / m²). The estimated glomerular filtration rate (eGFR) was calculated by the Cockcroft Gault Calculator. Smoking was defined as one pack per day.

Hematological inflammatory indexes were calculated as follows [5,6]

Neutrophil lymphocyte ratio (NLR): Neutrophil counts / Lymphocyte counts

Lymphocyte monocyte ratio (LMR): Lymphocyte counts / Monocyte counts

Platelet lymphocyte ratio (PLR): Platelet counts / Lymphocyte counts

The Atherogenic indexes were calculated as follows [9,12]

Atherogenic Index of Plasma (AIP) = log (TG / HDL-c)

Atherogenic Coefficient (AC) = (Total cholesterol – HDL-c) / HDL-c

Castelli's Risk Index (CRI) = LDL-c / HDL-c

Statistical Analysis

All analyses were performed using SPSS for Windows version 25.0 (IBM Corp., Armonk, NY, USA). The compliance of the data to normal distribution was evaluated using the Kolmogorov-Smirnov test. Normally distributed continuous data were expressed as mean ± standard deviation, while continuous variables that were not normally distributed were specified as median with interquartile range (25–75th percentiles). Categorical variables were specified as the number with a percentage. Student's t-tests were used to compare parametric continuous variables, and the Mann-Whitney U test was used to compare nonparametric continuous variables. Chi-square analysis was used to compare categorical variables. The correlation between CRI and mTFC was tested with the Spearman rho analysis. Logistic regression analysis was performed to assess the association of clinical and laboratory parameters with the presence of CSF. Univariate analyses were performed by including parameters that were significantly different between the groups. The Hosmer-Lemeshow test was used to determine sufficient goodness of fit for the regression model. Then, all significant parameters in univariate analysis were evaluated individually in a multivariate model with possible confounding factors (mTFC, age, gender, hypertension, smoking). All odds ratios were presented with their respective 95% confidence intervals (CI). A two-sided p<0.05 was considered significant.

Results

The comparison of the main clinical characteristics and laboratory findings of the groups are shown in Table 1. There was no significant difference between the groups with respect to BMI, family history of premature coronary artery disease (CAD), DM, systolic blood pressure. Age (55.76 ± 12.16 years; 52.03 ± 11.94 years, p = 0.047), female gender (n=27 [30.3%]; n=61 [67%], p <0.001), smoking (n=26 [28.9%]; n=14 [15.4%], p =0.022), hypertension (n=41 [45.6%]; n=27 [29.7 %], p =0.020) were significantly higher in patients with CSF. When the CSF and NCF groups were compared with the laboratory parameters, there was no significant difference between the CRP and glomerular filtration rate. In complete blood count, hemoglobin was significantly higher in patients with CSF (14.66 ± 1.8 g/dL, 13.91 ± 1.7 g/dL, p =0.003). On the other hand, platelet count was significantly higher in the NCF group (244.69 ± 73.52 x10³/mm³, 265.66 ± 63.57 x10³/mm³, p =0.008). Neutrophil count, monocyte count and lymphocyte count were similar among groups. On a lipid scale, the TG levels were similar in the groups. Total cholesterol (134 [100.75-216] mg/dL, 123.5 [93-175.75] mg/dL, p <0.001) and LDL-c (116.98 ± 41.71 mg/dL, 90.42 ± 29.81 mg/dL, p <0.001) were higher in patients with CSF. HDL-c (40 [34-48] mg/dL, 50.5 [40.25-62] mg/dL, p <0.001) was lower in CSF group. As expected, the mTFC (31 [25-37], 14 [12-16], p <0.001) was significantly higher in the CSF group.

Comparisons of the hematological inflammatory indexes and atherogenic indexes are shown in Table 1. NLR, LMR, and PLR were similar between the groups. AIP (3.4 [2.1-5.4], 2.2 [1.8-3.8], p =0.027), AC (3.59 ± 1.66, 2.34 ± 1.19), p <0.001), and CRI (2.92 [1.94-3.84], 1.72 [1.17-2.53], p <0.001) were significantly higher in the CSF group. Univariate and multivariate logistic regression analyses were used to evaluate independent predictors of CSF and are shown in Table 2. In multivariate regression analysis, total cholesterol (OR = 1.946; 95% CI = 1.359 - 2.787, p =0.024), LDL-c (OR = 1.032, 95% CI = 1.005-1.059, p =0.018), CRI (OR = 2.74, 95% CI = 1.21-6.207, p =0.016) were significant independent predictors of the presence of CSF. Also, there was a moderate positive correlation between mFC and CRI (r = 0.419, p <0.001) (Figure 1).

Discussion

In the present study, we found that CRI, LDL-c, and total cholesterol were independent predictors of the presence of CSF. We also found that CRI activity was moderately correlated with mTFC.

CSF is a term professionally known to interventional cardiologists that delays opacification in distal segments without >40% angiographic lesions in main epicardial coronary arteries [1]. Previous studies have shown that CSF is a condition involving systemic inflammatory factors rather than just locally acting inflammation [13]. Systemic inflammatory effects can alter the parameters and rates of blood count. PLR, LMR, and NLR are inexpensive systemic inflammatory markers. Considering the relationship between many cardiovascular diseases in which cardiovascular risk factors are well documented by various previous studies, these parameters can be used as markers for conditions involving an inflammatory process [5,6]. PLR, NLR, and LMR were similar between groups in our study. Inflammatory infrastructure pattern was recessive in CSF. These results may be explained by the presence of near-normal coronary arteries in our patient selection, because inflammation has been blamed in the initial stage rather than the advanced stage of the atherosclerotic process [15].

The benefits of increasing HDL-c level as well as lowering LDL-c level for the prevention of atherosclerotic cardiovascular disease are well known and potential benefits have been reported in patients at an early age [16]. Besides, it has been reported that statin therapy reduces CSF and increases coronary flow reserve in patients with CSF [17]. Decreased coronary reserve in CSF required a new definition as INOCA [2]. The increased microvascular tone has been blamed in the CSF examined in the INOCA subgroup. In the increase of coronary microvascular tone, oxidative stress and pro-inflammatory process in which adipokines are blamed together with sympathetic hyperactivity [18]. Recent adipokine gene studies have found significant regulatory and correlative effects of adipokines on LDL-c and total cholesterol in obese male and female patients [19]. According to the results of our study, the increased LDL-c and total cholesterol level associated with atherosclerosis and increased microvascular tone is an independent predictor of CSF.

CSF may include diffuse atherosclerosis not only in the epicardial coronary arteries but also in the coronary microvascular circulation [20]. The relationship of atherogenic indexes with cardiovascular diseases and cardiovascular death based on atherosclerosis has been defined [9,12]. A recent study found that the SYnergy between PCI with TAXUS and Cardiac Surgery (SYNTAX) score, which indicates the severity of obstructive coronary artery disease, is associated with AIP [21]. Besides, saphenous vein graft stenosis associated with distal flow insufficiency, endothelial damage, and accelerated atherosclerosis has been associated with AIP and AC [22]. Furthermore, in a study based on intracoronary imaging in CAD, there was a significant relationship between plaque lipid content and plaque sensitivity and CRI [23].

In our comparison of the CSF group with the NCF group, AIP, AC, and CRI were found to be significantly higher in the CSF group. CRI, the equivalent of LDL-c/HDL-c, was an independent predictor of CSF.

The present study has some limitations that need to be mentioned. First, this study used a retrospective study design with a relatively small sample size based on single-center experience. Second, since we used a single CRI value and a limited number of patients for our analysis, rather than a transient trend, no prognostic values were determined on cardiovascular outcomes. Third, Cohen's kappa value was not determined to detect inter-observer and intra-observer variations in TFC measurements. Finally, we did not evaluate for endothelial dysfunction and microvascular tone.

Our findings suggested that higher total cholesterol level, LDL-c level, and CRI were independently related to the presence of CSF. In addition to that, CRI was significantly positively correlated with mTFC. The findings of our study also confirmed that CRI had a moderate positive correlation with mTFC. Studying these markers may provide more informative data about the pathogenesis of microvascular atherosclerosis in patients with CSF. Additionally, it can be said that the inflammatory activity in CSF changes into atherosclerotic structures but more studies are needed to confirm our findings in CSF.

Support and conflicts of interest

The authors report no involvement in the research by the sponsor that could have influenced the outcome of this work. The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Scientific Responsibility Statement: The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement: All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Figure Legend:

Figure 1. Correlation between mFC and CRI (r = 0.419, p <0.001)

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Table 1. Comparison of baseline clinical, laboratory parameters and indexes of study groups .

	Patient with CSF (n=90)	Patient with NCF (n=91)	p
Clinical parameters			
Age, years	55,76±12,16	52,03±11,94	0.047
Female gender	27(30.3 %)	61(67 %)	< .001
BMI, kg/m ²	27.1(25-30.1)	26.6(2.7-31,2)	0.671
Smoking	26(28.9 %)	14(15.4 %)	0.022
Family history premature CAD	20(22.2 %)	14(15.4 %)	0.162
Diabetes mellitus	10(11.1 %)	14(15.4 %)	0.265
Hypertension	41(45.6 %)	27(29.7 %)	0.020
Systolic blood pressure, mmHg	127.5(120-140)	125(110-130)	0.140
Laboratory parameters			
eGFR, mL/min	90(83.8-90)	90(85.8-90)	0.247
CRP, mg/L	3,4(2,25-6,7)	3,2(1,93-5,35)	0.176
Hemoglobin, g/dL	14,66±1.8	13.91±1.7	0.003
Platelet, X 10 ³ /mm ³	244.69±73.52	265.66±63.57	0.008
Neutrophil, X 10 ³ µL	4.7(3.61-5.72)	4.52(3.58-6.09)	0.627
Monocyte, X 10 ³ µL	0.48(0.36-0.64)	0.48(0.38-0.62)	0.989

Lymphocyte, X 10 ³ µL	2.15(1.68-2.56)	2.32(1.93-2.68)	0.100
Triglyceride mg/dL	134(100.75-216)	123.5(93-175.75)	0.191
Total cholesterol, mg/dL	187.11±52.65	154.64±38.36	<.001
LDL-c, mg/dL	116.98±41.71	90.42±29.81	<.001
HDL-c, mg/dL	40(34-48)	50.5(40.25-62)	<.001
mTFC	31(25-37)	14(12-16)	<.001
Hematological inflammatory indexes			
NLR	2.17(1.60-3.01)	2.10(1.59-2.81)	0.342
LMR	4.37(3.10-5.66)	4.40(3.33-5.95)	0.261
PLR	120.6(93.0-151.2)	118.8(90.2-140.0)	0.066
Atherogenic indexes			
AIP	3.4(2.1-5.4)	2.2(1.8-3.8)	0.027
AC	3.59±1.66	2.34±1.19	<.001
CRI	2.92(1.94-3.84)	1.72(1.17-2.53)	<.001

Abbreviations: AC, atherogenic coefficient; AIP, atherogenic index of plasma; BMI, body mass index; CAD, coronary artery disease; CRI, Castelli's risk index; CRP, C-reaktif protein; CSF, coronary slow flow; eGFR, estimated glomerular filtration rate; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; LMR, lymphocyte to monocyte ratio; mTFC, mean TIMI frame count; NCF, normal coronary flow; NLR, neutrophil to lymphocyte ratio; PLR, platelet to lymphocyte ratio; TIMI, thrombolysis in myocardial infarction.

Table 2. Univariate and multivariate logistic regression analyses of independent variables of SCF

Variables	Univariate Logistic Regression Analysis			Multivariate Logistic Regression Analysis*		
	Odds Ratio	95% CI	p value	Odds Ratio	95% CI	p value
Hemoglobin	1.302	1.091-1.553	0.003			
Platelet	0.995	0.991-1.000	0.035			
Total cholesterol	1.012	1.004-1.020	0.002	1.946	1.359-2.787	0.024
LDL-C	1.017	1.008-1.026	<0.001	1.032	1.005-1.059	0.018
HDL-C	0.984	0.964-1.003	0.103			
AIP	1.101	0.97-1.251	0.138			
AC	1.662	1.269-2.177	<0.001			
CRI	1.94	1.44-2.61	<0.001	2.74	1.21-6.207	0.016

Abbreviations: AC, atherogenic coefficient; AIP, atherogenic index of plasma; CRI, Castelli's risk index; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol. * Multivariate logistic regression analyzes were performed for each parameter adjusted for mTFC, age, sex, hypertension, and smoking coefficients, and only significant results were presented.

OP-075 EXPERIMENTAL INVESTIGATION OF EFFECT OF RADIOPAQUE SUBSTANCES ON FIBRINOGEN RESULTS

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Background: Radiopaques are pharmacological agents that enable diagnostics to visualize organs. It has been reported that the use of radiopaque affects some biochemical tests. Since the presence of these molecules in the blood sample interfere with some measurement methods, false low or high analysis results may be encountered. Accurate measurement of fibrinogen levels in thromboembolic events is important for consumption coagulopathy. The effect of radiopaque on fibrinogen concentrations has not previously been demonstrated. The aim of this study is to experimentally demonstrate the effect of commonly used radiopaques on fibrinogen analysis.

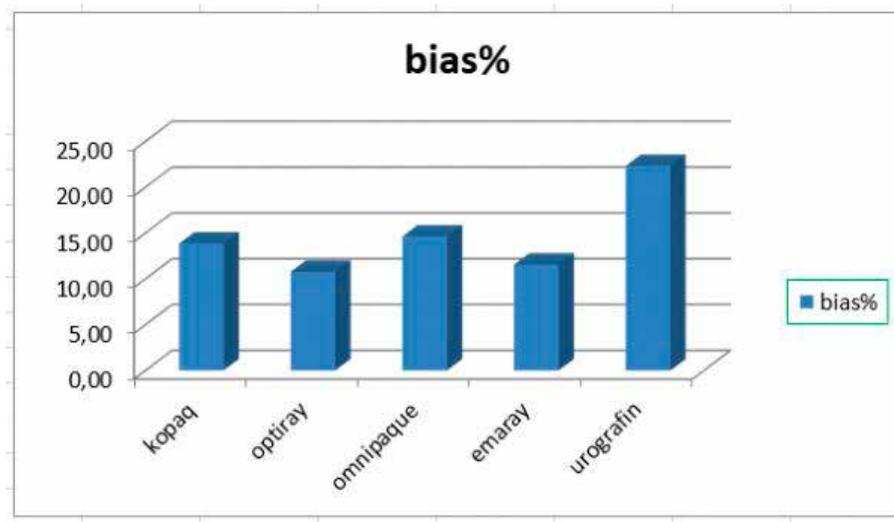
Method: Control solution containing fibrinogen (Siemens, Germany, lot: 507792) was used as a sample. Kopaq 300 mg / ml (iohexol1), optiray 300 mg / 100ml (ioversol), omnipaque 350mg / 50 ml (iohexol2), emaray 469mg / ml IV (gadopentetic acid dimeglumine), urografine 50ml (meglumine diatrizoate) were used as radiopaque. The mixture was prepared by taking 20µL of each drug solution separately and 180µL of the biochemical control solution with the same content as the blood. Fibrinogen test was performed from the prepared mixture in coagulation autoanalyzer (sysmex cs2100). The same run was repeated by adding 20µL of distilled water to the control solution. Deviation amounts were calculated with bias %.

Results: The detected deviation range was calculated between 10.77% and 22.31%. The greatest deviation was caused by the meglumine diatrizoate with a rate of 22.31%. Deviation from the target value was observed at a rate of 13.85% due to iohexol1, 10.77% due to ioversol, 14.62% due to iohexol2, and 11.54% due to gadopentetic acid dimeglumine. Positive interference was observed as all bias rates occurred in a positive direction (Figure 1).

Conclusion: Drugs that cause false low measurement of blood fibrinogen concentration may cause late diagnosis or miss diagnosis in patients who are consumed. Care should be taken for negative interference in fibrinogen measurements, especially after the use of meglumine diatrizoate.

Keywords: Fibrinogen, iohexol, ioversol, gadopentetic acid, analytical error

Figure 1. Demonstration of the effect of radiocontrast agents on fibrinogen test results



OP-077 OPTIMAL TIMING FOR COMPLETE REVASCLARIZATION IN PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION: INDEX HOSPITALIZATION VERSUS EARLY AFTER DISCHARGE

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Objective: Most of the patients undergoing primary PCI for STEMI have multivessel coronary artery disease. Revascularization of non-culprit lesions is not recommended during primary percutaneous coronary intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI). According to the current guidelines, complete revascularization via staged PCI during index hospitalization is highly recommended. But, many patients are discharged from the hospital due to patient's preference or health insurance problems without complete revascularization. Therefore, the question of optimal intervention timing for non-culprit lesion remains unclear. We aimed to compare the success of complete revascularization at index hospitalization with early after discharge in patients with STEMI.

Methods: A total of 310 STEMI patients treated with primary PCI between June 2020-December 2020 were screened. Among them, 75 patients who underwent intervention for non-culprit lesions were enrolled and retrospectively analyzed. Patients with need for emergency CABG operation, in-hospital mortality, no other coronary stenosis except culprit lesion and severe comorbidities were excluded from the study. Patients were divided into two groups according to the timing of complete revascularization, which were defined as index hospitalization (0-3 days) and early after discharge (1 week to 1 month). There were 43 patients in index hospitalization group, whereas 32 patients in early after discharge group. Primary endpoints were defined as death, MI, hospitalization and contrast-induced nephropathy (CIN).

Results: Demographic, clinical characteristics and laboratory measurements were similar between groups. There were no statistically significant difference between the groups in terms of primary end-points. (Table-1) CIN was numerically higher in index hospitalization group, but did not reach statistical significance. (Table-1)

Conclusion: Our results demonstrated that complete revascularization of non-culprit lesions early after discharge may be as safe as staged PCI during index hospitalization in selected STEMI patients.

Key words: primary PCI, staged PCI, non-culprit lesion

Table-1: Adverse clinical events according to study groups during follow-up

	Index hospitalization group (n=43)	Early after discharge group (n=32)	p value
Death	3 (7%)	0 (0%)	0.13
Myocardial infarction	5 (11.6%)	7 (21.9%)	0.23
Hospitalization	8 (18.6%)	7 (21.9 %)	0.72
Contrast-induced nephropathy	10 (23.3%)	5 (15.6%)	0.41

OP-078 THE EFFECT OF TOTAL OCCLUSION PATTERN IN HOSPITAL AND LONG TERM CLINICAL OUTCOMES IN ACUTE ST SEGMENT ELEVATED MYOKARDIAL INFARCTION

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Aim: The aim of our study is to determine the relationship between the total occlusion pattern and in hospital no-reflow development and long-term mortality in patient admitted to our hospital with STEMI.

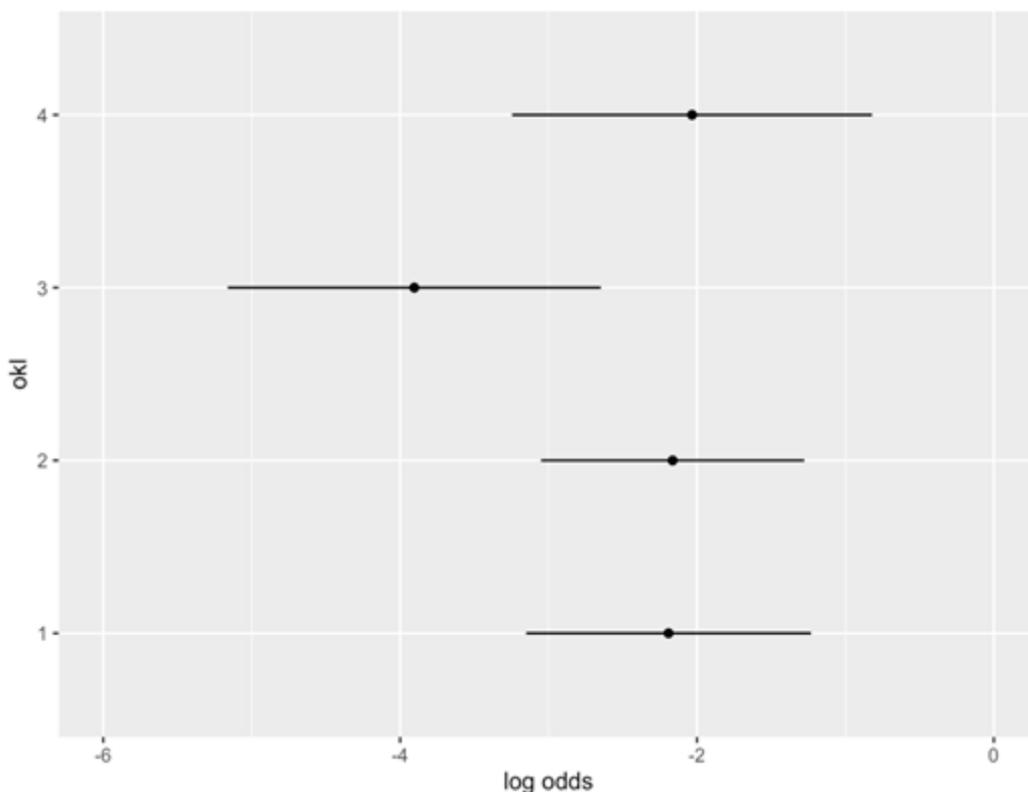
Method: This study was designed as a single center cross-sectional study. Study population was chosen from patients older than 18 years old who admitted to emergency department of Dr Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital between march 2017 and february 2019 with symptoms suggesting myocardial ischemia who diagnosed as STEMI 1541 patients met these criteria and were enrolled to the study. Patients who underwent coronary angiography were divided into 4 classes according to the total occlusion pattern. Primary end points include no-reflow development after PCI and long term death.

Results: In the study, it was found that the no-reflow rate in the hospital was 10% (153 patients), and the mortality rate in the long-term follow up (median 19 months) was 7% (98 patients). In order to evaluate the relationship between no-reflow and subtotal occlusion in logistic regression analysis. (OR;0.175, 95% CI;0,060–0,505, p value;0.0016). In addition, there was a significant relationship between no-reflow and age and total ischemia time. (OR;1.903, 95% CI;1.18-3.046 and OR;1.091, 95% CI;1.029-1.156) To evaluate the relationship between long-term mortality and candidate predictors, there was no significant relationship between total occlusion types and long-term mortality in Cox proportional hazard regression analysis. (HR;0.881, 95% CI;0.368–2.106 p value;0.5711). Among the independent predictors of long-term mortality, a significant relationship was observed between age, duration of ischemia and previous MI. (HR;2.47, 95% CI;1.606-4.699, HR;1.082, 95% CI;1.026-1.140 and HR;2.752 95% CI;1.366-5.545)

Conclusion: We found that in patients presenting with STEMI, the total occlusion pattern was associated with in hospital no-reflow phenomenon, but not with long-term mortality.

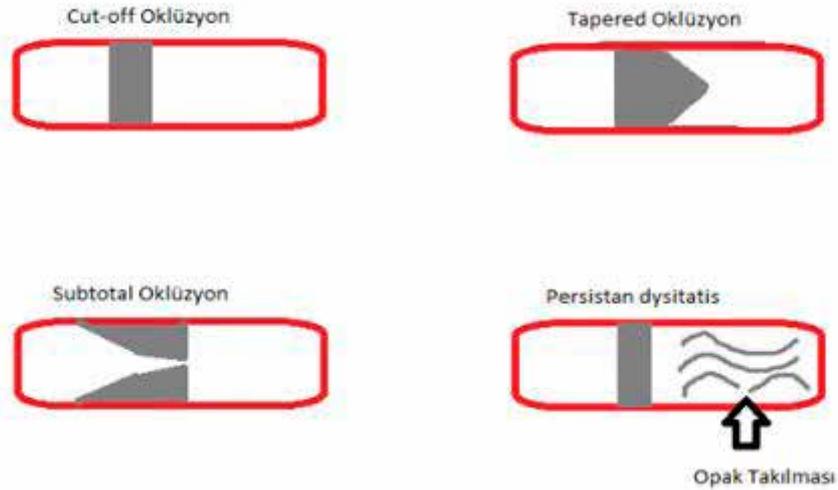
Keywords: Total occlusion pattern, no-reflow, long term mortalite, ST elevation myocardial infarction

Log Odds Graph Between Total Occlusion Types and No-reflow



Log Odds graph showing the relationship between total occlusion types and no-reflow.

Total Occlusion Patterns



Patients who underwent coronary angiography were divided into 4 classes according to the total occlusion pattern.

Independent Predictors Of No-reflow and Logistic Regression Analysis Table

Variable	Odds Ratio (OR)	95% Confidence Interval	P Value
Age	1,903	(1,18 - 3,046)	0,0073
Gender	0,813	(0,37 - 1,769)	0,6033
HT	0,740	(0,379 - 1,444)	0,3779
DM	1,319	(0,679 - 2,563)	0,4132
Smoking	0,785	(0,375 - 1,641)	0,5206
Total ischemia time	1,091	(1,029 - 1,156)	0,0035
MI History	1,656	(0,823 - 3,331)	0,1569
total occlusion type 1:2	0,972	(0,496 - 1,906)	0,9364
total occlusion type 3:2	0,175	(0,060 - 0,505)	0,0016
total occlusion type 4:2	1,139	(0,420 - 3,089)	0,7605

A significant relationship was found between subtotal occlusion and no-reflow. No-reflow was significantly reduced in the subtotal occlusion group.

OP-079 THE PREDICTION OF NO-REFLOW IN YOUNG ST SEGMENT ELEVATION MYOCARDIAL INFARCTION PATIENTS BY SIMPLE ELECTROCARDIOGRAPHIC SCORE

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Objective: No reflow is defined as a condition associated with inadequate myocardial perfusion of the coronary artery without evidence of angiographic epicardial occlusion. The recently developed electrocardiographic risk score, including anatomical pathology (Q waves, left ventricular hypertrophy (LVH)), depolarization abnormality (QRS duration), and repolarization abnormality (JTc prolongation), was associated with sudden arrhythmic death in patients with coronary heart disease (CHD). As we know, there is no electrocardiographic parameter in the literature that predicts coronary no reflow. Therefore, we aimed to examine the relationship between electrocardiographic risk score and no reflow in young patients (≤ 45 years) that admitted to our hospital with ST elevation myocardial infarction (STEMI).

Method: The study included 131 patients who did not have a prior percutaneous coronary intervention (PCI), and/or coronary artery bypass grafting (CABG), who were hospitalized with the diagnosis of STEMI and underwent CAG between January 2016 and March 2021 in cardiology department of Kafkas University. The information of the patients included in the study was obtained from the electronic file system of our hospital. Patients' ECG strips were scanned and transferred to computer. ImageJ digital image processing software (imagej.nih.gov/ij/) was used to analyze ECG measurements. The patients were divided into two groups with and without no reflow. The univariate and multivariate analyses were used to assess the relationship between electrocardiographic risk score and no reflow phenomenon.

Results: In our study, the no reflow phenomenon was occurred in 40 patients (30.5%). The mortality rate was 6.1%, recurrent myocardial infarction rate was 13%, hospitalization with heart failure was 3.4%, in a mean follow-up of 18.7 months in patients who presented with STEMI and underwent percutaneous coronary intervention. Among the electrocardiography parameters included in the score, development of q wave, prolonged JTc, prolonged QRS duration and left ventricle hypertrophy findings were significantly higher in the no reflow group. Also, electrocardiographic risk score was higher in no reflow group compared with reflow group (median: 2 (1.5-3) vs. 1(1-2)) In multivariate analyses, electrocardiographic risk score was associated with no reflow (HR: 1.459; 95% CI: 1.022-2.082; p= 0.038).

Conclusion: Our study demonstrated that the defined ECG score could be an independent predictor of no reflow in STEMI patients. ECG score, may be used in clinical practice to evaluate the risk of no reflow in STEMI patients.

Table 1: demographic, clinical and laboratory characteristics of all patients with and without no-reflow

	All patients (n:131)	No-reflow (-) (n:91)	No-reflow (+) (n:40)	p value
Age; years	41 ±6	41 ±5	40 ±6	0.229
Male gender; n(%)	117 (89.3)	81 (89.0)	36 (90.0)	0.866
Diabetes mellitus; n(%)	8 (6.1)	7 (7.7)	1 (2.5)	0.253
Hypertension; n(%)	12 (9.2)	9 (9.9)	3 (7.5)	0.662
Hyperlipidemia; n(%)	1 (0.8)	1 (1.1)	0 (0.0)	0.509
Family history of CAD; n(%)	24 (18.5)	22 (24.4)	2 (5.0)	0.008
Smoking; n(%)	104 (80.0)	76 (84.4)	28 (70.0)	0.057
CFA on admission; n(%)	2 (1.5)	0 (0.0)	2 (5.0)	0.083
Total ischemic time; hours	2.11 ±0.41	2.05 ±0.40	2.31 ±0.38	0.001
Systolic blood pressure; mmHg	125 ±19	125 ±19	124 ±19	0.692
Heart rate; bpm	81 ±16	81 ±16	82 ±16	0.803
Hemoglobin; g/dl	15.2 ±1.8	15.4 ±1.9	14.8 ±1.4	0.018
White blood cell count; 10 ³ /µl	12.0 ±3.5	11.6 ±3.5	13.1 ±3.2	0.006
Platelet count; 10 ³ /µl	240 (205-291)	253 (207-292)	237 (203-296)	0.254
Glucose; mg/dl	110 (102-137)	110 (102-137)	111 (102-138)	0.803
Creatinine; mg/dl	0.96 (0.76-0.98)	0.83 (0.75-0.96)	0.88 (0.78-0.99)	0.546
Sodium; mmol/L	137 ±4	138 ±4	137 ±4	0.457
Potassium; mmol/L	4.1 ±0.4	4.1 ±0.4	4.1 ±0.4	0.683
Alanine transaminase; mg/ml	33 (21-46)	29 (20-44)	36 (25-51)	0.202
Baseline Creatine Kinase MB (ng/mL)	78 (32-158)	68 (32-115)	158 (69-228)	<0.001
Peak Creatine Kinase MB (ng/mL)	142 (74-279)	103 (53-185)	262 (168-523)	<0.001

Table 2: Angiographic and electrocardiographic findings of all patients and patients with and without coronary no-reflow

		All patients (n:134)	No-reflow (-) (n:91)	No-reflow (+) (n:43)	p value
Localization of myocardial infarction	Anterior, n (%)	65 (48.1)	40 (44.0)	25 (57.5)	0.024
	Inferior, n (%)	62 (47.3)	49 (53.8)	13 (32.5)	
	Others, n (%)	6 (4.6)	2 (2.2)	4 (10.0)	
Number of leads with ST segment elevation		3 (3.5)	3 (3.5)	4 (3.6)	
Number of patients with \geq 70% ST segment resolution, n (%)		67 (51.1)	53 (58.2)	14 (35.0)	0.034
Number of patients with \geq 50% ST segment resolution, n (%)		104 (77.1)	74 (81.3)	27 (67.5)	0.083
Contiguous Q waves, n (%)		57 (43.5)	34 (37.4)	23 (57.5)	0.06
LV hypertrophy		26 (22.2)	3 (3.3)	13 (32.5)	<0.001
Profound Tc		23 (17.6)	10 (11.0)	13 (32.5)	0.003
QRS duration, ms	80-100	54 (41.2)	45 (49.5)	9 (22.5)	0.001
	81-110	59 (41.2)	40 (44.0)	19 (47.5)	
	>110	38 (33.7)	6 (6.6)	12 (30.0)	
Score		1 (3.2)	1 (1.2)	2 (1.5-3)	<0.001
LV ejection fraction (%)		41.8 \pm 9.6	42.8 \pm 9.3	36.5 \pm 9.2	0.076
Infarct related artery, n (%)	LAD	64 (48.9)	41 (45.1)	23 (57.5)	0.401
	CX	25 (19.1)	19 (20.9)	6 (15.0)	
	RCA	38 (29.0)	29 (31.9)	9 (22.5)	
	others	4 (3.1)	2 (2.2)	2 (5.0)	
Pre-procedure TIMI flow	0	75 (55.7)	48 (53.7)	25 (62.5)	0.301
	1	1 (1.5)	0 (0.0)	2 (5)	
	2	24 (18.3)	0 (0.0)	24 (60)	
	3	105 (80.2)	91 (100.0)	14 (35)	
Myocardial blush grade	0	26 (19.8)	0 (0.0)	26 (65.0)	<0.001
	1	14 (10.7)	0 (0.0)	14 (35.0)	
	2	35 (26.7)	35 (38.5)	0 (0.0)	
	3	56 (42.7)	56 (61.5)	0 (0.0)	
High-grade thrombotic burden, n (%)		92 (70.2)	69 (75.9)	32 (80.0)	0.105
Pre-dilatation		37 (28.2)	21 (23.1)	16 (40.0)	0.048
Post-dilatation		16 (21.5)	8 (17.0)	8 (20.0)	0.174
Stent length, mm		24 (18.90)	22 (18.28)	28 (23.38)	
Stent diameter, mm		3 (2.5-3)	3 (2.75-3)	3 (2.5-3)	
Non-IRA PCI		26 (19.8)	20 (22.0)	6 (15.0)	0.376
Thrombus		8 (6.1)	2 (2.2)	6 (15.0)	0.016
Recurrent myocardial infarction		15 (13.0)	10 (12.5)	5 (14.3)	0.794
Heart failure hospitalization		4 (3.4)	1 (1.3)	3 (8.3)	0.053
Death, re-infarction, re-hospitalization		24 (18.3)	12 (13.2)	12 (30.0)	0.022
Follow-up, months		18.7 \pm 14.6	17.9 \pm 14.0	20.7 \pm 15.9	0.440

univariate and multivariate analyzes of no-reflow prediction

		univariate analysis				multivariate analysis		
	p value	HR	95% CI lower	95% CI upper	p value	HR	95%CI lower	95% CI upper
CPA on admission	<0.001	174.664	13.579	2246.724				
Heart Rate	0.005	1.039	1.012	1.068				
Glucose	0.018	1.005	1.001	1.009				
LVEF	0.038	0.949	0.903	0.997				
ECG score	0.007	1.560	1.127	2.159	0.038	1.459	1.022	2.082

OP-080 PRESENCE OF FRAGMENTED QRS IS ASSOCIATED WITH MAJOR ADVERSE CARDIAC EVENTS IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Introduction: The incidence of acute coronary syndrome is increasing and it is one of the most important causes of morbidity and mortality. Due to the high risk of morbidity and mortality in patients with acute coronary syndrome, various scoring systems have been developed to predict major adverse cardiac events (MACE). The presence of fragmented QRS (F-QRS) in 12-lead electrocardiography, as a possible marker of myocardial fibrosis, has been found to be associated with adverse cardiac events in a variety of cardiomyopathies. The aim of this study is to evaluate the prevalence of F-QRS in patients with acute coronary syndrome and to determine the effect of F-QRS on major adverse cardiac events.

Methods: 397 consecutive patients with acute coronary syndrome were included in this study. F-QRS was defined as the presence of various RSR' patterns such as additional R wave (R prime) or notching of the R or S wave in at least two consecutive leads. Patients were compared in two groups according to the presence or absence of F-QRS in the admission electrocardiogram. All patients were followed for one year after ST-segment elevation and non-ST-segment elevation myocardial infarction (STEMI and NSTEMI) and major adverse cardiovascular events (recurrent angina, recurrent myocardial infarction, revascularization, heart failure, and mortality) were documented.

Results: Of the 397 patients included in this study, there were 257 men and 140 women with a mean age of 61.4 ± 11.6 years. There were 195 patients (49.1%) in the group with F-QRS, and 202 patients (50.9%) in the group without F-QRS (Table 1). Parameters including age, gender, smoking, diabetes mellitus, hypertension, hyperlipidemia, left ventricular ejection fraction (LVEF) were not significantly different between the two groups. Disseminated coronary artery disease (≥ three vessels) was more common in the group with F-QRS compared to the group without F-QRS (p<0.001) (Table 1). The incidence of recurrent MI (13.4% vs. 2.2%), recurrent angina (16.4% vs. 3.3%), heart failure (14.9% vs. 1.1%), revascularization (17.9% vs. 5.6%), cardiac mortality (5.9% vs. 1.1%), and MACE (46.2% vs. 3.3%) were higher in STEMI patients with F-QRS compared to the patients without F-QRS (Table 2). There was no statistically significant difference between F-QRS positive and negative groups in terms of adverse cardiac events in NSTEMI patients.

Conclusion: F-QRS was associated with major adverse cardiac events especially in patients with STEMI. Consequently, as a simple and convenient clinical parameter, F-QRS, may be used as a predictor of adverse cardiac events after STEMI.

Keywords: Acute Coronary Syndrome, Fragmented QRS, Major Adverse Cardiac Events

Adverse cardiac event rates at follow-up among patients by type of myocardial infarction and presence of F-QRS

Table 2 Adverse cardiac event rates at follow-up among patients by type of myocardial infarction and presence of F-QRS

Variables	STEMI F-QRS(+)	STEMI F-QRS(-)	p-value	NSTEMI F-QRS(+)	NSTEMI F-QRS(-)	p-value
Recurrent MI (n,%)	9(13.4)	2(2.2)	<0.001*	3(2.3)	1(0.8)	0.12
Recurrent angina (n,%)	11(16.4)	3(3.3)	<0.001*	3(2.3)	1(0.8)	0.32
Heart failure (n,%)	10(14.9)	1(1.1)	<0.001*	2(1.5)	1(0.8)	0.19
Revascularization (n,%)	12(17.9)	5(5.6)	<0.001*	2(1.5)	1(0.8)	0.34
Mortality (n,%)	4(5.9)	1(1.1)	<0.001*	1(0.7)	1(0.8)	0.32
MACE (n,%)	31(46.2)	3(3.3)	<0.001*	2(1.5)	1(0.8)	0.23

*P<0.05

Abbreviations: F-QRS fragmented QRS complex, STEMI ST-segment elevation myocardial infarction, NSTEMI non-ST-segment elevation myocardial infarction, MI myocardial infarction, MACE major adverse cardiac events

*P<=0.05 Abbreviations: F-QRS fragmented QRS complex, STEMI ST-segment elevation myocardial infarction, NSTEMI non-ST-segment elevation myocardial infarction, MI myocardial infarction, MACE major adverse cardiac events

Clinical characteristics of patients with and without F-QRS

Variables	F-QRS(+) (n = 195)	F-QRS(-) (n = 202)	p-value
Age (years)	60.3 ± 10.5	62.5 ± 12.7	0.25
Male (n,%)	123(63)	134(66.3)	0.17
Smoking (n,%)	104(53.3)	119(58.9)	0.32
Hypertension (n,%)	87(44.6)	85(42)	0.39
DM (n,%)	98(50.2)	102(50.4)	0.26
Hyperlipidemia (n,%)	57(29.2)	49(24.2)	0.18
LVEF (%)	38.3 ± 10.8	39.4 ± 9.4	0.28
F QRS positive	3.1 ± 1.4	-	
STEMI (n,%)	67(34.3)	89(44)	<0.001*
NSTEMI (n,%)	128(65.6)	113(55.9)	<0.001*
CAD ≥ three vessels (n,%)	64(32.8)	14(6.9)	<0.001*

*P<=0.05 Note: Data are presented as number and percentage or mean ± standard deviation. Abbreviations: F-QRS fragmented QRS complex, DM diabetes mellitus, LVEF left ventricular ejection fraction, STEMI ST-segment elevation myocardial infarction, NSTEMI non-ST-segment elevation myocardial infarction, CAD coronary artery disease

OP-082 INFLAMMATORY PARAMETERS AND HOMOCYSTEINE DILEMMA IN PATIENTS WITH DIABETES MELLITUS PRESENTING WITH FIRST TIME ACUTE CORONARY SYNDROME

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İstanbul Sancaktepe Şehit Profesör Doktor İlhan Varank Eğitim ve Araştırma Hastanesi

Objective: Our aim in this study was to investigate the role of serum inflammatory parameters in diabetic patients presenting for the first time with the acute coronary syndrome and to bring up the homocysteine debate.

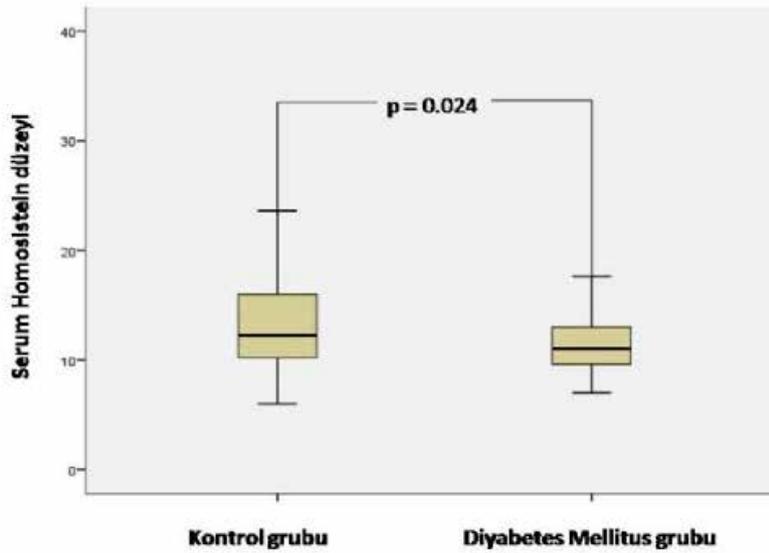
Materials-Methods: A total of 228 patients who underwent coronary angiography with a diagnosis of the acute coronary syndrome were included in the study. 64 of them were patients with type-2 DM, while 164 of them were the healthy control group. Patients were screened retrospectively and their demographic characteristics and laboratory findings were obtained from their medical records.

Results: The mean age of the patients with type-2 DM included in the study was (50.8 ± 7.1 years), and that of the control group was (48.6 ± 7.9 years). In the type-2 DM group, hypertension rate (p<0.000), blood glucose levels (p<0.000), triglycerides (p=0.002), C-reactive protein/albumin (CAR) ratio (p=0.012), vitamin B12 (p=0.015) and urine microalbumin levels (p=0.006) were found to be higher, while serum homocysteine levels (11.3±3.7 versus 13.7±5.8, p=0.010) were found to be lower. We performed multivariable logistic regression analyses to determine predictors that may be associated with diabetic coronary artery disease, CAR (OR:4.115 95%CI [1.007-16.812], p=0.018), serum homocysteine (OR:0.843 95% CI [0.771-0.997], p= 0.030) and urinary microalbumin levels (OR:1.044 95%CI [0.996-1.167], p=0.024) were found to be possible independent predictors of diabetic CAD.

Conclusion: In our study, we found that decreased serum homocysteine levels and increased inflammatory parameters are possible independent risk factors for patients with DM and CAD. Conducting more comprehensive studies on homocysteine may increase the accuracy of these results.

Keywords: Coronary artery disease, diabetes mellitus, homocysteine

Figure 1



Diyabetes mellitus grubu ile kontrol grubunun serum homosistein düzeylerinin karşılaştırılması.

Table 1.

değişkenler	Diyabetes mellitus grubu (n=64)	Kontrol grubu (n=164)	p-value
yaş (yıl)	50.8±7.1	48.6±7.9	0.097
Hipertansiyon, n (%)	41 (64.0)	114 (69.9)	0.000
STEMI, n (%)	25 (39.0)	67 (41.1)	0.092
trigliserid (mg/dL)	117.4±32.7	180.4±264.4	0.002
C-Reaktif protein/albumin	0.3±0.8	0.1±0.3	0.012
homosistein (µmol/L)	11.3±3.7	13.7±5.8	0.010

Koronar arter hastalığı olan diyabetes mellitus tanılı hastalar ile ve kontrol grubunun demografik, klinik ve laboratuvar bulguları açısından karşılaştırılması

OP-083 NUTRITIONAL STATUS AS A NEW PREDICTION TOOL FOR CORONARY COLLATERAL DEVELOPMENT

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Background: The nutritional status is predictive for the prognosis of cardiovascular diseases. The relationship of Prognostic Nutritional Index (PNI), which is an immunonutritional parameter, with cardiovascular diseases has been studied for a long time.

Objectives: Our aim in this study is to investigate whether PNI is associated with coronary collateral development.

Methods: This retrospective study included 172 patients with chronic total occlusion. The patients were diagnosed with stable coronary artery disease and coronary angiography was performed. PNI was calculated by using serum albumin level and lymphocyte count. Collateral circulation was classified according to the Rentrop grade.

Results: There was a positive correlation between PNI and Rentrop grade ($r=0.168$, $p=0.026$) and negative correlation between CRP and PNI ($r=-0.353$, $p<0.001$) and multivariate logistic regression analyses showed that uric acid and PNI were independent predictors of Rentrop grade ($p=0.008$ and $p=0.037$, respectively).

Conclusion: This study showed that PNI, which is easily calculated using the serum level of albumin and lymphocyte count, was a predictor of coronary collateral development in terms of Rentrop grade.

Keywords: Inflammation, Chronic Total Occlusion, Rentrop Grade

Univariate and multivariate logistic regression analysis for assessment of predictors of Rentrop grade.

Variables	OR (%95 CI)	P value	OR (%95 CI)	P value
CRP	0.986 (0.970-1.002)	0.087	0.990 (0.971-1.009)	0.283
Hypertension	0.481 (0.177-1.307)	0.151		
Uric Acid	0.731 (0.595-0.898)	0.003	0.736 (0.586-0.923)	0.008
LDL-C	0.976 (0.958-0.994)	0.009	1.006 (0.997-1.014)	0.201
Hemoglobin	1.159 (1.002-1.340)	0.047	1.115 (0.927-1.343)	0.248
PNI	1.055 (1.006-1.107)	0.028	1.006 (1.003-1.072)	0.037

OP-084 THE RELATIONSHIP BETWEEN LEVELS OF SEX STEROIDS AND CORONARY COLLATERAL CIRCULATION IN MEN PATIENTS WITH CORONARY ARTERY DISEASEAslan Erdoğan¹, Ender Özgün Çakmak², Cevat Kirma²¹Çam and Sakura education and training hospital²Kartal Kosuyolu education and training hospital

The Relationship Between Levels of Sex Steroids and Coronary Collateral Circulation in Men Patients with Coronary Artery Disease

Background and Aims: Coronary collateral circulation (CCC) is a natural bypass system for restoring blood flow and well-developed CCC is held to protect myocardial function and improve survival after coronary obstruction in patients with CAD. Sex steroids has been suggested to play a potentially protective role against the development and progression of CAD. We explored the relationship serum levels of sex steroids and coronary collateral circulation.

Methods-Results: A total of 115 males with stable coronary artery disease and whose have at least one total coronary artery occlusion included. Patients were divided into two groups including well CCC group (Rentrop grades 2-3, n=64) and poorly developed CCC group (Rentrop grades 0-1, n=51). Serum levels of total testosterone, free testosterone, sex hormone-binding globulin (SHBG) and dehydroepiandrosterone sulfate (DHEA-S) were recorded. Serum total testosterone (ng/dl; 274.5 ± 57.7 vs. 329 ± 64.8, p<0.001), free testosterone (pg/ml; 8.2 ± 2.4 vs. 12 ± 3.2, p<0.001), DHEAS (µg/dl; 111 [58] vs. 160 [85.5], p<0.001), and SHBG concentrations (nmol/L; 29.3 ± 8.6 vs. 33.2 ± 10.2; p=0.027) were significantly higher in the well coronary collateral group (WCG). According to the results of multiple regression analyses, free testosterone level (OR:0.57, CI [0.44-0.74], p<0.001), and SHBG level (OR:0.91, CI [0.84-0.99], p=0.022) were determined as independent predictors.

Conclusions: To the best of our knowledge, the present study is the first clinical study to identify high serum levels for sex steroids are a predictor of well coronary collateral development in male patients with coronary artery occlusion.

Keywords: Coronary Artery Disease, Gonadal Steroid Hormones, testosterone

OP-085 DOES ADDING DURATION OF ST DEPRESSION TO DUKE TREADMILL SCORE EFFECT DIAGNOSTIC ACURACY OF EET TO PREDICT OBSTRUCTIVE CORONARY ARTERY DISEASE ?

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Background:

Exercise electrocardiography (EET) is a safe and cost-effective method to predict the presence, prognosis, and severity of coronary artery disease (CAD).

Recent guidelines diminished the importance of EET, because EET has a lower sensitivity and specificity to predict obstructive CAD compared to other noninvasive imaging modalities. Yet, EET still remains in the diagnostic algorithm of CAD. Especially in centers where other diagnostic modalities are not feasible. Various score models have been developed to increase predictive power of EET. But still Duke treadmill score (DTS) is the most used and accepted scoring system.

Objective:

We aimed to evaluate whether adding ST depression (STD) duration could have an effect on increasing the value of Duke treadmill score (DTS) in predicting obstructive CAD.

Methods:

Patient population:

This study was designed as a single-center, cross-sectional observational prospective study.

A total of 258 consecutive patients without a history of prior CAD, admitted to the outpatient clinic with chest pain, underwent EET, and had subsequent invasive coronary angiography (ICA) according to their clinical likelihood of obstructive CAD were recruited.

The decision to perform EET for CAD diagnosis was given by the patients' primary cardiologists who were blinded to the study.

Excercise Electrocardiography Test:

EET was performed according to Bruce protocol. DTS was calculated for all patients. The duration of ST depression were calculated in milliseconds.

The total duration of ST depression was calculated as the time from the onset of the ST-segment depression during exercise, till the ST-segment depression resolution time in the recovery phase.

The new score -revised DTS- was calculated by adding total ST depression time to classical DS parameters. We compared area under the curve (AUC) of DTS and revised DTS by Delongi method.

Results:

Mean age of the group was 58.43±9.37, and 37.2% (n=96) were female.

Mean total ST-depression duration was 171 msec in normal artery group, 241 msec in non-obstructive CAD group, and 281 msec in obstructive CAD group. ST-depression duration in both exercise and recovery, and total ST depression duration were significantly higher in obstructive CAD group than non-obstructive and normal artery groups.

Revised DTS had significantly higher predictive value of obstructive CAD compared to classical DS.

The AUC of DS was significantly lower than the new score (z-score:3.274, p=0.011).

Discussion:

The diagnostic performance of DTS for obstructive CAD improved by adding total STD duration to traditional parameters of DTS.

Revised-DTS had a higher sensitivity, specificity, PPV, and NPV compared to classical DTS (p<0.001).

The predictive accuracy of DTS only ranges from 65-77% in various studies.

STD (horizontal or downsloping) is the most reliable indicator of exercise induced ischaemia.

However, the diagnostic accuracy of the exercise induced ST segment depression is only about 65%.

Time-course analysis of STD has also a crucial role in EET accuracy. Early onset and late offset of STD is correlated with high prevalence of cardiovascular events and advanced coronary obstruction.

Our study was concordant with these studies.

the duration of STD was longer in patients who had obstructive CAD, both in excersie and recovery phase, than non-obstructive CAD and normal arteries.

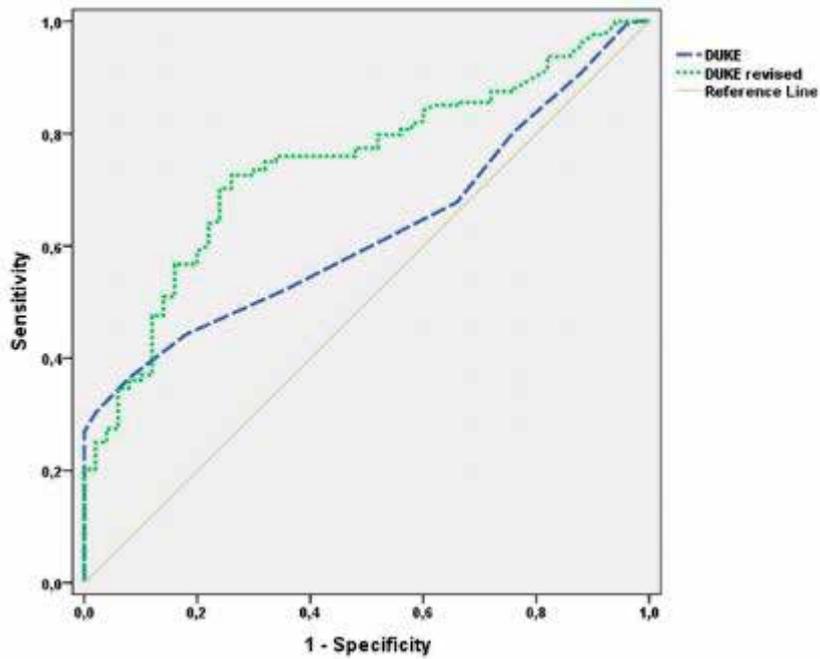
Conclusion:

To the best of our knowledge, although various STD duration patterns during EET have been shown to predict severe CAD in many studies, addition of total STD duration to DTS parameters has not been evaluated before.

Addition of total ST depression duration to the classical DTS improves the diagnostic accuracy of DTS.

Revised-DTS may be considered as an alternative scoring system for EET.

ROC curves of DUKE score and DUKE revised score in predicting obstructive CAD



OP-086 CHA2DS2-VASC SCORE AS A PREDICTOR OF GRAFT FAILURE AFTER CORONARY ARTERY BYPASS SURGERY

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Background: Graft patency is one of the major concerns after coronary artery bypass graft (CABG) surgery. CHA 2 DS 2 -VAsc is a score that developed to predict the risk of thrombotic events in patients with atrial fibrillation (AF). In this study, we evaluated the CHA 2 DS 2 -VAsc score as a simple tool for predicting graft failure (GF) among patients who underwent CABG surgery.

Method: In this retrospective case-control study, after the exclusion criteria had been applied, a total of 280 patients were enrolled. Angiograms were analyzed by using QCA software system for each patient. A graft was described as failed if it had 70% or more stenosis or was completely occluded. Patients were classified into two groups, group one included patients without graft failure (GF-) and group two included patients with graft failure (GF+). Thereafter, CHA 2 DS 2 -VAsc risk score was calculated for each patient.

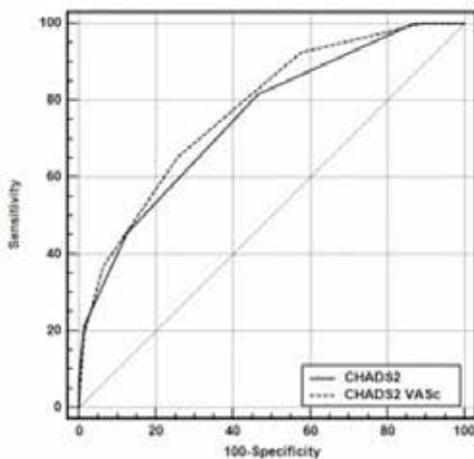
Results: 136 patients had graft failure (GF+ group) and 144 patients did not have graft failure (GF- group). GF- and GF+ patients had their angiography performed 100.31 ± 8.04 and 103.49 ± 8.41 months after CABG, respectively. GF+ group had significantly higher rate of DM, hypertension and HFREF. GF+ group had higher CHA 2 DS 2 (GF- group: 1.47 ± 0.91 vs. GF+ group: 2.57 ± 1.17, P= 0.0001) and

CHA 2 DS 2 -VAsc score (GF- group: 2.80 ± 1.11 vs. GF+ group: 4.15 ± 1.25, P=0.0001). Analyses showed that only CHA 2 DS 2 -VAsc was associated with presence of GF and was independent predictor of GF while other parameters of DM, hypertension, HFREF, creatinine and CHA 2 DS 2 were not found to be independent predictors of GF. A CHA 2 DS 2 -VAsc score of >3 predicted GF with a sensitivity of 65.44%, a specificity of 74.31%.

Conclusions: CHA 2 DS 2 -VAsc score might be used as feasible and simple method to predict the risk of graft failure after CABG surgery.

Keywords: CHA2DS2-VAsc, Coronary artery bypass surgery, Graft patency, Graft, failure

Figure-1



ROC analysis of CHA 2 DS 2 -VAsc score for graft failure

Table 1

Table 1. Demographic, clinical, laboratory and angiographic characteristics of the patients

	Graft Failure (-) n=144	Graft Failure (+) n=136	P value
Age, years	58.52 ± 8.1	60.24 ± 7.96	0.074
Gender			0.048
Female, n (%)	71 (11.36%)	35 (25.74%)	
Male, n (%)	73 (51.64%)	101 (74.26%)	
Smoking, n (%)	34 (23.6%)	67 (49.2%)	0.001
Diabetes Mellitus, n (%)	64 (44.4%)	84 (61.7%)	0.004
Hypertension, n (%)	106 (73.6%)	131 (96.3%)	0.0001
Previous MI, n (%)	71 (49.3%)	69 (50.7%)	0.841
Previous PCI, n (%)	64 (44.4%)	36 (26.5%)	0.581
History of PAD, n (%)	11 (7.6%)	16 (11.7%)	0.242
History of Cardiovascular Event, n (%)	20 (13.9%)	26 (19.1%)	0.239
HFREF, n (%)	54 (37.5%)	66 (48.5%)	0.041
Time interval between operation and angiography (months)	100.31 ± 8.04	103.49 ± 8.41	0.093
CHA2DS2	1.47 ± 0.91	2.57 ± 1.17	0.0001
CHA2DS2-VAsc	2.80 ± 1.11	4.15 ± 1.25	0.0001
Total Cholesterol, mg/dl	202.06 ± 52.92	207.69 ± 65.26	0.366
HDL, mg/dl	39.63 ± 12.92	38.26 ± 10.32	0.349
LDL, mg/dl	130.53 ± 42.68	131.94 ± 42.42	0.846
Triglyceride, mg/dl	192.87 ± 91.17	209.41 ± 115.39	0.178
Glucose, mg/dl	132.17 ± 36.44	143.07 ± 64.32	0.413
HbA1c, %	6.9 ± 1.68	7.19 ± 1.98	0.099
Creatinine, mg/dl	0.91 ± 0.23	1.09 ± 0.36	0.0001
Hemoglobin, g/dl	13.58 ± 1.66	13.66 ± 1.74	0.701
Number of Grafts	3.19 ± 0.8	3.19 ± 0.9	0.101
LAD-IMA, n (%)	177 (45.14%)	131 (96.32%)	0.625
LAD Saphenous, n (%)	9 (6.2%)	18 (13.2%)	0.100
D1 Saphenous, n (%)	69 (48.7%)	69 (50.7%)	0.857
IM Saphenous, n (%)	18 (12.7%)	24 (17.6%)	0.053
CX Saphenous, n (%)	32 (22.2%)	30 (22.0%)	0.974
OM Saphenous, n (%)	87 (60.8%)	87 (63.9%)	0.540
BCA Saphenous, n (%)	71 (49.3%)	68 (50%)	0.908
PDA Saphenous, n (%)	40 (27.8%)	30 (22.0%)	0.349
PL Saphenous, n (%)	4 (2.8%)	5 (3.6%)	0.670

MI: myocardial infarction, PCI: percutaneous coronary intervention, PAD: peripheral arterial disease, HFREF: heart failure with reduced ejection fraction, AF: atrial fibrillation, HDL: high-density lipoprotein, LDL: low-density lipoprotein, LAD: left anterior descending, LIMA: left internal mammary artery, D1: first diagonal artery, IM: intermediate artery, CX: circumflex artery, OM: obtuse marginal artery, RCA: right coronary artery, PDA: posterior descending artery, PL: posterolateral artery

Demographic, clinical, laboratory and angiographic characteristics of the patients

OP-087 SUCCESSFUL TREATMENT OF ACUTE TOTAL OCCLUSION OF THE LEFT MAIN CORONARY ARTERY (LMCA) LESION IN A PATIENT PRESENTED WITH CARDIOGENIC SHOCK

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Objective: Myocardial infarctions due to the acute total occlusion of the left main coronary artery (LMCA) are very rare clinical conditions with very poor prognostic outcomes. In acute total occlusion of LMCA, patients usually present with cardiogenic shock, since most of the myocardium is fed by LMCA. Fatal arrhythmias and sudden cardiogenic deaths are also common clinical conditions.

Method: A 57-year-old male admitted to the emergency department with new-onset chest pain and cold sweating, whose consciousness was blurred and blood pressure could not be measured suggesting cardiogenic shock. He did not have any notable medical history. Widespread ST segment elevation in the anterior leads and ST depression in the inferior leads were observed in the ECG of the patient. We performed an emergency coronary angiography which revealed that the LMCA was totally occluded. He was managed with percutaneous intervention with TAP (T And Protrusion) technique stenting to LMCA. The patient who was intubated in the emergency department was extubated in the coronary intensive care unit after the procedure. The patient was transferred to cardiology in-patient clinic 36 hours after the procedure and discharged 4 days after the procedure without any complications.

Results: Treatment options for acute total occlusion of the LMCA include coronary artery stenting, CABG, and thrombolytic therapy. Nevertheless, studies have shown that patients do not benefit from thrombolytic therapy. Additionally, coronary artery stenting was found to be superior to CABG, in achieving earlier reperfusion. However, it is shown that the incidence of ventricular tachycardia (VT) is higher in coronary artery stenting. On this basis, the most important factors in providing a successful treatment are the rapid diagnosis of the patient, the rapid admission to the catheter laboratory and the provision of TIMI 3 flow, which are all achieved in our patient, explaining his favorable outcome.

Conclusion: LMCA acute total occlusions are rare clinical conditions with a very high mortality rate despite successful reperfusion therapy. In patients with suspicious symptoms suggesting LMCA total occlusion and typical ECG findings, it is crucial to establish the diagnosis and decide the reperfusion procedure to be applied as soon as possible.

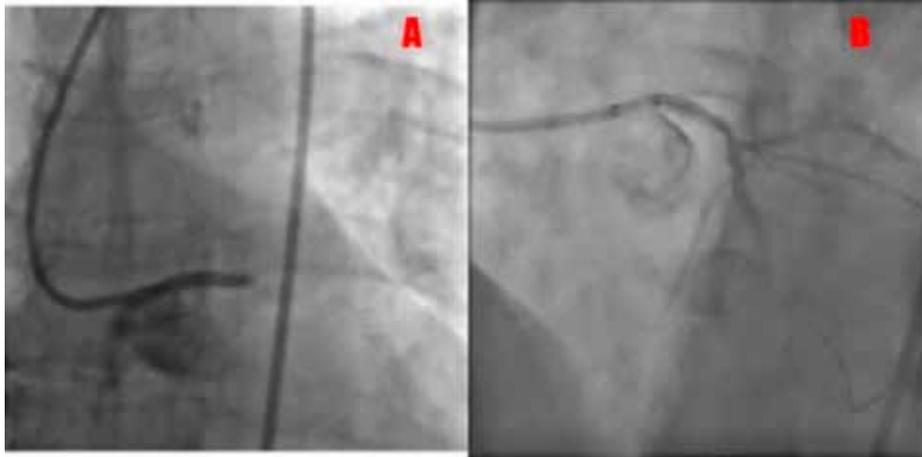


Figure 1: A: Patient's coronary angiography image that total occlusion of LMCA

B: Patient's coronary angiography image that stenting from ostium of LMCA to LAD

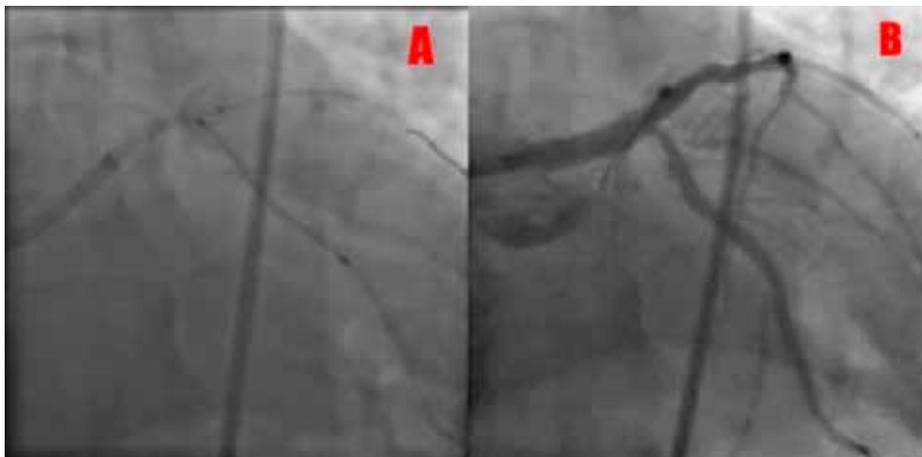


Figure 2: A: Patient's coronary angiography image that stenting CX by TAP technique

B: Patient's coronary angiography image that TIMI 3 flow after stenting

OP-088 WHICH TYPE OF DYSLIPIDEMIA IS MORE EFFECTIVE IN THE SEVERITY OF CORONARY ARTERY DISEASE?

Turgay Aslan

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Objective: Dyslipidemia can be defined as elevation of plasma total cholesterol, LDL cholesterol, triglycerides (TGs), or a low HDL cholesterol level that contributes to the development of atherosclerosis. Most of the cardiovascular risk calculators, such as SCORE risk charts, use total cholesterol level to calculate the cardiovascular risk. In the treatment of dyslipidemia, the first goal is to reduce LDL cholesterol. However, many patients have severe coronary artery disease although LDL values are not high or coronary artery disease continues to progress despite using statins and it is not clear which type of dyslipidemia causes more severe coronary artery disease. In this study we aimed to investigate which type of dyslipidemia is more effective in the frequency and severity of cardiovascular disease who have not used cholesterol-lowering drugs previously among a population sample from Turkey.

Methods: The severity of coronary artery disease was assessed by the syntax score. Dyslipidemia types were defined as low HDL [HDL <50mg / dL (in women), HDL <45 mg / dL (in men)] or high Triglycerides (TG> 200mg / dL) or high LDL (LDL> 100mg / dL) or high total cholesterol (total cholesterol> 200mg / dL) or Atherogenic dyslipidemia (AD: TG> 204mg / dL and HDL <33.9mg / dL)

Results: 1458 patients who underwent coronary angiography for the first time with indications of stable angina pectoris or acute coronary syndromes were retrospectively included in this study. Median age of patients were 61(24-87). According to the multivariate logistic regression analysis, male gender increases the risk of CAD by 3.487 times, atherogenic dyslipidemia 1.941 times, DM 1.789 times, low HDL 1.322 times, respectively. When evaluated with multivariate logistic regression analysis, other types of dyslipidemia do not seem to increase the risk of CAD. In patients with AD group, the median SYNTAX score was statistically significant higher compared to non- AD group [8(0-55) vs 2(0-55) p<0.001 respectively].

Conclusions: Although AD is not included in many cardiovascular risk assessment models, it was found to be the strongest risk factor among dyslipidemias in our study. AD which can be simply calculated in clinical practice, was correlated with the severity and frequency of CAD and should be kept in mind as a risk factor for coronary artery disease.

Keywords: coronary artery disease, syntax score, dyslipidemia

OP-089 REVASCULARIZATION OF ACUTE RENAL ARTERIAL OCCLUSION

Kubilay Erselcan

Tekirdag Dr. I. Fehmi Cumalioglu City Hospital

A 41 year old male patient who had a story of coronary stents and smoking, applied to emergency service with acute right flank pain. It was noted that costo-vertebral angle tenderness had been positive but defence and rebound had been negative. Grade 1 increase in parenchymal echojenity and 102X51mm dimensions in right kidney and was seen on abdominal USG, other organs was normal. Renal arterial resistive index had been measured as 0.69 and 0.60 in right interlobular artery and right renal artery respectively (RI normal range: 0.5 - 0.7) on doppler USG. After that, marked low perfusion according to the perfusion symmetry and severe stenosis had been seen in right renal artery on abdominal contrast-enhanced CT. The patient was sent to our hospital on the recommendation of angiography. Patient's pain was continuing when he was arrived to the catheter laboratory. An angiography was performed via right femoral punctation. Left renal artery was open but right renal artery was occluded. Coronary arteries were viewed to control coronary stents and were observed as open. We sat on to the right renal arterial ostium via a right coronary Judkins guiding catheter. 10000 IU UFH was given through the catheter. The lesion was crossed with 0.014' PT Graphix guidewire. Predilatation was performed 3.0x12mm coronary balloon. After first predilatation a weak filling was observed behind the lesion. The vessel revascularized via several predilatations with the same baloon after making sure the guidewire was in the true lumen. The lesion narrowing the lumen and thrombus was observed with blood flow. A 4.5x19mm coronary bare metal stent was impanted to the lesion and the vessel was completely opened. The procedure was ended without any complications. Loading doses of 300 mg ASA and 300 mg Clopidogrel were given. The pain disappeared within the first hour after the revascularization. 100 mg/day ASA and 75 mg/day Clopidogrel maintenance doses were planned. Oral and parenteral hydration were done int the post-op follow up. Renal disfunction was not observed during hospital stay and improvement in renal function was observed in outpatient clinic controls. We managed this case like myocardial infarction. Immediate transfer to the angiography center, imaging and revascularization were completed succesfully and renal functions were protected.

Keywords: renal artery occlusion, angiography, revascularization

occluded right renal artery



total occlusion was observed in right coronary artery

revascularized right renal artery



right renal artery and its branches after stent implantation

changing of renal functions

Date	Creatinine	BUN	eGFR
17.03.2021	0,8	36	111
13.04.2021	1,23	46	72
14.04.2021	1,26	34	70
15.04.2021	1,3	32	68
16.04.2021	1,24	29	71
22.04.2021	0,89	47	106
16.05.2021	0,89	37	106

patient's renal functions decreased during the occlusion and normalized rapidly after revascularization

OP-090 TAKOTSUBO CARDIOMYOPATHY AFTER MRNA-1273 SARS-COV-2 VACCINATION

Büşra Mavi, Mehmet Pişirici, Nihan Çağlar

Bakırköy Dr Sadi Konuk Eğitim Araştırma Hastahanesi

Takotsubo syndrome, it is also called stress cardiomyopathy is transient heart failure. This syndrome imitate myocardial infarction but there is not obstruction in coronary artery. In some case, stress cardiomyopathy cause mortality. Stress cardiomyopathy was first described in Japan. "Takotsubo" is the Japanese name of an octopus trap, which has a shape that is similar to the systolic apical ballooning appearance of the left ventricle. The pathophysiology is believed to be due to increased catecholamine levels in blood that results microvascular spasm and sistolic disfunction.

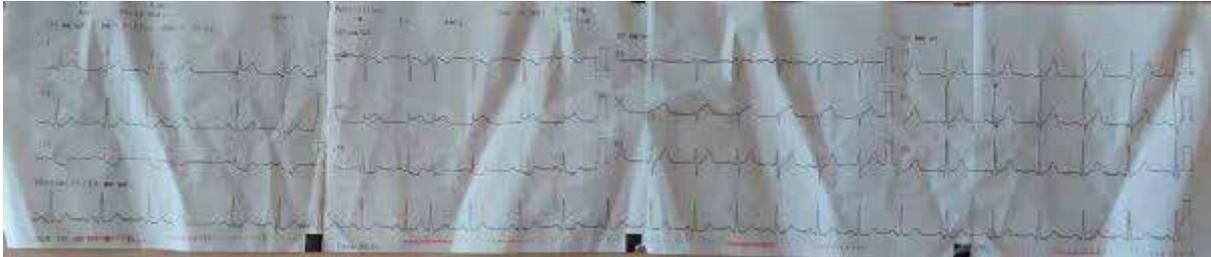
The health care systems concern about side effects of SARS-CoV-2 vaccines. We report a case of 55-year-old woman who didnt have any chronic disease, presented chest pain after two hours from SARS-CoV-2 vaccination associated with shortness of breath. The day before the second dose of vaccination she didn't have any complaint such as vomiting, diaphoresis, palpitation, fever, or chill. Blood work, including complete blood count, BUN, creatinine, and D-Dimer were within normal limits. Inflammatory markers, including C-reactive protein, sedimentation rate, D-dimer, lactate dehydrogenase were normal. The electrocardiogram showed evolving ST-T changes suggestive of anterolateral ischemia. By transthoracic echocardiography (TTE), there was systolic disfunction, especially left ventricular apical segments had been seen severely hypokinetic. Her ejection fraction was %40. The patient underwent coronary angiography, there was not critical coronary lesion.

We have observed our patient for three days. Right after coronary angiography patients symptoms were relieved. In lung auscultation there were fine crackles during inspiration. Apart from that physical examination was normal. During the observation period there was not any significant change in our daily echocardiographic examination. Patient's cardiac markers which have been taken one time per a day showed almost 6 times elevation from 80ng/l to 480ng/l. On the last day of hospitalisation Troponin T level was 461.4 ng/l. The ST elevations that we saw in the first ECG was disappeared. The last ECG before discharge was at sinusoidal rhythm without elevation. The patient was discharged on aspirin, perindopril, atorvastatin, bisoprolol, s pironolactone, pantoprazole. We recommended routine control one month after from discharge.

To sum up, we witnessed a serious cardiovascular event occuring suddenly after COVID 19 vaccination with mRNA vaccine. It is impossible from a single case to improve casuality. But authorities should be aware of possible adverse reaction of COVID 19 vaccination. We should emphasise, in spite of the fact that we report an adverse reaction to vaccination balance of risks favors COVID 19 vaccination, because COVID 19 itself can precipitate severe cardiovascular diseases. It will be important to search for other unusual events and further assesments on particular cases to better understand this new mRNA vaccine.

Keywords: Takotsubo, SARS-CoV-2 vaccines, Covid-19

ECG



ANTEROLATERAL ELEVATION

Echocardiography



apical ballooning

OP-091 FABRY DISEASE WITH ATRIOVENTRICULAR BLOCK AND SEVERE AORTIC STENOSIS: A DANGEROUS COMBINATION

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Objective: Fabry disease (FD) is an Xlinked lysosomal storage disorder caused by mutations in the GLA gene encoding a lysosomal hydrolase enzyme, α -galactosidase A. FD is a systemic disorder with multiple organ involvement. In the fourth decade, patients may manifest cardiomyopathy, arrhythmia, and cerebrovascular complications. Cardiac involvement as left ventricular hypertrophy (LVH), hypertrophic cardiomyopathy, and conduction disturbances are detected in 60% of Fabry patients. Electrocardiographic changes in patients with FD include LVH, short PR interval, supraventricular tachycardia, ventricular tachycardia, atrioventricular (AV) node blocks. A short PR interval is a common electrocardiographic finding in FD, but 2:1 AV block is a rare complication.

Here we report a case of a 43 years old man diagnosed with FD admitted to our hospital with 2:1 AV block, severe aortic valve stenosis (AS)

Case: A 43-year-old man diagnosed with FD was presented to our hospital with dizziness. His electrocardiogram (ECG) showed a 2:1 AV block (figure 1). He was diagnosed with Fabry in 2006. He was end stage kidney disease patient undergoing hemodialysis routinely. He took high dose beta-blocker (BB) regularly in recent years. The pulse rate was 45 beats/min. 5/6 grade murmur was heard at the Erb's point. The ECG showed a 2:1 AV block. Laboratory studies revealed end-stage renal failure; potassium was high level. Echocardiography revealed marked LV hypertrophy measured with no regional wall motion abnormalities. Doppler echocardiography showed severe AS (AVmax: 4.1 m/s, gradient: 67/37 mmHg) and mild mitral valve stenosis.

We stopped BB and we started medical treatment for hyperkalemia and fixed it. The next day his heart rhythm was atrial flutter, his heart rate was 80-90 beats/min, and the patient's complaints regressed.

Result: Cardiac complications are associated with high morbidity and mortality because of arrhythmia and heart failure and are currently the leading cause of death in FD. ECG findings in FD include PR interval changes, voltage signs of LVH and various degrees of an AV block. As FD progresses, lipid accumulation and fibrosis lead to the development of AV and bundle-branch blocks and sinus node dysfunction, which may require a pacemaker. In this case, the patient's rhythm spontaneously changed to atrial flutter rhythm. There is no need to install a pacemaker.

Conclusion: In FD, Aortic root dilation may also occur in the late stages of the disease, thereby contributing to aortic regurgitation. Unlike the literature, we found severe AS at an early age in our patient due to possible lipid accumulation and not detected aortic root dilatation.

We think that the AV block that developed in this patient was not only due to the electrolyte imbalance and the use of BB but also to the comorbidities closely related to AV blocks, such as FD and severe AS. Therefore, echocardiography and ECG follow-up should be done carefully in Fabry patients.

Keywords: Fabry, atrioventricular block, aortic valve stenosis, Fabry disease

figure 2



left ventricular hypertrophy and severe aortic valve stenosis, heart rhythm is atrial flutter

figure 1



2:1 av block

OP-093 A CASE REPORT OF PERMANENT PACE MACKER INSERTED, A CASE OF LARGE VESSELS VASCULITIS THAT CONSISTS WITH SWEATING, WEIGHT LOSS AND COUGH

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72-year-old female patient,

A permanent pacemaker was inserted 2 years ago due to the complete AV block developing while being followed up with diagnosis of hypertension, diabetes mellitus type 2, sarcoidosis. She applied to our clinic with night sweats, loss of appetite, weight loss and cough.

Physical examination: Active, oriented to place, time and person. Able to attend cooperatively with examiner. No edema, No lymphadenomegaly.

Blood pressure: 130/70 mmHg, Pulse: 70 b.p.m., regular. Normal S1, S2; no murmurs, rubs, or gallops. No prominent neck veins noted. Pulses bilaterally normal (radial, posterior tibial, dorsalis pedis).

Chest wall motion symmetric. Bronchovesicular breath sounds. No wheezes, rhonchi heard.

The abdominal examination revealed no features.

Labs: Ferritin: 231 ng/mL, LDH: 328 U/L, Total protein: 7.1 g/dL, Creatinine: 0.7 mg/dL, Calcium: 10 mg/dL, Albumin: 3.44g/dL, WBC: 8100/μL, PLT: normal, Hemoglobin:9.8 g/dL, CRP: 76 mg/L, Erythrocyte sedimentation rate: 123 mm/hour

Imaging

ECG: Sinus, Pacemaker rhythm, normal heart rate.

Echocardiography: There are degenerative changes in the mitral and aortic valve, 2 (+) Aortic insufficiency, 2-3 (+) Mitral insufficiency, 2 (+) Tricuspid insufficiency were detected. There was no evidence for endocarditis.

Thorax Computed Tomography: No signs were found for active infection or sarcoidosis activation in the lungs.

PET: The involvement of arcus aorta, thoracic aorta, brachiocephalic artery, bilateral subclavian, bilateral common carotid arteries and abdominal aorta up to L3 level has been evaluated in relation to typical vasculitis. No involvement was detected in terms of endocarditis.

The patient was hospitalized, followed and treated. It was reported that there was no evidence of sarcoidosis activation or tuberculosis in Respiratory Diseases consultation and no treatment was recommended.

After the rheumatology consultation, the patient was evaluated as large vessel vasculitis and prednisolone 1mg/kg dose was started as treatment. In the meantime, she was closely followed in terms of blood pressure and blood sugar, and her necessary treatment was arranged. With treatment, CRP and Erythrocyte sedimentation rate values gradually decreased and returned to normal. In addition to laboratory parameters, the patient improved clinically. The patient was taken out for follow-up. In patients who have permanent pacemakers and have the possibility of endocarditis and whose diagnosis cannot be excluded by echocardiography, it will be appropriate to consider PET as an additional examination.

Keywords: Large Vessels Vasculitis, endocarditis, sarcoidosis

OP-095 CLINICAL USABILITY OF THE MOST IMPORTANT THREE ATHEROGENIC INDICES FOR PREDICTION OF HYPERTENSION IN YOUNG POPULATION

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Introduction: Cardiovascular diseases are the leading cause of mortality all over the world. Atherogenic index of plasma, Castelli's risk index 1 and Castelli's risk index 2 are the novel cheaper prognostic markers for cardiovascular diseases. As hypertension is one of the leading causes of cardiovascular disease, its early prediction is very important for clinicians. In this study, we wanted to investigate whether these 3 new parameters are predictive for young hypertensive patients and which one is the most important for correct prediction.

Method: 666 young patients (age 18-40) who were admitted to a cardiology clinic with a 24 hours ambulatory blood pressure monitoring were included in the study. Patients' demographic data, blood parameters and ambulatory blood pressure recordings were noted. Patients with mean systolic blood pressure ≥ 130 mmHg and diastolic blood pressure ≥ 80 mmHg was assumed as hypertensive in accordance with European Society of Cardiology Hypertension Guidelines. Atherogenic index of plasma, Castelli's risk index 1 and Castelli's risk index 2 were calculated as log (Triglycerit/HDLc), Total cholesterol/HDLc and LDLc/HDLc, respectively. T-test was used for availability of these parameters for hypertension prediction. Also, these parameters were compared with ROC Curve analysis and K-fold cross validation methodology among themselves.

Results: 411 (61.7 %) patients were hypertensive. Median age was 33 (17-40). All of these indeces can be used for hypertension prediction ($p < 0.001$). For comparison of these indeces, ROC Curve analyses showed that Atherogenic Index of plasma, Castelli's risk index 1 and Castelli's risk index 2 have similar impact on prediction of hypertension ($p < 0.001$ AUC= 0.635, $p < 0.001$ AUC= 0.630 and $p < 0.001$ AUC= 0.615). Cut-off values were 0.5249, 4.0698 and 2.2808. Also, we used K-fold cross validation for advanced analysis to compare these parameters and found that Atherogenic index, Castelli's risk index 1 and 2 have similar outcome (accuracy= 0.635, kappa= 0.132; accuracy= 0.634, kappa= 0.109; accuracy= 0.620, kappa= 0.055, respectively)

Discussion: Routine blood parameters are cheap and easy accessible for developing countries. Indices generated from blood parameters are used for prediction of cardiovascular diseases. In this study, we investigated that whether three of major atherogenic indices can be used for hypertension prediction in young population. We found that Atherogenic index of plasma, Castelli's risk index 1 and Castelli's risk index 2 can be used for hypertension prediction. Although there is no statistically significant difference, Atherogenic index of plasma has a little better prediction than other indeces for young hypertensive patients.

Figure 1. ROC Curve Analyses of Atherogenic index of plasma, Castelli's risk index 1 and Castelli's risk index 2, respectively.

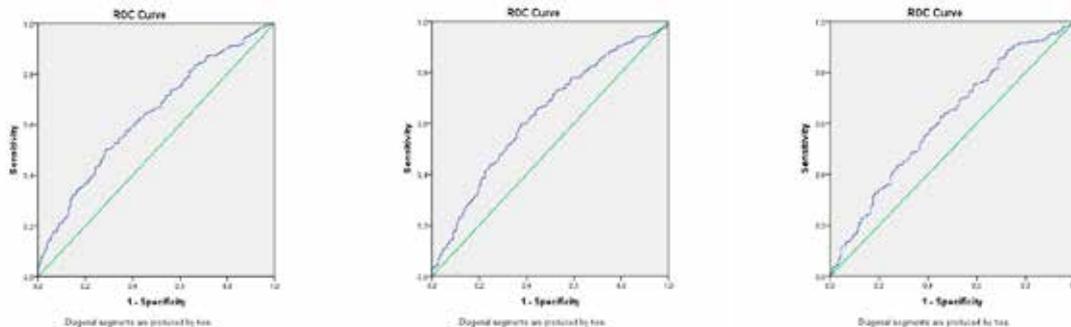


Table 1. Comparison of atherogenic indices according to T-test, K-fold cross validation and ROC Curve analysis

Parameters	Atherogenic index of plasma	Castelli's risk index 1	Castelli's risk index 2
T-Test results (p value)	<0.001	<0.001	<0.001
K-fold cross validation Accuracy Kappa	0.635 0.132	0.634 0.109	0.620 0.055
ROC curve analyses p value Area under curve	<0.001 0.635	<0.001 0.630	<0.001 0.615
Cut off points for ROC curve	0.5249	4.0698	2.2808

OP-096 THE ASSOCIATION OF NON-DIPPER HYPERTENSION WITH AORTIC SCLEROSIS IN HYPERTENSIVE PATIENTS

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Abstract

Background: Non-dipper blood pressure (NDBP) pattern is associated with end-organ damage and cardiovascular mortality. Predicting and managing the NDBP pattern can prevent cardiovascular adverse events. The study aims to investigate the relationship between NDBP pattern and aortic valve sclerosis (AVS) detected during routine echocardiography in hypertensive patients.

Method: A total of 222 patients using at least one antihypertensive drug were included in the study. The patients were divided into two groups as dipper and NDBP pattern according to ambulatory blood pressure monitoring (ABPM). NDBP pattern was defined as reducing night systolic BP by <10% compared to daytime systolic BP. Patients outside this definition were defined as dipper hypertensives. AVS was defined as the thickening or calcification in the semilunar cusps, without stenosis in transthoracic echocardiography.

Results: The average age of patients with the NDBP pattern was 68.21±6.22, while it was 66.80±5.38 for patients with dipper BP pattern (p=0.075). As a result of ABPM, dipper BP (45%) was detected in 100 patients, and NDBP (55%) was detected in 122 patients. AVS was detected in 39 patients (17.6%), and the majority of them were in the NDBP group (3.6% vs. 14%, p<0.001). As a result of multivariate regression analysis, the only independent predictor of the NDBP pattern was AVS (OR=3.078, 95% CI 1.280-7.403, p=0.012)

Conclusion: In hypertensive patients, AVS detected by transthoracic echocardiography is associated with NDBP. The presence of AVS may be an essential factor in the detection of the NDBP pattern, that is closely related to major cardiovascular events.

Keywords: Non-dipper blood pressure; aortic valve sclerosis; ambulatory blood pressure monitoring; echocardiography

Introduction

Hypertension (HT), known as high blood pressure, is the most common cardiovascular disease. Blood pressure (BP) has a circadian pattern that shows a 10% or more drop in BP at night compared to daytime. Non-dipper BP (NDBP) is the absence of 10% or more BP drop at night. ⁽¹⁾ Non-dipper BP poses high risk for end-organ damage, such as myocardial infarction, chronic kidney disease, stroke, and carotid artery disease. ⁽²⁻⁴⁾ Additionally, NDBP is observed more frequently in patients with aortic arch calcification. ⁽⁵⁾

Aortic valve sclerosis (AVS) is the thickening or calcification in the semilunar cusps not accompanied by stenosis and is closely related to HT. ⁽⁶⁻⁸⁾ Hypertension is an important factor in the development of valvular diseases. Hence, it can lead to the progression of early-stage aortic valve pathologies. ^(9,10) It is estimated that valvular diseases' progression can slow down with optimal BP management in hypertensive AVS patients. On the other hand, aortic sclerosis may lead to a decrease in aortic distensibility and compliance, leading to arterial stiffness and a disruption in the circadian pattern of BP. ⁽¹¹⁾ In the present study, we aimed to evaluate the relationship between aortic sclerosis detected by echocardiography and the NDBP pattern that causes cardiovascular events and end-organ damage in HT patients.

Method

Study population

Our research is a retrospective, cross-sectional, multicenter study. Patients presented to cardiology clinics with at least one antihypertensive drug usage and underwent ambulatory blood pressure monitoring (ABPM) between July 2019 and January 2020 due to unregulated blood pressure, were included in the study. Exclusion criteria were pregnancy, malignancy, moderate or severe heart valve disorders, chronic renal failure (eGFR <30 ml / min / 1.73m²) and <85% valid measurement rate of ABPM (n=117). Demographic, physical examination, laboratory, transthoracic echocardiography, and ABPM data of the patients were recorded from the hospital registry system. Our study was carried out under the Helsinki Declaration's principles and was approved by local Ethics Committee.

Ambulatory blood pressure monitoring

Ambulatory blood pressure monitoring was performed using the Mobil-O-Graph (MoG; IEM, Germany) device. Blood pressure measurements were obtained automatically at 30-minute intervals. The first hour data of the measurements were removed from the analysis. Daytime, nighttime, and 24-hour BP data were recorded. Daytime and night hours were defined as 07:00-23:00 and 23:00-07:00, respectively. Also, the time intervals, defined as night and day hours, were changed according to the patient's feedback. Based on the ABPM results, patients were divided into two groups as dipper-BP (DBP) and NDBP.

Obtaining Demographic and Laboratory Data

Diabetes mellitus was defined as antidiabetic usage, or fasting plasma glucose level above 126 mg/dL or blood glucose level above 200 mg/dL in any measurement. Patients who continued to use tobacco products, and those smoking cessation periods did not exceed one year were considered smokers. Coronary artery disease was considered as a history of coronary artery by-pass grafting or percutaneous coronary stent implantation. A history of stroke or transient ischemic attack was considered a cerebrovascular disease. Fasting blood sugar, total cholesterol, high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglyceride, and creatinine were measured by standard laboratory methods.

Echocardiography

Transthoracic echocardiography standard parasternal and apical images were obtained in the left lateral decubitus position using VIVID S5 (GE-Vingmed Ultrasound AS, Horten, Norway). The images were digitally stored and examined by a cardiologist blinded to patients' data to avoid inter-reader variability. Standard B-mode and M-mode parameters were measured according to the American Society of Echocardiography guideline. Aortic valve stenosis was defined as the thickening or calcification in aortic valve cusps while the transvalvular gradient was ≤2 m/s, in the absence of rheumatic valve disease. ⁽¹²⁾ (Figure 1)

Statistical analysis

Continuous variables were defined as mean \pm standard deviation; categorical variables were defined as percentages. Data and histograms were tested for normal distribution using the Kolmogorov-Smirnov test. The Student t-test was used for univariate analysis of normally distributed continuous variables, Mann-Whitney U test was used for non-normally distributed numerical variables, and Chi-Square test was used for categorical variables. $P < 0.05$ was defined as significant. A multivariate logistic regression test was used to identify NDBP predictors. The goodness of fit was evaluated by the Hosmer-Lemeshow test. The Statistics Program for Social Sciences (Windows 15; SPSS Inc, Chicago, Illinois) was used for statistical calculations.

Results

A total of 222 hypertensive patients (94 men, 128 women) were included in the study. As a result of ABPM, DBP was detected in 100 patients (45%), and NDBP was detected in 122 patients (55%). The average age of patients with NDBP pattern was 68.21 ± 6.22 years, whereas it was 66.80 ± 5.38 years for patients with DBP pattern ($p=0.075$). There was no gender difference between the groups ($p=0.145$). The number of antihypertensive drugs used among the groups was similar (2.3 ± 1.1 vs. 2.1 ± 1.1 , $p=0.073$). Aortic valve sclerosis was detected in 39 patients (17.6%). Of the AVS patients, 31 (14%) were in the NDBP group, eight (3.6%) were in the DBP group, and patients with AVS were more common in the NDBP group ($p < 0.001$). The presence of coronary artery disease (CAD) was statistically higher in the NDBP group ($p=0.029$). (Table 1)

There was no difference of 24-hour systolic BP ($p=0.948$) and the 24-hour diastolic BP ($p=0.642$) between the groups. Daytime systolic BP ($p=0.042$) and diastolic BP ($p=0.003$) were higher in the DBP group. Night systolic BP ($p < 0.001$) and diastolic BP ($p < 0.001$) were significantly higher in the NDBP group (Table 2).

In multivariate regression analysis, AVS was the only independent predictor for NDBP (OR=3.078, 95% CI 1.280-7.403, $p=0.012$).

Discussion

Whether blood pressure is regulated is often based on daytime office BP measurements. The nighttime BP and NDBP patterns are often disregarded in clinical practice due to the difficulty of measurement. Abnormalities in BP measurements at night are closely related to cardiovascular mortality. In the circadian cycle of BP, a decrease of 10% or more is expected in nighttime systolic BP levels than daytime systolic BP levels. Every 5% decline from the reduction in night BP levels causes an approximately 20% increase in cardiovascular mortality risk. It is claimed that cardiovascular mortality can be reduced by providing a better nighttime BP control.⁽¹³⁾ Therefore, nighttime BP control is emphasized in HT treatment. Today, ABPM is the only method that can be used to diagnosis of BP patterns at night.

Aortic valve sclerosis is the thickening or calcification in aortic valve leaflets, provided that the transvalvular gradient is ≤ 2 m/s and is not accompanied by rheumatic valve disease.⁽¹²⁾ Aortic valve sclerosis usually occurs as chronic inflammation, fibrosis, and calcification when the normal processes of aortic valve interstitial cells are impaired.^(14,15) Histopathological abnormalities include lipoprotein accumulation, cellular infiltration, and extracellular matrix formation, resulting in progressive macroscopic valve thickening. Increased calcification leads to outflow obstruction due to limitation of motion in the valve, and in the last stage, it causes aortic stenosis.⁽¹⁶⁻¹⁸⁾ Besides, AVS is associated with increased end-organ damage and cardiovascular mortality.⁽¹⁹⁾ It has been reported that the diagnose of AVS can increase the sensitivity and specificity of diagnosing CAD by echocardiography, and assist in the management of cardiovascular prevention strategies.^(20,21)

Aortic sclerosis is known to be associated with hypertension.⁽⁹⁾ The relationship between arterial BP and AVS is paradoxical. Increased arterial BP can facilitate aortic valve calcification and vice versa. Patients with NDBP pattern are exposed to an abnormal nocturnal BP load, accelerating aortic calcification and stiffness.⁽⁵⁾ This result suggests that AVS may be related to the NDBP. However, there is insufficient data regarding which of the dipper BP or NDBP pattern is more related to AVS. Aortic valve sclerosis and NDBP pattern increase the risk of end-organ damage and cardiovascular mortality independently. As a result of the research, we observed a high rate of NDBP patterns in HT patients with AVS. If we speculate, AVS and NDHT, which are associated with an increase in cardiovascular mortality, may lead to an additive increase in the risk of end-organ damage and cardiovascular mortality in case of coexistence. This hypothesis reveals the importance of detecting AVS patients with NDHT. It should be considered that the possibility of having NDHT by AVS detection during routine echocardiography may be high, and it should be kept in mind that the ABPM test may be useful for diagnosing NDHT in hypertensive patients.

Limitation

The diagnosis of NDBP pattern was based on night and daytime systolic blood pressure changes. Although this definition is the most commonly used one for the NDBP pattern, changes in diastolic BP levels at night and daytime can also be used to describe the NDBP pattern.

Conclusion

The AVS detected during routine echocardiography in patients with hypertension is independently associated with the NDBP pattern. In clinical practice, the diagnosis of AVS may contribute to the detection and control of night BP. Thus, it can provide early diagnosis and treatment against the risks that may arise from NDHT.

Conflict of interest

The authors report no conflicts of interest

Funding Statement

There were no external funding sources for this study.

Ethical approval

The study was approved by the Ethic Committee of the Recep Tayyip Erdogan University (December 03, 2020, KA 2020/230).

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Table 1 Baseline characteristics of the study groups

Variables	Dipper (n=100)	Non-Dipper (n=122)	P
Age (year)	66.80 ± 5.38	68.21 ± 6.22	0.075
Aortic sclerosis, n (%)	8 (8)	31 (% 25.4)	0.001
CAD (n, %)	60 (60)	90 (% 73.8)	0.029
Gender	37 (37)	57 (% 46.7)	0.145
Diabetes (n, %)	14 (% 14)	25 (% 20.5)	0.206
Heart failure (n, %)	8 (% 8)	7 (% 5.7)	0.504
Hyperlipidemia (n, %)	35 (% 35)	53 (% 43.4)	0.201
Smoking (n, %)	16 (% 16)	28 (% 23)	0.196
GFR (mL/min/1.73 m ²)	82.54 ± 19.03	80.15 ± 18.93	0.369
Creatinine (mg/dL)	0.86 ± 0.32	0.88 ± 0.30	0.601
Glucose (mg/dL)	107.64 ± 33.24	105.39 ± 28.80	0.592
BUN (mg/dL)	27.26 ± 15.56	25.72 ± 13.85	0.437
Uric Acid (mg/dL)	5.54 ± 1.58	5.72 ± 1.71	0.494
T cholesterol (mg/dL)	210.60 ± 48.67	200.32 ± 55.43	0.165
Triglyceride (mg/dL)	139.35 ± 71.83	145.07 ± 67.75	0.559
LDL-C (mg/dL)	131.84 ± 42.04	123.18 ± 47.83	0.176
HDL-C (mg/dL)	52.05 ± 12.70	49.10 ± 12.14	0.092
CRP (mg/dL)	5.30 ± 14.94	3.92 ± 4.52	0.386
WBC (mg/dL)	7.32 ± 2.16	7.42 ± 1.88	0.715
Hemoglobin (mg/dL)	13.65 ± 1.51	13.51 ± 1.36	0.475
LA diameter (mm)	37.78 ± 3.94	38.11 ± 4.14	0.575
LVD diameter (mm)	52.25 ± 5.38	45.07 ± 5.95	0.168
LVS diameter (mm)	29.39 ± 4.81	29.61 ± 5.74	0.769
Ejection fraction	60.67 ± 5.80	60.28 ± 6.12	0.645
IVS (mm)	12.01 ± 1.65	12.32 ± 1.80	0.215
PW (mm)	10.98 ± 1.45	11.11 ± 1.25	0.492
ACEI (n, %)	44 (% 44)	58 (% 47.5)	0.598
ARB (n, %)	33 (% 33)	27 (% 22.1)	0.070
Nitrat (n, %)	11 (% 11)	16 (% 13.1)	0.631
Diüretik (n, %)	64 (% 64)	49 (% 40.2)	<0.001
Statin (n, %)	46 (% 46)	68 (% 55.7)	0.149
Betablocker (n, %)	54 (% 54)	73 (% 59.8)	0.382
CCB (n, %)	41 (% 41)	47 (% 38.5)	0.708
Number of HT drugs	2.3±1.1	2.1±1.1	0.073

ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, BUN: Blood urea nitrogen, CAD: Coronary artery disease, CCB: Calcium channel blocker, CRP: C-reactive peptide, GFR: Glomerular filtration rate, HDL-C: High-density lipoprotein cholesterol, HT: Hypertension, IVS: Interventricular septum, LA: Left atrium, LDL-C: Low-density cholesterol, LVD: Left ventricular diastole, LVS: Left ventricular systole, PW: Posterior wall, WBC: White blood cell

Table 2 Ambulatory blood pressure variables of the study groups

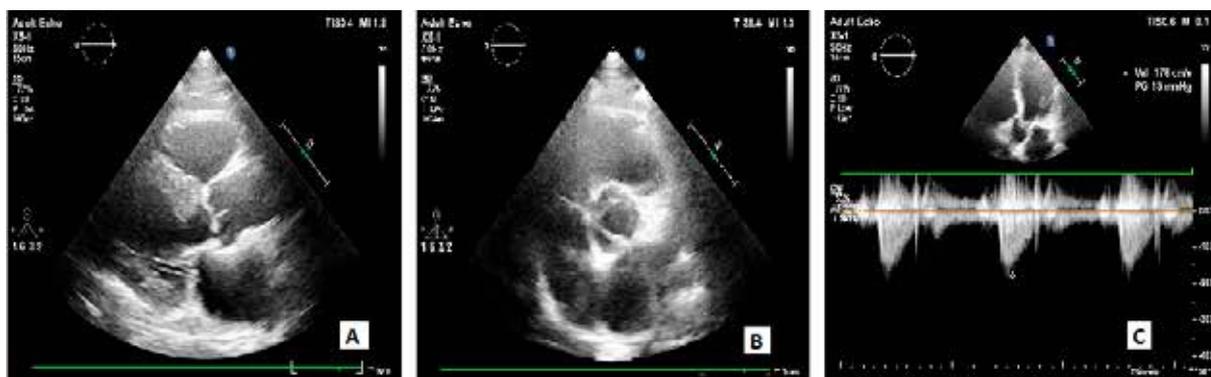
Variables	Dipper (n=100)	Non-Dipper (n=122)	P
Mean SBP (mmHg)	128.75 ± 17.73	128.89 ± 14.76	0.948
Mean DBP (mmHg)	77.88 ± 11.18	77.22 ± 9.86	0.642
Daytime mean SBP (mmHg)	133.54 ± 18.33	128.94 ± 15.18	0.042
Daytime mean DBP (mmHg)	82.11 ± 11.37	77.78 ± 10.06	0.003
Night mean SBP (mmHg)	118.47 ± 18.55	128.71 ± 15.11	<0.001
Night mean DBP (mmHg)	69.53 ± 11.59	75.73 ± 10.26	<0.001
% SBP (mmHg)	12.60 ± 5.55	-0.58 ± 6.32	<0.001
% DBP (mmHg)	16.25 ± 5.01	1.61 ± 7.13	<0.001

DBP: Diastolic blood pressure, SBP: Systolic blood pressure

Table 3 Multivariate analysis of non-dipper blood pressure pattern

Variables	Odds Ratio, 95 CI%	p value
Aortic valve sclerosis	3.078 (1.280-7.403)	0.012
Coronary artery disease	1.439 (0.710-2.915)	0.313
Diuretic	0.572 (0.222-1.476)	0.248
Angiotensin receptor blocker	0.822 (0.390-1.733)	0.606
Number of hypertension drugs	0.962 (0.658-1.405)	0.840
Age	1.042 (0.986-1.100)	0.143

Figure 1: The Appearance of Aortic Sclerosis Echocardiographically



OP-097 THE RELATIONSHIP OF SLEEP QUALITY WITH DIPPER AND NON-DIPPER BLOOD PRESSURE PATTERNS IN ESSENTIAL HYPERTENSIVE PATIENTS DURING COVID 19 PANDEMIC

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Objective; The aim of this study was to evaluate the relationship between dipper/non-dipper patterns and impaired sleep quality in patients with essential hypertension during Covid 19 pandemic. Additionally, we aimed to explore the effect of anxiety due to pandemic on the sleep quality, and the circadian rhythm of ambulatory blood pressure.

Method; Fifty patients with essential hypertension aged between 35-50, who applied to the cardiology outpatient clinic during the Covid pandemic, were included in the study. Individuals who did not have sleep disorders before the Covid period and did not have any psychiatric disorders were included in the study. Patients with any other chronic disorder, with hypo-hyperthyroidism, with a BMI > 25 kg/m², and patients using medications which are known for their negative impact on sleep quality, including diuretics, b blocking agents were also excluded. The blood pressure patterns of the individuals participating in the study were classified as dipper and non-dipper after 24-hour ambulatory blood pressure measurement. Sleep quality and sleep disorders were evaluated with the Pittsburg sleep quality index (PSQI) scale, and anxiety levels were evaluated with the Beck Anxiety Inventory. The obtained data were evaluated with appropriate statistical methods.

Results; 46% (n=23) of the individuals participating in the study were classified as dipper and 54% (n=27) as non-dipper. Poor sleep quality (PSQI > 5) was found higher in the non-dipper group compared to the dipper group (p < 0.05) and there was a significant difference in PSQI score between dipper and non-dipper groups (3.78 ±2.51 vs. 5.48 ±3.32, p < 0.001). In addition, the 24-hour mean systolic blood pressure values of individuals with non-dipper sleep patterns were found to be higher than those with dipper sleep patterns (p=0.034). There was a significant correlation between PSQI score and anxiety levels in patients with hypertension (r = 0.638, p < 0.05). Also, there was a significant positive correlation between Beck anxiety score and systolic blood pressure values (r = 0.594, p < 0.05). The logistic regression model was statistically significant, $\chi^2(12) = 27,402$, p < 0005 and binary logistic regression analyzed showed that sodium (Na) levels, PSQI score), and Beck anxiety score were factors that could predict non dipper pattern in patients with essential hypertension (Table 1).

Conclusion: In our study, it was found that there was a relationship between non-dipper blood pressure pattern and impairments of sleep quality, and that expected circadian changes in blood pressure were not observed as sleep quality deteriorated. Also, there was a significant correlation between sleep quality and anxiety levels patients with hypertension. Higher anxiety scores and lower sleep quality were factors that could predict non dipper pattern in patients with essential hypertension. In this context, it is obvious that increasing sleep quality and controlling anxiety levels will have a positive effect on blood pressure regulation. Studies with large samples are needed in this area.

Table 1. Logistic Regression of Non-dipper pattern in patient with essential hypertension

	B	P value	Odds ratio (95% CI)
Age	0.06	0.80	1.006
Sex	1.950	0.48	1.089
BMI	0.54	0.44	0.934
Na	0.098	<0.001	.172
Cl	-.053	0.58	.156
K	-.038	0.77	.178
Ca	0.030	0.65	.183
Average Blood pressure (24 Hours)	6.56	0.28	3.181
Average Heart rate (24 Hours)	4.38	0.12	1.156
Average Systolic Blood pressure (24 Hours)	7.04	0.26	1.06
Smoking	0.05	0.56	0.385
Beck anxiety scores	1.34	0.02	.353
PSQI scores (<5, >5)	0.47	0.006	.063

* Statistically significant (p<0.05)

OP-098 THE EFFECT OF RAMADAN FASTING ON ENDOTHELIAL FUNCTION IN PATIENTS WITH HYPERTENSION

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Introduction: Ramadan month is accompanied by fasting which starts at early sunrising and ends at sundown. In Ramadan months, several changes are occurred in muslims daily and eating habits. Because drinking and eating are prohibited in daytime in Ramadan fasting, people may take excessive calories, sweat or junk food after fasting dinner. It may affect endothelial function negatively. Hypertension is a disorder which is closely associated with unbalanced nutrition and sedentary lifestyle. Many negative effects of hypertension are resulted of impaired endothelial functions. Previously, there was few data about the effects of Ramadan fasting on endothelial function. Flow mediated dilatation is accepted as surrogate marker of endothelial function. In this study we evaluate flow mediated dilatation in 40 hypertensive patients in 1-3 days before Ramadan and last 1-3 days of Ramadan.

Methods: 40 hypertensive patients (23 male, age average: 51,4 ± 9,1; 17 female age average: 50,1 ± 8,6) which fast during Ramadan month with their self-consents, were enrolled to study. The study was performed during 9 April 2021 to 12 May 2021 in Kayseri City Education and Research Hospital. Patients with diabetes mellitus, known coronary artery disease, chronic kidney and liver disease and any health problem to prevent to get Ramadan fasting were excluded from the study. Baseline characteristics features such as anti-hypertensive drugs, smoking habits, weight and height were recorded at 1-3 days before Ramadan month. Blood pressure, heart rate, weight and flow mediated dilatation were measured at 1-3 days before Ramadan month and last 1-3 days of Ramadan month. Flow mediated dilatation were measured from brachial artery. Baseline brachial artery diameter was measured. Inflating of cuff 50 mmHg above systolic blood pressure was used for occurring ischemia. 30-90 second after ischemia, brachial artery diameter was measured again. The diameter difference percentage to baseline brachial artery diameter was called as flow mediated dilatation.

Results: 17 patients had smoking habit. Patients' drug using condition was shown in table-1. Patients numbers of under treatment of 2, 3, 4 anti-hypertensive drugs combination were 18, 9 and 4, respectively. Baseline and after Ramadan month weight and bmi were 79,5 ± 12,7 kg vs 78,1 ± 12,0 kg and 25,1 ± 4,0 vs 24,6 ± 3,8, respectively (p=0,01 and p= 0,01) (table-1). Baseline and after Ramadan month systolic, diastolic blood pressure and heart rate were 137,3 ± 15,0 mmHg vs 129,4 ± 10,5 mmHg, 81,7 ± 8,8 mmHg vs 78,6 ± 6,5 mmHg and 77,4 ± 10,8 vs 73,4 ± 7,0 beat/min, respectively (p=0,01, p=0,05 and p=0,01) (table-1). Baseline and after Ramadan month flow mediated dilatation were 8,8 ± 3,7 % vs 10,8 ± 3,8 % (p=0,01) (table-1).

Conclusion: This study showed that Ramadan fasting have positive effects on weight loss, blood pressure and flow mediated dilatation.

Keywords: Ramadan fasting, endothelial function, flow mediated dilatation

Baseline characteristics, blood pressure, heart rate and flow mediated dilatation chancing after Ramadan fasting

	Baseline	After Ramadan Fasting
Age	50,9 ± 8,8	
Male Sex, n, %	23, 57,5%	
Use of ACE-i, n, %	17, 42,5%	
Use of ARB, n, %	11, 22,5%	
Use of Ca-Blockers, n, %	23, 57,5%	
Use of Diuretics, n, %	20, 50%	
Use of Beta-Blockers,n	13, 32,5%	
Systolic Blood Pressure, mmHg	137,3 ± 15,0	129,4 ± 10,5 p:0,01
Diastolic Blood Pressure, mmHg	81,7 ± 8,8	78,6 ± 6,5 p:0,05
Heart Rate, beat/min.	77,4 ± 10,8	73,4 ± 7,0 p:0,01
Weight, kg	79,5 ± 12,7	78,1 ± 12,0 p:0,01
BMI	25,1 ± 4,0	24,6 ± 3,8 p:0,01
FMD, %	8,8 ± 3,7	10,8 ± 3,8 p:0,01

ACE-i: angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blocker, Ca-blockers: Calcium channel blockers, BMI: Body mass index, FMD: Flow mediated dilatation

OP-099 PROGNOSTIC VALUE OF FRONTAL PLANE QRS-T ANGLE IN PULMONARY HYPERTENSION: EXPERIENCE FROM A TERTIARY CENTER

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Objective: Frontal plane QRS-T (fQRS-T) angle is a marker of ventricular repolarization heterogeneity and increased fQRS-T angle is associated with arrhythmias, adverse events, and mortality. However, the prognostic role of the electrocardiogram (ECG) in pulmonary hypertension (PH) is not fully known. We aimed to investigate the association between fQRS-T angle and PH. The main goal was to demonstrate wider fQRS-T angle has an unfavorable effect on adverse events in PH patients.

Methods: This study included 191 PH patients between September 2009 and June 2020. Survivors and nonsurvivors were compared regarding baseline fQRS-T angle. fQRS-T angle was calculated as the absolute difference between QRS and T wave axis, and it was categorized as normal ($\leq 90^\circ$) or abnormal ($> 100^\circ$).

Results: Clinical assessment included basic demographics, World Health Organization Functional Class (WHO FC), N-terminal pro-BNP, 12-lead ECG, transthoracic echocardiography, right heart catheterization (RHC) and six-minute walk test (6MWT) has shown in Table-1. Overall mortality was 23.5%. Nonsurvivors had significantly wider fQRS-T angle compared to survivors ($56.3^\circ \pm 12.4^\circ$ vs. $33.9^\circ \pm 13.5^\circ$; $P < 0.001$). A wide QRS-T angle $\geq 100^\circ$ was present in 10 (5.2%) of the subjects and 8 of them were nonsurvivors. The fQRS-T angle $\geq 100^\circ$ was associated with an increased hazard for all-cause mortality (HR: 2.94; 95% CI: 1.53 - 5.68; $p = 0.001$). The Spearman correlation test showed a moderate positive correlation between the fQRS-T angle and mean pulmonary artery pressure (mPAP) at RHC (correlation coefficient $r = 0.75$, $p < 0.01$), WHO FC ($r = 0.65$, $p < 0.001$). Tricuspid annular plane systolic excursion (TAPSE) ($r = -0.45$, $p < 0.03$) and right ventricular systolic doppler velocity (RVSD) ($r = -0.51$, $p < 0.01$) showed a low negative correlation.

Conclusion: In the present study, wider fQRS-T angle is associated with poor prognosis in patients with PH. Therefore, fQRS-T angle may be useful in the baseline monitoring of the PH patients.

Keywords: pulmonary hypertension, frontal plane QRS-T angle, electrocardiogram

Table-1: Baseline demographics, right heart catheterization, electrocardiographic and echocardiographic data

	PH (n:191)
Demographic parameters	
Age, years	56 (44 - 76)
Gender, n; females (%)	26.2 \pm 3.8
BMI, kg/m ²	26.2 \pm 3.8
WHO FC, n (%)	
I	34 (18)
II	65 (34)
III	70 (37)
IV	22 (11)
Risk groups, n (%)	
low	70 (37)
intermediate	89 (46)
high	17 (17)
6MWT, m	344 \pm 166
Median duration of follow-up, years	3.7 \pm 2.5
NT pro- BNP (pg/ml)	430 (200 - 660)
Echocardiographic parameters	
Peak tricuspid regurgitation velocity, m/sec	3.4 (2.9-3.9)
Left ventricular Ejection fraction, %	61.3 \pm 9.5
Estimated systolic pulmonary artery pressure, mmHg	53 (40 - 66)
Tricuspid annular plane systolic excursion, mm	18.1 \pm 4.1
Right ventricular systolic doppler velocity, cm/ sec	11 (9.5 - 12.5)
Right atrium area, cm ²	18 (10 - 26)
Hemodynamic parameters	
mPAP, mmHg	49.5 \pm 15.1
PVR, Wood Units	9.7 (4.6-14.8)
PCWP, mmHg	10 (6.5 - 13.5)
Cardiac index, L/min/m ²	2.2 \pm 0.49
RAP, mmHg	10 (6-14)
Electrocardiographic parameters	
QRS angle, $^\circ$	49.5 \pm 15.1
T angle, $^\circ$	37 (26-58)
QRS-T angle, $^\circ$	41 \pm 13.8
QRS duration, msn	106 (65 - 135)
qR pattern in V1, n (%)	99 (52)
ST segment depression in V1-3, n (%)	103 (54)

BMI: Body mass index; WHO FC: World Health Organization Functional Class; 6MWD: 6 minutes walking distance; NT-Pro BNP: N-terminal Pro brain natriuretic peptide; mPAP: mean pulmonary arterial pressure; PVR: pulmonary vascular resistance; RVCPO: right ventricular cardiac power output; PCWP: pulmonary capillary wedge pressure; RAP: right atrial pressure.

OP-100 PULMONARY HYPERTENSION PATIENT WITH A MEAN PAP OF 23 MMHG BY RIGHT HEART CATHETERIZATION

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Objective: Pulmonary hypertension is defined as an increase in mean pulmonary arterial pressure (PAPm) ≥ 25 mmHg at rest as assessed by right heart catheterization. Available data have shown that the normal PAPm at rest is 14 ± 3 mmHg with an upper limit of normal of approximately 20 mmHg. The clinical significance of a PAPm between 21 and 24 mmHg is unclear.

Case: 60-year-old woman was admitted to our clinic with complaints of breathlessness increased recently. She had no history of cardiovascular disease except hypertension. Transthoracic echocardiography did not detect left ventricular systolic and diastolic dysfunction, or aortic and mitral valve pathologies. But right atrium and right ventricle enlargement, mildly depression of right ventricular systolic functions (TAPSE: 1.9cm), moderate-to-severe tricuspid regurgitation, tricuspid velocity 3.5 m/sec, estimated systolic pulmonary artery pressure 55mmHg, hepatic veins, main pulmonary artery, right and left pulmonary arteries dilatation and large secundum ASD was detected. The diameter of the defect was measured as 4.0 cm by transesophageal echocardiography and no significant shunt was observed. The PAPm was 23mmHg and the Qp/Qs ratio was 3.56 with right heart catheterization. Surgical closure of the ASD was decided at the cardiology-cardio-surgery council for the patient with minimal coronary artery disease in coronary angiography.

Conclusion: The diagnosis of pulmonary hypertension should not be excluded in patients whose PAPm is found to be between 21-24 mmHg by right heart catheterization, and additional findings suggesting pulmonary hypertension should be considered in echocardiographic examination.

Keywords: Pulmonary hypertension, mean pulmonary artery pressure, congenital heart disease

OP-101 THE EFFECT OF SUPERFICIAL FEMORAL ARTERY OCCLUSION ON PRIMARY PATENCY AND AMPUTATION FREE SURVIVAL AFTER ENDOVASCULAR MANAGEMENT OF INFRARENAL AORTA TOTAL OCCLUSIONS

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Objective: Endovascular therapy (EVT) has been increasingly utilized after the development of new techniques and technologies. EVT has displayed durable early and mid-term outcomes for infrarenal aorta occlusions (IAO). It is documented that baseline SFA occlusion increases hazard of primary aortofemoral bypasses (AFB) failure and concomitant SFA revascularisation did not improve AFB durability. Nonetheless, little is known regarding the role of SFA occlusion on long-term outcomes of IAO managed by endovascular techniques.

Methods: We retrospectively analyzed a single center database of 55 consecutive patients (age, 58.8 ± 6.97 years; 67.2% male; 42% critical limb ischaemia) undergoing EVT for IAO between January 2011 and March 2019. The outcome measures were primary patency rate and amputation free survival calculated by the Kaplan–Meier method. Independent predictors of restenosis were assessed by Cox proportional hazard regression model.

Results: Technical success was achieved in 49 patients (89.1%). In total, 190 stents (65 self-expandable stents, 60 balloon-expandable stents) were implanted. 24 (%43.6) patients had SFA occlusion. During the follow-up of 34.5 ± 28 months, loss of patency was detected in 7 patients. Primary patency rates were 96%, 82%, and 75% at 1, 3, and 5 years, respectively. Amputation free survival rates were 100%, 90%, and 82% at 1, 3, and 5 years, respectively.

Conclusion: In this study, five year outcomes of primary patency and amputation free survival for EVT of infrarenal aorta total occlusive lesions were favorable. Neither primary patency nor restenosis rate was associated with the presence of SFA occlusion. Also, none of the demographic, lesion and device factors were independently associated with loss of patency.

Keywords: endovascular therapy, aortoiliac occlusive disease, infrarenal aorta occlusion, primary patency rate, amputation free survival

Figure 1

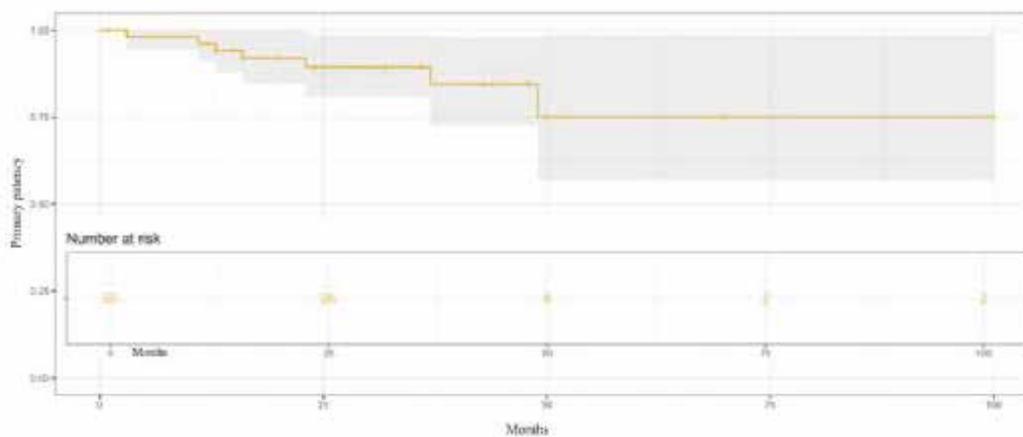
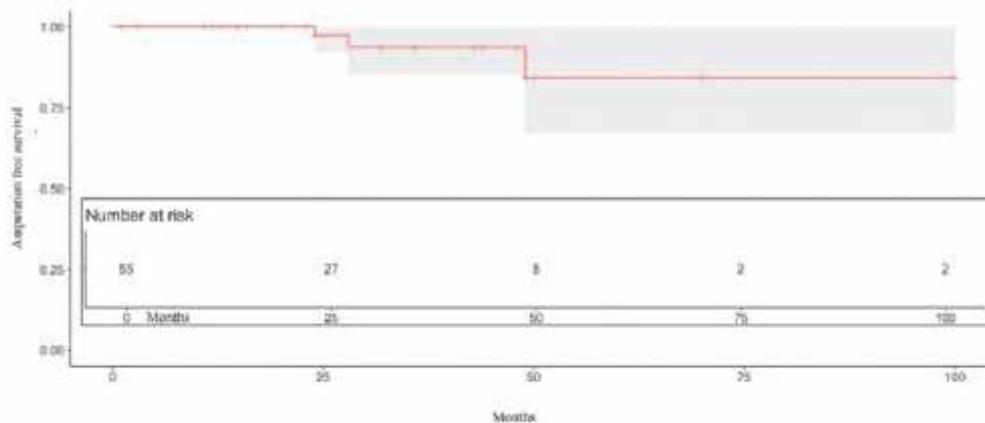


Figure 2



OP-102 THE LINK BETWEEN VENTRICULAR REPOLARIZATION VARIABLES AND CAROTID ARTERY STENTING

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Background: The spatial dispersion (QT prolongation) of repolarization are in part associated with carotid intima media thickness, which is an established risk marker of subclinical atherosclerosis. To evaluate the relationship between cardiac repolarization variables (QT, Tp-Te interval and Tp-Te/QT ratio) and carotid artery stenting indices of ventricular arrhythmogenesis were investigated.

Methods: Patients (24 male, 10 female) that are suitable for carotid artery stenting, without uncontrolled hypertension, severe coronary artery or valvular heart disease, were enrolled to the study. The QT, Tp-Te interval and Tp-Te/QT ratio were measured by 12-lead electrocardiogram and corrected for heart rate at the beginning, and at the 24 hour of the procedure.

Results: There were significant changes in Tp-Te interval (75.3 ± 7.8 vs 71.3 ± 10.5 , $p=0.013$), cTp-Te interval (82.4 ± 8.2 vs 76.9 ± 9.7 , $p=0.014$), Tp-Te/QT (0.20 ± 0.02 vs 0.18 ± 0.03 , $p=0.002$) and Tp-Te/QTc ratio (0.19 ± 0.02 vs 0.17 ± 0.02 , $P<0.001$) after the procedure. But spatial dispersion (QT) of repolarization parameters did not show any significant changes after the procedure.

Conclusions: These data suggested that transmural dispersion of repolarization shortened but spatial dispersion of repolarization did not change after the carotid artery stenting. These changes may decrease the risk of development of ventricular arrhythmia in carotid artery disease.

Keywords: Carotid artery disease, carotid artery stenting, ventricular arrhythmia, Tp-Te/QTc ratio

Table 1

	Prior Carotid Artery Stenting	After Carotid Artery Stenting	P value
RR distance (ms)	826.3 ± 120.7	831.8 ± 200.0	0.215
PR distance (ms)	159.2±18.3	160.4±22.1	0.820
QTmax (ms)	386.4±25.0	390.6 ±26.1	0.604
cQTmax(ms)	417.2 ±24.3	419.3 ±25.1	0.738
QTmin (ms)	366.6±24.3	372.4±27.2	0.412
cQTmin (ms)	397.4 ±23.7	404.1 ±25.9	0.224
QTd (ms)	23.6±6.7	23.2±6.9	0.957
cQTd (ms)	26.5±7.1	26.1±6.8	0.813
Tp-Te (ms)	75.3±7.8	71.3±10.5	0.013
cTp-Te (ms)	82.4 ±8.2	76.9 ±9.7	0.014
Tp-Te/QT ratio	0.20± 0.02	0.18± 0.03	0.002
Tp-Te/QTc ratio	0.19± 0.02	0.17 ± 0.02	<0.001

Electrocardiographic findings of the study population.

OP-103 RELATIONSHIP BETWEEN ATHEROSCLEROTIC RISK FACTORS AND VERTEBRAL ARTERY FLOW IN ASYMPTOMATIC ADULTS

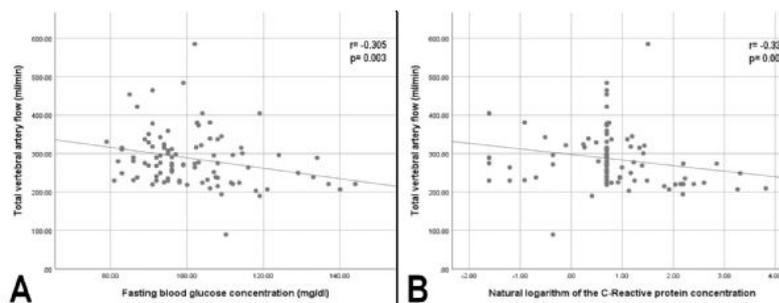
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OBJECTIVE: Vertebral arteries supply blood flow to the posterior cerebral circulation, and a reduction or interruption of this flow causes posterior cerebral syndrome (PCS) that is characterized with dizziness, unilateral limb weakness, dysarthria, headache, nausea and vomiting. The most common cause of PCS is atherosclerotic obstruction of VA, but the relationship between risk factors of atherosclerosis and VA obstruction remains incompletely understood. Ultrasonographic examination of the VA is the most common technique used to detect VA stenosis, but not all segments are directly visible with ultrasonography. As a result, VA flow is used as an indirect marker of VA obstruction. In the present study, we sought to understand the relationship between risk factors of atherosclerosis and VA flow in asymptomatic individuals undergoing a routine check-up examination. **METHODS:** Records for a total of 567 individuals undergoing a check-up examination were examined. 96 out of these 567 records had VA flows available. Demographic, clinical and laboratory records of these cases were retrieved from the institutional electronic medical database. Cases were divided into tertiles for analysis. **RESULTS:** Patients within the lowest VA flow tertile (total flow <239 ml/min) were older, had a significantly higher incidence of glucose intolerance or diabetes, and had a higher fasting blood glucose concentration, HbA1c% and C-reactive protein (CRP) (Table 1). While not statistically significant, there was a trend towards higher LDL-cholesterol in the lowest tertile. Age (OR: 1.04, 95%CI 1.01 – 1.07, p=0.016), presence of diabetes/glucose intolerance (OR: 4.62, 95%CI: 1.40 – 15.25, p=0.012), fasting glucose concentration (OR: 1.05, 95%CI: 1.01 – 1.08, p=0.006) and CRP (OR: 1.27, 95%CI: 1.08-1.51, p=0.005) were univariate predictors of a low VA flow. On multivariate analysis, only fasting glucose concentration (OR: 1.04, 95%CI: 1.00 – 1.08, p=0.038) and CRP (OR: 1.23, 95%CI:1.04 – 1.44, p=0.014) were independent predictors of a low VA flow. Both variables showed a statistically significant correlation with VA flow (Figure 1). **CONCLUSIONS:** Traditional risk factors are associated with the VA flow, with inflammation and abnormal glucose metabolism being the most important ones.

Keywords: vertebral artery, atherosclerosis, risk factor

Figure 1



Scatter plots depicting the association between total vertebral artery flow, fasting blood glucose (A) and C-reactive protein (B). In panel B, log-transformed values of C-reactive protein are given.

Table 1

Characteristic	1st Tertile (n=32)	2nd Tertile (n=32)	3rd Tertile (n=32)	P value
Age (years)	54 ± 16*	48 ± 12	44 ± 14	0.03
Gender (%female)	43.8%	40.6%	34.4%	0.74
BMI (kg/m ²)	29.9 ± 4.5	27.9 ± 3.6	28.5 ± 3.9	0.50
Systolic Blood Pressure (mmHg)	121.9 ± 24.4	127.3 ± 19.4	125.9 ± 14.5	0.88
Diastolic Blood Pressure (mmHg)	76.6 ± 9.8	76.8 ± 11.8	75.4 ± 8.6	0.65
Heart Rate (bpm)	74 ± 12	73 ± 6	70 ± 9	0.34
Smoking (%)	38.5%	52.2%	39.1%	0.56
Alcohol (%)	17.2%	50.0%*	18.2%	0.02
Family History of Coronary Artery Disease (%)	51.7%	77.3%*	36.0%	0.02
Hypertension (%)	21.9%	6.3%	12.5%	0.18
Glucose Intolerance / Diabetes (%)	28.1%*	3.1%	12.5%	0.02
Glucose (mg/dl)	107.2 ± 18.9*	100.3 ± 12.4	95.3 ± 9.2	0.04
HbA1c (%)	6.03 ± 0.60**	5.8 ± 1.4	5.7 ± 0.6	0.004
Creatinine (mg/dl)	0.86 ± 0.16	0.87 ± 0.26	0.81 ± 0.09	0.37
Hemoglobin (mg/dl)	14.3 ± 1.9	14.3 ± 1.3	14.1 ± 1.4	0.81
C-reactive protein (mg/dl)	2.60 (2.00 – 8.50)*	2.00 (2.00 – 2.00)	2.00 (1.83- 2.33)	0.02
Total cholesterol (mg/dl)	213.9 ± 41.0	213.9 ± 52.1	201.9 ± 45.7	0.30
Triglycerides (mg/dl)	153.7 ± 80.9	136.2 ± 82.0	164.3 ± 122.7	0.60
HDL-cholesterol (mg/dl)	50.8 ± 14.1	51.6 ± 15.1	47.4 ± 13.8	0.47
Non-HDL cholesterol (mg/dl)	163.1 ± 40.0	162.3 ± 47.0	154.5 ± 46.3	0.42
LDL-cholesterol (mg/dl)	136.4 ± 32.4	129.7 ± 41.1	122.5 ± 29.2	0.08

Demographic, clinical and laboratory characteristics of patients within the vertebral artery flow tertiles. BMI, body mass index; HbA1c%, hemoglobin A1c%, HDL, high-density lipoprotein; LDL, low-density lipoprotein. * p<0.05 ** p<0.01

OP-104 APPROACH TO THE CASE OF RENAL ARTERY PERFORATION OCCURRED DURING THE STENTING PROCEDURE PERFORMED IN THE RENAL ARTERY STENOSIS

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Introduction: Renal artery stenosis is one of the important causes of secondary hypertension. While the most common cause of renal artery stenosis is atherosclerosis in the community, fibromuscular dysplasia (FMD) is the most common cause in young adults. In this case, we present a case who developed perforation in the renal artery after post-dilatation during percutaneous transluminal renal angioplasty.

Case: A 23-years-old female patient was admitted to our clinic due to headache and increased blood pressure follow-up for the last few months. Renal angiography was recommended to the patient. A 6F sheath was placed in the right femoral artery of the patient. Bilateral renal arteries were monitored with the JR4 guiding catheter. The left renal artery was observed to be normal. In the mid-region of the right renal artery, 98% focal stenosis was observed (Figure 1). In the same session, the lesion in the right renal artery was passed with a 0.014" guidewire. First, a 2.0x15 mm balloon was performed, then a 4.0x28 mm drug-coated (Everolimus) stent was implanted with 16 atm pressure. Then a 5.0x9 mm noncompliant balloon post dilatation was performed. After performed post-dilatation, contrast medium extravasation was observed in the middle and distal of the stent, and the patient complained of severe abdominal pain simultaneously (Figure 2). Immediately, it was inflated 3 times at 3-minute intervals with a 5.0x9 mm non-compliant balloon and waited. No perforation was observed in the control renal angiography and the stent was observed patent. The procedure of the patient, whose hemodynamics was stable, was ended.

Discussion and Conclusion: Percutaneous transluminal renal angioplasty has become the preferred treatment for renovascular hypertension caused by FMD. Albeit rare, complications can occur during percutaneous transluminal renal angioplasty. Mainly, renal artery dissection, renal artery perforation, atheroma embolism, cholesterol embolism and other complications can be seen. The frequency of iatrogenic perforation has been increasing in recent years due to the increasing number of endovascular treatments. Renal artery perforation is a rare complication with a frequency of 2-6%. Fast and effective patient management is critical in renal artery perforation. Considering the high morbidity of vascular repair with emergency surgical intervention, the use of covered (balloon and self expandable) stents to repair vascular injuries has recently gained popularity. In addition to different types of stents, various embolization materials are available. The packing application with balloons and then the use of covered stents is usually sufficient in the treatment.

A multidisciplinary team approach should be adopted, since there will be an urgent need for surgery in cases that fail with percutaneous intervention. It is required not only to decide the best interventional modality, but also to manage potential iatrogenic vascular complications.

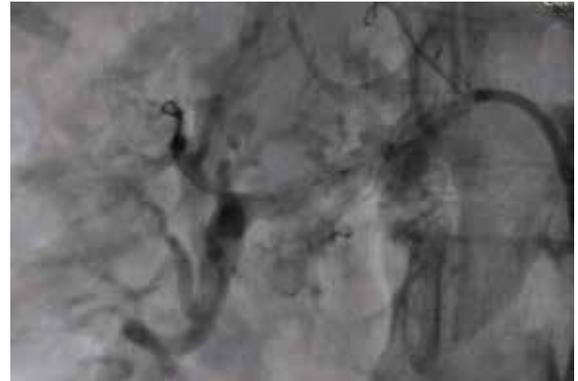
Keywords: Renal artery stenosis, Renal artery perforation, Complications of renal angioplasty

Figure 1



98% focal stenosis is observed in the mid-region of the right renal artery

Figure 2



Renal artery perforation and extravasation of contrast medium to the perirenal area after post-dilatation with non-compliant balloon

OP-105 THE EFFECT OF SUCCESSFUL ENDOVASCULAR REVASCLARIZATION ON AORTIC AUGMENTATION INDEX IN PERIPHERAL ARTERY PATIENTS

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Aim: The aortic augmentation index (Alx), a marker of arterial stiffness, and peripheral arterial disease (PAD) are associated with an increased cardiovascular risk. Previous studies have shown that Alx elevated in peripheral arterial diseases. In our study, we investigated the impact of successful percutaneous revascularization on Alx.

Methods: 50 patients which admitted to the cardiology department with a diagnosis of peripheral artery disease and recruited for revascularization were included in the study. 13 patients of them were excluded from study for various reasons {6 patients were amputated during the first month of treatment; Atrial fibrillation developed in 2 patients; Kidney failure requiring dialysis developed in 1 patient after the procedure; 3 patients were revascularized; 1 patient died} Our study was completed with 37 patients (32 males, 5 females). Alx values of 37 patients were evaluated on the first day and first month of revascularization.

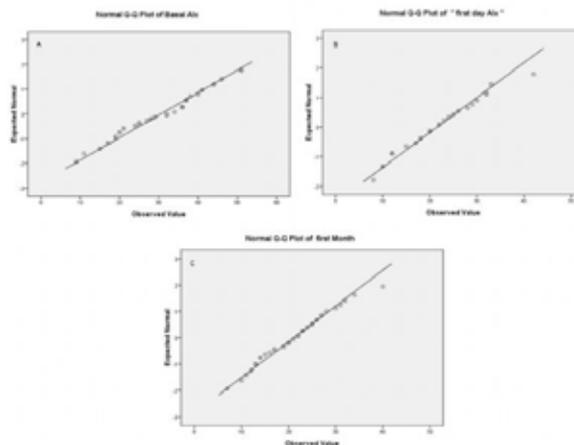
Results: For assessing before then after of the data, paired-samples t-test test applied to “Basal Alx %” and “first day Alx” The values of the variable significantly changed. It was then seen to be significantly decreased. Basal Alx %: Mean: 30.88. (min: 11.0). (max: 51.0). “first day Alx”: Mean: 21.54. (min: 8.0). (max: 42.0). P value: < 0.001.

For assessing before then after of the data, paired-samples t-test test applied to “Basal Alx %” and “first month Alx” The values of the variable significantly changed. It was then seen to be significantly decreased. “Basal Alx %”: Mean: 30.59. (min: 9.0). (max: 51.0). “first month Alx”: Mean: 21.14. (min: 7.0). (max: 40.0). P value: < 0.001.

Conclusion: Our study showed that successful revascularization of peripheral artery disease was significantly reduced the aortic augmentation index (Alx).

Keywords: Endovascular therapy, Peripheral Artery Disease, Augmentation Index

analiz numeric data distribution



The Kurtosis statistic value is found as -1.39. Expected normal distribution range: (-1.5 - 1.5). Normally distributed.

assessing before then after of the data

Paired Samples Statistics				
	Mean	%	Std. Deviation	Std. Error Mean
Pair 1 "Basal Alx"	30,88	26	16,420	3,287
"First day Alx"	21,54	26	6,477	1,262
Pair 2 "Basal Alx"	30,59	37	10,963	1,806
"First month Alx"	21,14	37	7,363	1,208

Paired Samples Correlations				
Pair	Variables	%	Correlation	Sig.
Pair 1	"Basal Alx" & "First day Alx"	26	,276	,000
Pair 2	"Basal Alx" & "First month Alx"	37	,214	,000

Paired Samples Test									
Pair	Variables	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference		t	df	Sig. (2-tailed)
					Lower Bound	Upper Bound			
Pair 1	"Basal Alx" - "First day Alx"	9,346	12,947	1,520	6,307	12,485	6,112	25	,000
Pair 2	"Basal Alx" - "First month Alx"	8,450	8,598	1,086	7,266	11,645	8,760	36	,000

For assessing before then after of the data, paired-samples t-test test applied to “Basal Alx & First day Alx” and “Basal Alx & First month Alx”

OP-107 ORAL ANTICOAGULANTS IN EARLY DIAGNOSIS OF CANCER

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Our main goal in cancer prevention is to prevent cancer development with primary prevention methods. Secondary goal is to be able to make an early diagnosis with screening tests. Most of the patients can only be diagnosed during their symptomatic periods. Unusual bleeding may occur in the early or late period of many cancers. Anticoagulant treatment is used for the prevention and treatment of thromboembolism in different clinical situations. The biggest handicap of these drugs is bleeding. The risk increases in elderly patients with comorbidities and multiple drug use. Sometimes bleeding can provide a diagnosis opportunity to clinicians as in our patients. Case 1: A 79-year-old female patient was diagnosed as non-valvular AF. Apixaban 5mg 2x1 was started. The patient was admitted to the clinic with minimal bleeding from the bottom. Endo-colonoscopy was planned by the Gastroenterology Department. The patient's advanced examinations were completed, and the patient's pathology was identified as intramucosal carcinoma (TONO). Case 2: A 70-year-old woman was diagnosed with non-valvular AF. Apixaban 5mg 2x1 was started. She was admitted with vaginal bleeding while under treatment. Trans-abdominal USG performed by the Department of Obstetrics and Gynecology and a 45*43mm solid mass filling the endometrium was revealed. The patient underwent hysterectomy and bilateral salpingo-oophorectomy. The pathology result came as endometrial polyp and adenomyosis. Case 3: A 71-year-old male had mechanical prosthetic mitral valve surgery in 2004 and was followed up under warfarin treatment. He had also an operation anamnesis due to bladder cancer and he had been on cure for 7 years. The patient came with a complaint of hematuria. In the therapeutic range of INR 2.6 was detected. The patient was evaluated by the Urology Department. In his cystoscopy examination revealed two lesions 1 cm in size at right lateral wall of the bladder. Urothelial papillary neoplasia was detected in the biopsy and TUR was planned. The bleeding is a common problem in cancer patients related to local invasion of the tumor, tumor angiogenesis, systemic effects of cancer. Bleeding in patients may be life-threatening and may occur as bruising, petechiae, epistaxis, hemoptysis, hematemesis, hematochezia, melena, hematuria, or vaginal bleeding. This problem is exacerbated by the use of anticoagulant drugs. Gastrointestinal system cancers, metastatic cancers, BMI ≥ 40 , creatine clearance < 30 mL/min, presence of thrombocytopenia have been identified as additional factors increasing the risk of bleeding. Bleeding can be a life-threatening complication that is difficult to manage, as well as an alert symptom of early diagnosis of cancer patients in the asymptomatic period. Our patients had no additional symptoms other than bleeding to suggest a diagnosis of cancer. The bleeding complication that accelerated with OAC enabled early diagnosis in our patients. They received their diagnoses at the premalignant or early stage of cancer.

Keywords: cancer, bleeding, anticoagulants

OP-108 ALWAYS WONDERED BUT NOT KNOWN: WHAT IS THE BEST TIME TO TAKE WARFARIN FOR PATIENTS WITH METALLIC PROSTHETIC VALVES?

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INTRODUCTION AND OBJECTIVE: In order to provide anticoagulation in patients with metallic prosthetic valves, the only drug treatment we have is warfarin, a vitamin K antagonist. Blood drug levels in warfarin treatment vary depending on the person's metabolism rate, nutritional habits, and other medications used. Since there is no clear information about the timing of the drug in daily practical cardiology practice, there are empirical applications varying from doctor to doctor (such as after breakfast, after dinner).

Our aim in this study is to evaluate the effect of medication timing on anticoagulation stability in users of warfarin stability in patients with with metallic prosthetic valves.

Material-Method: Sixty patients with prosthetic heart valves, on warfarin therapy for at least 6 months, and stable INR values were included in the study. In our prospective study, patient recruitment and follow-up was completed in Bursa Yüksek İhtisas Training and Research Hospital Cardiology Department. All patients were instructed to first take warfarin for a month between 19.30 and 20.00. During this period, INR values were measured twice for a month, once every 15 days. Later, the warfarin intake time of the same patient group was arranged in the range of 09.30-10.00 and similarly, blood INR levels were evaluated twice with 15 days intervals from the patients. Considering the INR values in these samples, TTR evening (therapeutic range) and TTR morning values were calculated with the Rosendaal method.

Results: 60 patients with metallic valves were included in the study. 38 of these patients were female (63,3%) and 22 were male (36,7%). 35 of the 60 patients were followed up with MVR (58,3%), 15 with AVR (25%) and 10 with AVR + MVR (16,7%). When TTR values of patients with metallic heart valves were compared according to morning and evening warfarin use, no significant difference was found in TTR values (%66,23 ± 40,7 vs %64,12 ± 41,13, respectively, p=0.783).

However, considering the mean INR values, the mean INR value in morning use was found to be significantly higher compared the evening use (3,06 ± 0,47 vs 2,73 ± 0,53, respectively, p=0.001).

Conclusion: Although there was no significant change in the number of tablets used weekly between morning and evening use of warfarin, a significant increase was found in INR values in those who used warfarin in the morning. While there was a significant increase in the morning INR values of the patients, this significance was not reflected in the TTR values. An important reason for this may be the serious limitations of the Rosendaal method in evaluating the stability of INR in patients using warfarin due to the prosthetic valve.

In the light of the results of this study, we suggest that warfarin intake in the morning can be considered especially in patients with prosthetic valve and in the group of patients who have difficulty in achieving the target INR value.

Keywords: Warfarin, Metallic prosthetic valve, INR, Therapeutic range, Chronotherapy

Comparison of INR and TTR Values in Morning and Evening Dosing

	Evening Dosing	Morning Dosing	Evening Dosing vs Morning Dosing (p value)
INR (mean ± SD)	2,73 ± 0,53	3,06 ± 0,4	<0.001
TTR (mean ± SD)	64,12 ± 41	66,23 ± 40	0.783
Number of Tablets per Week (mean ± SD)	6,39 ± 2,3	6,43 ± 2,46	0.070

OP-109 BIOLOGICAL HEART VALVES VERSUS MECHANICAL HEART VALVES WITH EFFECTIVE ANTICOAGULATION WITH LOW-DOSE WARFARIN DURING PREGNANCY

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Background: Prosthetic heart valves (PHVs), especially mechanical ones, are highly thrombogenic, and pregnancy-related procoagulant status increases the risk of PHV thrombosis. Current evidence reports that pregnant women with effective INR under low dose warfarin may have optimal maternal and fetal outcomes. While current European guidelines advise to consider implantation of a biological PHV in women with a pregnancy wish, the underlying evidence is limited. The high deterioration rate of biological PHV at young age poses the woman at risk of going through pregnancy with a stenotic or regurgitant PHV. The goal of this study was to evaluate biological heart valves versus mechanical heart valves with effective anticoagulation with low-dose warfarin during pregnancy.

Methods: The outcomes of anticoagulation regimens were assessed retrospectively in pregnant women (71 women; 71 pregnancies) with PHVs. The study population was divided into two groups according to type of valve; mechanical heart valve with low-dose warfarin (≤ 5 mg/d) throughout pregnancy (group 1), biological heart valve prosthesis during pregnancy (group 2). Fetal complications included preterm birth, low birth weight, stillbirth, abortion, and congenital anomaly. Besides, maternal outcomes included mortality, valve thrombosis requiring therapy, major bleeding, preeclampsia, stroke, and endocarditis.

Results: Of the 71 pregnancies, 29 were included in group 1 and 42 were included in group 2. In terms of basic demographic and echocardiographic data, there was no significant difference except valve degeneration. PHV degeneration was statistically significantly higher in group 2 (0 vs. 52.4%, $p < 0.001$). While vaginal delivery was higher in group 2 (3.4% vs. 21.4%, $p = 0.032$), C/S delivery was statistically significantly higher in group 1 (82.6% vs. 59.5%, $p = 0.037$). There was no difference between the two groups in terms of poor fetal outcomes, including preterm birth, low birth weight, abortion, and congenital anomaly. There was no statistical difference between the two groups in terms of major bleeding, endocarditis, valve thrombosis requiring therapy, preeclampsia, stroke, maternal mortality.

Conclusion: The current data suggest that pregnant patients with mechanical PHVs on a low-dose warfarin (≤ 5 mg/day) regimen with therapeutic international normalized ratio levels have maternal-fetal outcomes that are as acceptable as biological valve pregnancy outcomes.

Key words: Prosthetic heart valves, pregnancy, prosthetic valve thrombosis, warfarin

Table 1: Baseline characteristics of pregnancies in patients with prosthetic heart valves

	Mechanical valve (N=29)	Bioprosthetic valve (N=42)	P value
Age (years)	29.1 \pm 4.6	27.9 \pm 5	0.290
ETSVS (months)	62.6 \pm 37	70.9 \pm 57.6	0.495
BMI (kg/m ²)	25.1 \pm 2.6	25.6 \pm 1.1	0.484
Diabetes Mellitus, n (%)	1 (3.4)	2(4.8)	0.787
Hypertension, n (%)	1 (3.4)	2(4.8)	0.787
Hyperthyroidism, n (%)	1 (3.4)	1(2.4)	0.789
Hypothyroidism, n (%)	3(10.3)	1(2.4)	0.153
Chronic anemia, n (%)	1(3.4)	4(9.5)	0.325
Smoking, n (%)	1 (3.4)	1 (2.4)	0.789
Heart failure, n (%)	2(6.9)	0	0.084
Atrial fibrillation, n (%)	5 (17.2)	8 (19)	0.847
Valve Position, n (%)			
AVR	6 (20.7)	6(14.3)	0.479
MVR	14(48.3)	22(52.4)	0.734
TVR	2 (6.9)	1(2.4)	0.353
PVR	0	5(11.6)	0.054
AVR+MVR	7 (24.1)	8(19)	0.606
Valve degeneration, n (%)	0	22 (52.4)	<0.001
Previous history of PVT, n (%)	3 (10.3)	1 (2.4)	0.153
Paravalvular leak, n (%)	2(6.9)	2 (4.8)	0.701
Underlying heart disease			
Rheumatic valve disease	21 (72.4)	31 (73.8)	0.896
ToF	0	4(9.5)	0.087
Pulmonary stenosis	0	1(2.4)	0.403
Marfan	1 (3.4)	4(9.5)	0.325
Other	7 (24.1)	2 (4.8)	0.016
Drugs, n (%)			
ASA 100 mg	5 (17.2)	16 (38.1)	0.058
B bloker	7 (24.1)	8 (19)	0.606

Table 2: Outcomes of pregnancies in the whole series

Variable	Mechanical valve (N=29)	Bioprosthetic valve (N=42)	P value
Termination time of pregnancy (weeks)	37.3±2.3	37.5±1.7	0.704
Vaginal delivery, n (%)	1 (3.4)	9 (21.4)	0.032
Caesarian section, n (%)	24 (82.6)	25 (59.5)	0.037
Healthy neonates, n (%)	26 (89.7)	36 (85.7)	0.624
Preterm birth, n(%)	3 (10.3)	6 (14.3)	0.624
Low birth weight, n(%)	3 (10.3)	3 (7.1)	0.634
Abortion, n (%)	5 (17.2)	7 (16.7)	0.949
Stillbirth, n (%)	0	0	-
Congenital fetal abnormalities, n (%)			
-Intrauterine growth retardation	1 (3.4)	0	0.226
-Bicuspid aortic valve	0	1 (2.4)	0.403
-Nasal hipoplasi	1 (3.4)	0	0.226

Table 3: Comparison of fetomaternal complications and pregnancy outcomes between bioprosthetic valve and mechanical prosthetic valve with low dose warfarin

Variables	Mechanical valve (N=29)	Bioprosthetic valve (N=42)	P value
Major bleeding, n (%)	1 (3.4)	2 (4.8)	0.787
Endocarditis, n (%)	1 (3.4)	0	0.226
PVT requiring therapy, n (%)	1 (3.4)	2 (4.8)	0.787
Preeclampsia, n (%)	0	1(2.4)	0.403
Stroke, n (%)	0	1(2.4)	0.403
Maternal mortality, n (%)	0	1(2.4)	0.403

PVT: Prosthetic valve thrombosis;

OP-110 CHANGES OF NUTRITION AND LIFESTYLE HABITS AMONG CARDIOVASCULAR DRUG USERS: A CVSCORE-TR SUB-STUDY

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Introduction: Cardiovascular diseases are the leading causes of death all over world. Despite major advances in invasive treatment, drugs are the cornerstone of treatment for cardiovascular diseases. Acceptance of having cardiovascular disease constitutes the first step of treatment. According to this perspective, drug usage indicates that the disease is accepted by the patient. In this study, we want to investigate lifestyle behavioral differences of the cardiovascular drug users.

Material and Method: CVSCORE-TR study database was used for this study. The study comprises 4209 patients from 33 medical centers. Participants aged ≥ 40 years who agreed to participate to the project were included. Their demographic data, systemic diseases, habitual status, drug usage, perception of cardiovascular risk, Systematic Coronary Risk Evaluation (SCORE) scores and basic blood parameters were noted. Participants were grouped according to cardiovascular drug usage. Categorical variables were compared using Chi-Square test. A binary logistic regression was constructed to determine patient's habitual status and cardiovascular risk estimation related with cardiovascular drug usage.

Results: Out of 4209 patients, 2359 (56%) were male, mean age was 59.29 ± 10.7 , mean body mass index (BMI) was 28.75 ± 4.61 and mean SCORE-TR percentage was 5.46 ± 7.19 . Table 1 shows compare of the basic demographic and clinical data according to study groups. Table 2 shows binary logistic regression analyses results related with study groups. Smoking status and salt usage are negatively correlated and statistically significance with drug usage ($p < 0.001$ and $p = 0.006$). In contrast, there were no statistically significance and correlation between physical activity, diet restriction, exercise and CVD drug usage ($p = 0.448$, $p = 0.924$ and $p = 0.309$).

Discussion: Drug usage positively affects patients' view of smoking cessation, more frequently blood pressure monitoring and restriction of salt usage. However, drug users do not give enough attention for exercise and diet. As a result, it should be emphasized that in addition to the drug usage, patients should also pay more attention to diet and exercise.

Table 1. Comparison of demographic characteristics, lifestyle behaviors and cardiovascular risk for participants according to CVD drug usage.

Parameter	At least CVD Drug usage		p
	Absent	Present	
Gender/Male (n, %)	329 (13.9)	2030 (86.1)	<0.001
Age (y)			
BMI (kg/m ²)			
Presence of obesity (n, %)	207 (14.1)	1260 (85.9)	<0.001
Presence of smoking (n, %)	202 (22.5)	695 (77.5)	<0.001
Presence of physical activity (n, %)	151 (15.9)	800 (84.1)	0.319
Presence of diet (n, %)	167 (16.3)	855 (83.7)	0.549
Presence of physical activity for obesity (n, %)	129 (18.7)	562 (81.3)	0.188
Presence of reduction of food amount for obesity (n, %)	52 (16.9)	256 (83.1)	0.973
Presence of additional salt use (n, %)	375 (20.8)	1431 (79.2)	<0.001
Frequency of BP measurement (n, %)			
Only at office	455 (24.8)	1380 (75.2)	<0.001
Monthly	124 (16)	653 (84)	
Weekly	43 (8)	492 (92)	
Two-three times a week	40 (6.9)	538 (93.1)	
Daily	50 (10.4)	433 (89.6)	

Frequency of fast food consumption (n, %)			
None	441 (15.3)	2439 (84.7)	<0.001
One-two times a month	181 (22.3)	631 (77.7)	
Weekly	69 (20.5)	268 (79.5)	
More than once a week	22 (12.2)	158 (87.8)	
Perceived CVD risk (n, %)			
Low	382 (26.8)	1045 (73.2)	<0.001
Mediocre	260 (14.2)	1577 (85.8)	
High	71 (7.5)	874 (92.5)	
Actual CVD risk			
Low	322 (50.4)	317 (49.6)	<0.001
Mediocre	150 (28.4)	379 (71.6)	
High	241 (7.9)	2800 (92.1)	

Table 2. Independent predictors of drug usage in binary logistic regression analyses that included relevant lifestyle behaviours of participants.

Parameter	O.R	95% CI	p
Smoking	0.64	0.518-0.791	<0.001
Obesity	1.22	0.999-1.491	0.052
BP measure frequency	1.34	1.242-1.446	<0.001
Pyhsical activity	0.909	0.711-1.163	0.448
Diet for obesity	1.038	0.833-1.294	0.739
Exercise for obesity	0.872	0.669-1.136	0.309
Low food consumption for obesity	1.017	0.714-1.449	0.924
Salt usage	0.768	0.636-0.927	0.006
Frequency of fast food	1.028	0.919-1.151	0.629

OP-111 INVESTIGATION OF CHANGES IN SUPERFICIAL ECG OF COMBINATIONS CONTAINING HYDROXYCHLOROQUINE USED IN THE TREATMENT OF COVID-19

Serdal Baştuğ

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Objective: The aim of this study is to examine the electrocardiographic effects of hydroxychloroquine in combination therapies used in the treatment of COVID-19 and to reveal which combination therapy has the greater arrhythmogenic effect.

Design: This cohort study include 684 patients aged 18 years and older who underwent SARS CoV -2 testing and baseline ECGs at Ankara City Hospital. The 12-lead superficial ECGs of the patients taken before the drug treatment, on the 3rd and 5th days after the drug treatment is started will be evaluated. ECGs were manually analyzed and the QTc intervals were serially evaluated with a standardized protocol by the standard tangent method and excluding U waves, preferably using lead II or V1 and corrected using Bazett formula. Within the scope of the study, PR interval, QRS duration, QT duration, corrected QT duration (QTc), QT dispersion will be examined in superficial electrocardiography. We calculated and compared proportions of patients who had QTc prolongation (QTc prolongation >500 ms). Baseline and follow-up QTc intervals were compared using the paired samples t-test.

Results: A total of 684 patients had a baseline ECG and on the 3rd and 5th days after the drug treatment ECGs was recorded. Hydroxychloroquine and combination therapies (Hydroxychloroquine + Azithromycin, Hydroxychloroquine + Levofloxacin, Hydroxychloroquine + Tetradoxine) resulted in a mean QTc prolongation of 23 ms. 17 patients (%2.49) had a QTc interval exceedin 500 ms during Hydroxychloroquine and combination treatment. No torsade de pointes or ventricular arrhythmia was observed.

Conclusion: Hydroxychloroquine and combination therapies prolongs the QTc interval however the clinical reflection of this situation is meaningless. ECG monitoring and QTc calculation in patients using hydroxychloroquine and its combinations, if QTc >500, it is appropriate to interrupt the treatment.

Keywords: Covid-19, hydroxychloroquine, QTc

Demographics and baseline characteristics of patients with COVID-19

Variables	Study population (n=684)
Age, years	62 (18-81)
men	399 (58.3)
women	285 (41.7)
Hypertension	213 (31.1)
Diabetes Mellitus	57 (8.3)
Coronary Artery Disease	42 (6.1)
Cerebrovascular disease	11 (1.6)
Lenght of hospital stay, days	6 (4-9)
Prolonged QT interval, n (%)	17 (2.5)
QT interval, msn	
Basal	418
After treatment	441

Results table will be added

OP-112 RESOLUTION OF COVID-19 INDUCED THROMBUS VIA ABSIKSIMAB IN A 33 YEARS OLD WOMAN PRESENTING WITH NSTEMI

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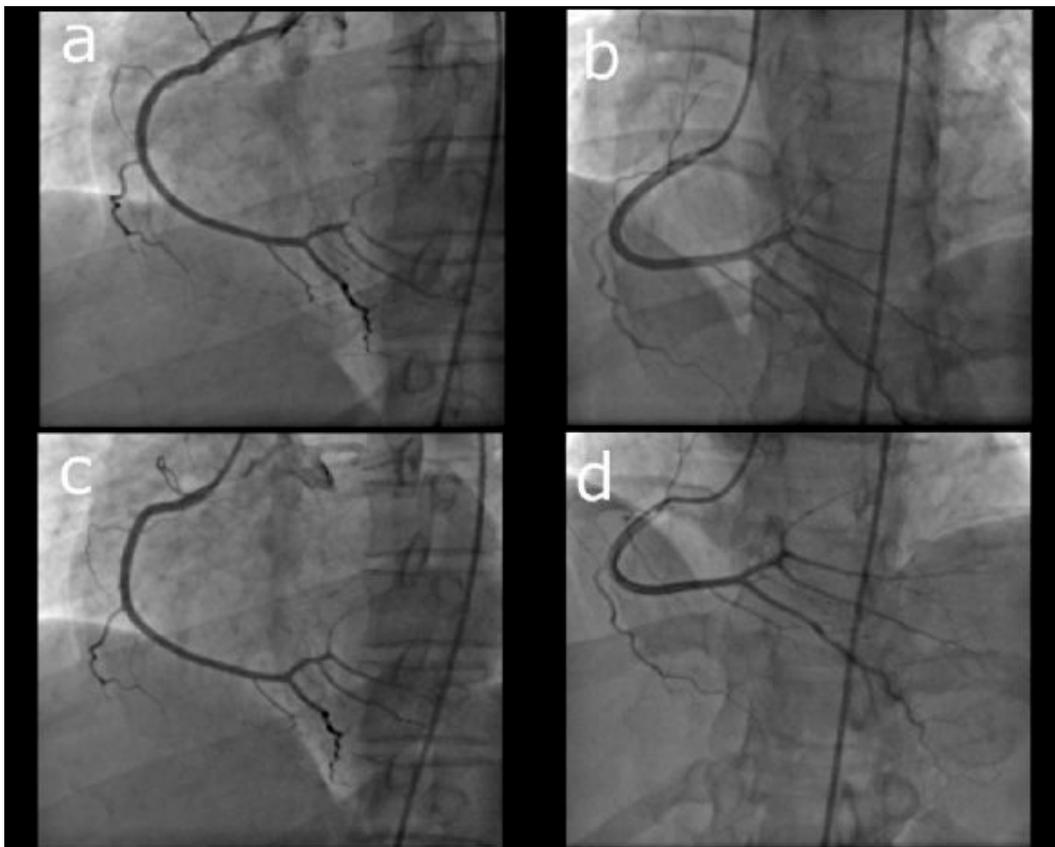
OBJECTIVE: The battle against COVID-19 pandemic continues all around the world. Although the virus primarily targets the respiratory system, the concern about its thrombogenic detrimental effects is growing up. Herein we report a case of COVID-19 induced thrombus causing NSTEMI in a 33 years old woman which recovered via administration of GpIIb-IIIa inhibitor.

CASE: A 33 years old woman was admitted to emergency department with chest pain lasting for six hours. The electrocardiogram was normal. Echocardiography revealed no wall motion abnormality with an ejection fraction of 65%. The first troponin I level was detected to be 0.028 (cut-off value: 0.014). Since the patient was very young, acute coronary syndrome was not considered at first. Nevertheless, the control troponin I level was 0.84. Although the increase in the troponin levels was not consistent with the most possible diagnosis which was acute pericarditis, acute coronary syndrome was also an unexpected diagnosis in a 33 years old woman with no risk factors. Ultimately, the patient was taken to the catheter laboratory and coronary angiography was performed in order not to overlook a possible acute coronary syndrome. The left coronary system was free of disease. The right coronary artery was also free of any plaque formation but there was a thrombus on the bifurcation angle of the posterolateral branch. (Figure 1a, 1b) Since there was not any atherosclerotic lesion, stenting was not performed. The patient was given acetylsalicylic acid, klopidogrel and intracoronary absiksimab on the table. The patient was then taken to coronary intensive care unit. Beta blocker and ACE inhibitor could not be given because of ongoing hypotension and bradycardia. The patient received maintenance dose of absiksimab, acetylsalicylic acid, klopidogrel, low molecular weight heparin and statin therapy and underwent control angiography after two days. It was observed that the thrombus had completely dissolved. (Figure 1c, 1d) The patient was discharged next day with acetylsalicylic acid and klopidogrel.

CONCLUSION: Thrombotic complications are sometimes reported during the course of COVID-19 infection. However it is generally controversial whether this coexistence is a coincidence or complication especially in the elder population. In our case, development of such a thrombus in the absence of any underlying atherosclerotic lesion raises the suspicion of association with COVID-19. This case is also worth of reporting in terms of guiding treatment strategy in these cases. GpIIb-IIIa inhibitors along with antiaggregant and anticoagulant therapy seems rational.

Keywords: COVID-19, thrombus, NSTEMI, GpIIb-IIIa inhibitor

Figure 1



a. Left oblique view showing thrombus in posterolateral artery, b. Left oblique cranial view showing thrombus in posterolateral artery, c. Resolution of thrombus after GpIIb-IIIa inhibitor, d. RCA free of thrombus in left oblique cranial view

OP-113 THE RELATIONSHIP BETWEEN PROLIDASE ACTIVITY AND PAROXYSMAL ATRIAL FIBRILLATION

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Background: Atrial Fibrillation (AF) is the most common type of arrhythmia in the world, affecting approximately 0.5% of the global population. Tissue fibrosis increases in the structure of the atrial tissue of atrial fibrillation patients. Prolidase enzyme regulates collagen synthesis. There may be an association between electrocardiography (ECG) findings and prolidase activity.

Objective: This study investigated the association between prolidase activity, and P-wave dispersion (PWD) in patients with Paroxysmal Atrial fibrillation (PAF).

Methods: A total of 43 patients with PAF (20 female, 23 male; mean age, 46.8 ± 5.7 years) and 42 healthy volunteers (21 female, 21 male; mean age, 43.9 ± 5.1 years) were included in the study. PWD was calculated using a 12-lead ECG was assessed using. All patients were performed doppler imaging and conventional echocardiography. Serum prolidase levels were measured in both groups.

Results: The echocardiographic and demographic characteristics of the PAF patients and healthy controls are presented in Table 1. The two groups were similar in terms of age, body mass index, blood pressure, sex, high- and low-density lipoprotein cholesterol, total cholesterol, and smoking status. Additionally, the LV endsystolic dimension, LV end-diastolic dimension, LV posterior wall thickness, interventricular septum thickness, LA size, and LV ejection fraction were similar in both groups. The atrial electromechanical coupling parameters of the different areas measured by tissue Doppler echocardiography as well as P-wave evaluations are shown in Table 2.

Intra and interatrial EMD, PWD, and Pmax were statistically significant in the PAF group. The serum prolidase level was significantly lower and the hs-CRP level was significantly higher in the PAF group. PWD, Pmin, and Pmax were also significantly higher in the PAF group. Correlation analysis revealed a negative correlation between prolidase level and inter and intraatrial EMD, PWD, and Pmax (r = -0.41, p < 0.001; r = -0.54, p < 0.001; r = -0.62, p = 0.001; r = -0.49, p < 0.001, respectively). Interatrial EMD showed a significant positive correlation with intraatrial EMD, Pmax, and PWD in the PAF group (r = 0.90, p < 0.001; r = 0.43, p < 0.05; r = 0.574, p < 0.001)

Conclusion: The decreased plasma prolidase activity in patients with PAF may explain the

irregularity of the collagen metabolism of different extracellular components and may indicate the onset of atrial remodeling. Changes in PWD, interatrial EMD, and serum prolidase level may predict PAF.

Keywords: Paroxysmal atrial fibrillation, prolidase, fibrosis, collagen synthesis, serum prolidase level, diagnosis.

Keywords: Paroxysmal atrial fibrillation, fibrosis, collagen synthesis, serum prolidase level

Table 1. Demographic and echocardiographic characteristics of the study population

Table 1. Demographic and echocardiographic characteristics of the study population

Variables	PAF group (n = 43)	Control group (n = 42)	P
Age (year)	46.9 ± 5.6	43.9 ± 5.1	0.110
Gender(n)	Female	20	0.310
	Male	23	0.422
Blood pressure	Systolic	127 ± 5	0.010
	Diastolic	77 ± 8	0.010
Heart rate (beats per minute)	64.8 ± 8	65 ± 7	0.971
Body mass index (kg/m ²)	21 ± 2.4	20.8 ± 2.2	0.162
Glucose (mg/dL)	91.9 ± 4.9	85.6 ± 6.2	0.763
Total cholesterol (mg/dL)	177 ± 59	173 ± 52.7	0.991
Triglyceride (mg/dL)	134 ± 35.8	132 ± 55	0.083
High density lipoprotein (mg/dL)	45 ± 16.3	45.3 ± 16	0.913
Low density lipoprotein (mg/dL)	122 ± 44	123 ± 43.3	0.951
Haemoglobin (g/dL)	13.9 ± 1.7	13.7 ± 1.8	0.765
White blood cell count (cells/μL)	11.2 ± 2.9	11.5 ± 3.4	0.524
Platelet count (cells/μL)	250 ± 71.9	246 ± 68.4	0.286
Sodium (mEq/L)	136 ± 2.6	137 ± 3.3	0.262
Potassium (mEq/L)	4.05 ± 0.55	4.0 ± 0.65	0.943
High sensitivity C-reactive protein (mg/dL)	6.6 ± 8.0	1.8 ± 1.6	<0.001
Creatinine (mg/dL)	0.93 ± 0.33	0.96 ± 0.22	0.040
Prolidase (U/L)	3.9 ± 1.1	8.5 ± 3.5	<0.001
Left atrial dimension (mm)	33.0 ± 0.5	33.4 ± 0.5	0.060
Left ventricular end-systolic diameters (mm)	35.3 ± 2.3	36.0 ± 2.8	0.121
Left ventricular end-diastolic diameters (mm)	50.4 ± 2.9	50.2 ± 3.3	0.081
Interventricular septum thickness (mm)	7.8 ± 1.5	8.9 ± 1.0	0.061
Left ventricular posterior wall thickness (mm)	7.4 ± 1.5	7.5 ± 1.6	0.116
Left ventricular ejection fraction (%)	58.3 ± 3.3	59.3 ± 3.9	0.135

Table 2. Comparison of prolidase, PWD, inter and intra-atrial mechanical delay in patients with and control group

Table 2. Comparison of prolidase, PWD, inter and intra-atrial mechanical delay in patients with and control group

Parameters	PAF group (n = 43)	Control group (n = 42)	P value
Lateral PA (ms)	74.40 ± 5.3	73.62 ± 5	0.491
Septal PA (ms)	47.95 ± 5.2	46.4 ± 4.7	0.152
Tricuspid PA (ms)	34.7 ± 3.4	37.9 ± 3.8	<0.001
Inter-atrial electromechanical delay (ms)	39.7 ± 2.7	35.7 ± 2.3	<0.001
Intra-atrial electromechanical delay (ms)	13.2 ± 2.6	8.5 ± 1.9	<0.001
Pmin (ms)	69.8 ± 8.8	66.7 ± 10.2	0.130
Pmax (ms)	114.8 ± 13	93.6 ± 8.6	<0.001
P wave dispersion (ms)	47.1 ± 11	24.1 ± 7.1	<0.001
Prolidase (IU)	3.96 ± 1.2	8.5 ± 3.56	<0.001

OP-115 A NEW SCORING TO PREDICT CEREBROVASCULAR EVENT IN PATIENTS WITH NEW-ONSET ATRIAL FIBRILLATION AFTER CORONARY ARTERY BYPASS GRAFT SURGERY

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Objective: Recommendations for the use of oral anticoagulants (OAC) in the treatment of patients with postoperative atrial fibrillation (POAF) are not clear as in non-surgery atrial fibrillation (AF). In the latest published AF guideline of the European Society of Cardiology (2020); considering the expected clear clinical benefit of OAC treatment and informed patient preferences, although it stated that long-term OAC therapy may be considered with IIb indication to prevent thromboembolic events in patients who develop POAF after cardiac surgery and who are at risk of stroke, issues such as whether the duration of stay in AF is under or above 48 hours is important in determining the initiation and duration of OAC treatment, and whether the CHA2DS2-VASc score or a different score will be used to determine the stroke risk remains unclear, as in non-surgery AF. Our aim is to evaluate the prognostic power of the CVA risk score in predicting the development of cerebrovascular accident (CVA) in patients who underwent isolated CABG surgery and developed POAF with a long AF episode.

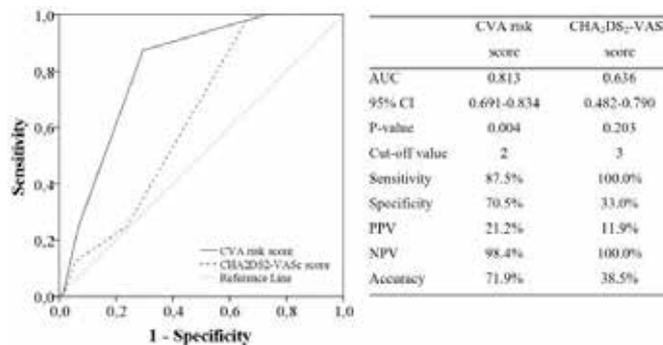
Method: 96 consecutive patients who underwent isolated CABG surgery and developed POAF which continued over 48 hours were retrospectively evaluated. CHA2DS2-VASc score and CVA risk score (> 70 years old: +1 point, hypertension: +1 point, CVA history: +1 point, left ventricular ejection fraction < 45%: +1 point) was calculated by using preoperative data for each patient. The CVA risk score was obtained from a recently published study investigating CVA risk factors in patients who developed AF after isolated CABG surgery and had an AF stay of less than 48 hours. The effectiveness of these two scores on CVA development after a mean follow-up of 36.1 ± 18.1 months was evaluated by using the Cox proportional hazards model.

Results: After the follow-up, eight (8.3%) patients developed CVA, in which two (2.1%) of them were in-hospital events. While in the regression analysis, there was no significant effect of CHA2DS2-VASc score on CVA development, CVA risk score was found to be associated with the development of CVA both in unadjusted analysis and when adjusted with different variables. Moreover, having a CVA risk score of two or more was more successful than the CHA2DS2-VASc score in detecting patients with a high CVA incidence.

Conclusion: Using the CVA risk score instead of the classical CHA2DS2-VASc score may be considered to evaluate the risk of CVA development in patients who develop POAF after isolated CABG surgery.

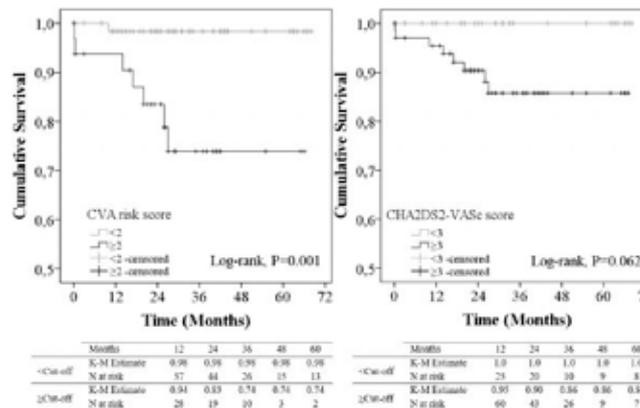
Keywords: Cerebrovascular accident, CHA2DS2-VASc score, CVA risk score, Postoperative atrial fibrillation

Figure 1: ROC curves for the detection of cerebrovascular accident in POAF patients and cerebrovascular accident prediction values of cut-offs.



CVA, cerebrovascular accident; AUC, area under curves; CI, confidence interval; PPV, positive predictive value; NPV, negative predictive value.

Figure 2: Kaplan–Meier curves for cerebrovascular accident stratified by CVA risk score and CHA2DS2-VASc score.



CVA, cerebrovascular accident; K-M, Kaplan–Meier.

Table 1: Unadjusted and adjusted cox proportional hazard analysis for cerebrovascular accident.

	HR	95% CI	P-value
CHA2DS2-VASc score			
Unadjusted	1.257	0.837-1.887	0.270
CVA risk score ≥ 2			
Unadjusted	14.745	1.813-119.939	0.012
Adjusted (Gender)	16.564	2.026-135.436	0.009
Adjusted (Diabetes mellitus)	17.722	2.136-147.058	0.008
Adjusted (Left atrial diameter)	12.237	1.483-101.007	0.020
Adjusted (Hematocrit)	25.461	2.866-226.157	0.004
Adjusted (eGFR)	18.743	2.166-162.228	0.008
Adjusted (Aspirin)	14.542	1.787-118.3296	0.012
Adjusted (P2Y12 inhibitors)	16.399	2.004-134.210	0.009
Adjusted (VKA)	18.603	2.229-155.264	0.007
Adjusted (AF at discharge)	14.243	1.743-116.353	0.013

HR, hazard ratio; CI, confidence interval; CVA, cerebrovascular accident; eGFR, estimated glomerular filtration rate; VKA, vitamin K antagonists

OP-116 PREDICTORS OF MAJOR ADVERSE CARDIAC EVENTS AFTER RENAL TRANSPLANTATIONUmut Kocabas*Department of Cardiology, Baskent University Izmir Hospital, Izmir, Turkey*

Objective: Renal transplantation is the optimal treatment strategy for patients with end-stage renal disease and results in reduced mortality compared to chronic dialysis treatment. However, cardiovascular diseases remain the leading cause of mortality and morbidity after renal transplantation. The higher incidence of major adverse cardiac events (MACEs) after renal transplantation is associated with presence of cardiovascular risk factors. Given that evidence is not conclusive, we aimed to investigate the prevalence of pretransplant cardiovascular risk factors, and to determine the association between pre-transplant cardiovascular risk factors and post-transplant MACEs.

Methods: We analyzed 174 patients (>18 years) with end-stage renal disease who underwent renal transplantation in the Baskent University Istanbul Hospital between January 2011 and January 2017. The primary outcome of the present study was occurrence of the MACEs after transplantation.

Results: Before transplantation, 21.2% of patients had a coronary artery disease and 10.9% of patients had a congestive heart failure. The most frequent comorbidities were anemia (76.4%), hypertension (66.7%), dyslipidemia (29.9%), and diabetes (28.2%). Approximately one-third of the study population had a history of smoking. After transplantation, the median follow-up of the study population was 4.7 years (range 2.9–8.1). During follow-up period, there were 67 MACEs in 44 patients. According to the multivariable analysis, independent pre-transplant risk factors for post-transplant MACEs were older age, non-use of RAS inhibitors, reduced left ventricular ejection fraction, increased left ventricular mass index, and high fasting blood glucose.

Conclusion: There is a high prevalence of cardiovascular risk factors in patients with end-stage renal disease, and there is increased risk for MACE and death.

Keywords: cardiac event, renal transplantation, risk factors

OP-117 RETROSPECTIVE ANALYSIS OF CARDIAC IMPLANTABLE DEVICE EXTRACTIONS IN ADULT PATIENTS

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Objective: The number of implanted permanent pacemaker (PPM), implantable cardioverter-defibrillator (ICD) and cardiac resynchronization therapy (CRT) devices is increasing all over the world. However, one of the most critical problems in implanted devices is removing the device due to infection. This study aimed to examine the characteristics of patients who had an implantable cardiac device extracted due to infection.

Method: Between January 2014 and June 2021, patients with PPM, ICD and CRT implanted, replaced, and partially or entirely removed cardiac devices in Adiyaman training and research hospital were identified using ICD 10 and interventional procedure codes via the hospital's electronic medical records system. In addition, demographic data and clinical features of patients whose implantable device was extracted were evaluated retrospectively.

Results: During the 88 months, 102 CRT- Defibrillator (CRT-D), 167 ICD, and 222 PPM were implanted or replaced. In this period, in 27 patients, the cardiac device due to local pocket infection was entirely or partially extracted (pulse generator removed, leads were left). Demographic and clinical characteristics of 27 patients whose implantable cardiac device was removed are summarized in Table 1. The mean age of the patients was 64.8 ±12.3 years, and 70.4% (n:19) were male. Twenty-four of these patients had a cardiac device previously implanted in our center. Of the removed devices, 13 were ICD, 9 were CRT-D, and 5 were PPMs. The mean removal time after the first implantation was 16.2 months (SD: 13.7). The most common comorbidities in these patients were coronary artery disease, heart failure and hypertension (79%, 76%, 68%, respectively). Bacteria were found in cultures obtained from the infected pocket area of six patients. However, microorganisms did not grow in any patient's blood culture, and no patient was diagnosed with infective endocarditis. Antibiotic treatment was given to all patients in the perioperative period and at discharge. A cardiac device was implanted again in 25.9% (n: 7) patients. Three of the patients whose device was removed died after the procedure due to non-cardiac causes.

Conclusion: Partial or complete extraction of cardiac devices due to infection was not uncommon in this single-center study. When we look at the patients whose cardiac device is removed, it is seen that more than one lead and larger pulse generators are used in these patients, and they also have additional comorbidities. Many of these patients were not subsequently implanted with a new cardiac device. A more selective decision should be made, considering the cost and effectiveness, in the first indication for cardiac device insertion. In addition, increasing the education and awareness of the implant team about cardiac device implantation and subsequent care is important in reducing device-related infections.

Keywords: implantable, device, extraction

Table 1. Demographic ve clinical characteristics of cardiac implantable device extracted (CIDE) patients

	CIDE (n=27)	%
Age, years (mean)	64,8±12,3	
Male	19	70,4
Coronary artery disease	20	76,9
Heart failure	21	77,8
Hypertension	19	70,4
Diabetes Mellitus	15	55,6
CABG or valve surgery	11	40,7
Chronic pulmonary disease	7	25,9
Malignancy	4	14,8
Renal disease (creatinine >=1.5 mg/dL)	3	11,1
Duration of implant, months	16,2±13,7	
Device Type		
Dual-chamber ICD	11	33,3
CRT-defibrillator	9	27,3
Dual-chamber permanent pacemaker	5	15,2
Single chamber ICD	2	6,1
Existing device location		
Pectoral	27	100
Entirely extracted	24	85,2
Culture organism		
Staphylococcus Epidermidis	3	11,1
Staphylococcus Hominis	1	3,7
Burkholderia Cepacia	1	3,7
Klebsiella Pneumonia	1	3,7

CABG; Coronary artery bypass graft, ICD; Intracardiac defibrillator

OP-119 ASSESSMENT OF EXERCISE STRESS TEST PARAMETERS IN PATIENTS WITH ERECTILE DYSFUNCTION

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Objective: Erectile dysfunction (ED) has a significant impact on quality of life, given its high prevalence and association with cardiovascular diseases. In the present study we aimed to evaluate exercise treadmill parameters in patients with ED.

Method: A total of 178 patients who applied to cardiology clinic was enrolled in the study. Patients were divided into two groups according to their International Index of Erectile Function-5 (IIEF-5) score. Exercise time, maximum heart rate (HR), resting HR, chronotropic index (CI), heart rate recovery at one minute (HRR1) and two minutes (HRR2) were evaluated for each patient.

Results: Subjects with ED were older, had similar rates of DM and HT compared to control group.. Exercise time and peak HR was higher in ED patients compared to the healthy counterparts (556.00 (61) sec. vs 575.5 (84) sec. $p=0.025$, and 156.00 (13) bpm vs 160.50 (11) bpm $p=0.001$, respectively). We did not find statistically significant differences with respect to resting HR, HRR1, HRR2, CI, maximum systolic and diastolic blood pressure, or rate pressure product between two groups. Baseline demographic, biochemical and exercise stress values of the groups is presented in Table 1. According to their IIEF-5 scores, 3 (1.7%) subjects had severe-moderate disease, 35 subjects (19.6%) had mild-moderate disease, 85 (47.8%) had mild disease and the rest (n=55, 30.9%) had normal erectile function. IIEF-5 score was negatively correlated with age ($r=-0.54$, $p<0.001$), TC ($r=-0.32$, $p<0.001$), LDL-C ($r=-0.34$, $p<0.001$), TG ($r=-0.17$, $p=0.02$) and positively correlated with exercise time ($r=0.19$, $p=0.01$) and maximal HR ($r=0.24$, $p=0.001$). We further investigated the prevalence of abnormal HRR1 values between two groups. Twenty-nine subjects (23.6%) had abnormal HRR1 values in the study group, whereas 6 subjects (10.9%) had abnormal HRR1 values in the control group ($p=0.04$).

According to Quade's ANCOVA results, exercise time and maximal HR were not different between two groups when age used as a covariate ($F=1.032$ $p=0.311$ and $F=1.264$, $p=0.262$, respectively).

Conclusion: It has been reported that exercise-induced increase in HR is primarily mediated by sympathetic activity whereas post-exercise 1 minute decrease in HR depends on parasympathetic activation. According to our results, abnormalities of parasympathetic system activity were more prevalent in ED patients. Since exercise treadmill testing is a useful tool for the assessment of autonomic nervous system activity, it can be used to assess autonomic activity in that group of patients.

Keywords: Erectile dysfunction, heart rate recovery, stress

TABLE 1

	Study group (n=123)	Control group (n=55)	p
Age (years)	46.83±6.752	40.38±6.072	<0.001
Exercise time (second)	556.00 (61)	575.5 (84)	0.025
Resting HR (bpm)	84.91±12.827	86.10±13.696	0.332
Max HR (bpm)	156.00 (13)	160.50 (11)	0.001
HRR1 (bpm)	26.00 (8.00)	28.00 (13.00)	0.111
HRV2 (bpm)	42.00 (11)	45.00 (18)	0.297
CI	0.77 (0.14)	0.815 (0.15)	0.059
Max SBP (mmHg)	150.00 (25.00)	150.00(30.00)	0.254
Max DBP (mmHg)	80.00 (10.00)	85.00 (10.00)	0.617
RPP (mmHg*bpm)	23400.00 (5660)	23885.00 (5108)	0.190
TC (mg/dl)	199.77±43.832	195.75±39.125	0.761
TG (mg/dl)	121.00 (100.00)	125.00 (113.00)	0.517
HDL (mg/dl)	43.00 (12.0)	42.00 (10.5)	0.143
LDL (mg/dl)	125.00 (53.0)	123.55 (56.5)	0.336
Smoking (n, %)	49 (39.8)	32 (58.2)	0.023
Alcohol (n, %)	24 (19.5)	21 (38.2)	0.010
DM (n, %)	7 (5.7)	2 (3.6)	0.552
HT (n, %)	11 (9.1)	11 (9.1)	0.685
HRR1 (<=18 bpm) (n, %)	29 (23.6)	6 (10.9)	0.040

Biochemical and treadmill stress test parameters of the groups.

OP-120 CARDIAC CHANGES WITH SUBCLINICAL HYPOTHYROIDISM IN WOMEN WITH METABOLIC SYNDROME

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Objective: Metabolic syndrome (MetS) is known to cause significant cardiovascular changes. The association of subclinical hypothyroidism with cardiovascular events has not been fully elucidated. Our purpose in this study is to investigate the effect of MetS on cardiac functions and the contribution of subclinical hypothyroidism in women.

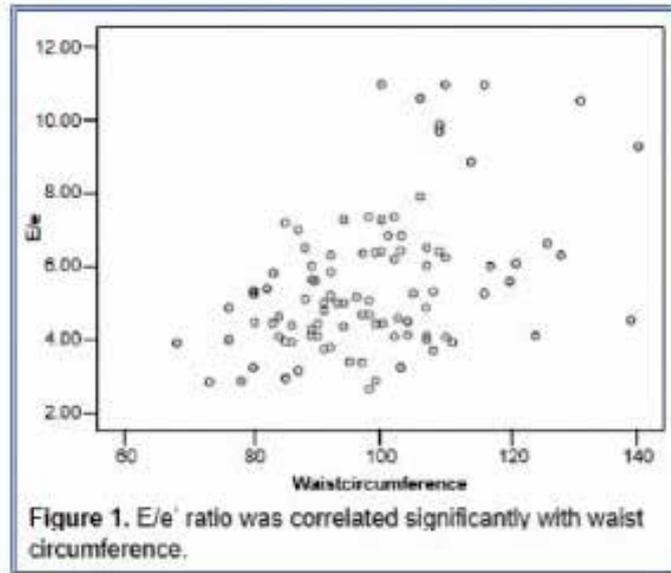
Method: 98 healthy women with a mean age of 30.9±6.2 were included in the study. Thyroid functions were evaluated in all cases and an echocardiographic examination was performed. Height, weight, waist, and hip circumference were measured. Patients who met at least 3 diagnostic criteria according to NCEP-ATP3 MetS criteria were included in the study.

Results: Left ventricular mass (LVM) was found to be higher in patients with MetS (p<0.001). Diastolic filling parameters of the left ventricle shown by Doppler were found to be abnormal in patients with MetS. E/e' ratio was found to be higher in MetS cases (p=0.001) and left atrial volume (SAH) was higher (p<0.001). Left ventricular TEI index was found to be higher in patients with MetS (p=0.035). TSH level was significantly higher in MetS patients (p=0.001). A positive correlation was found between TSH and BMI, waist circumference, LAV, LVM, and myocardial performance index of the left ventricle. It was observed that patients with MetS had impaired systolic and diastolic functions with high TSH values. The relationship of E/e' ratio with anthropometric and biochemical parameters was evaluated with multiple regression analysis. Waist circumference was found to be a strong independent variable associated with the E/e' ratio.

Conclusion: As a result, it was thought that structural and functional deterioration of the heart may be related to subclinical hypothyroidism in patients with MetS.

Keywords: Metabolic syndrome, myocardial performance index, subclinical hypothyroidism

Figure-1.



Tablo-1. Demographic and echocardiographic results

		Non-MetS group (n=49)	MetS group (n=49)		p
	Mean±SD	Min-Max	Mean±SD	Min-Max	
Age (years)	28.9±5.6	18-42	31.4±7.4	18-46	0.189
Waist circumference (cm)	91.4±8.2	69-106	106.9±9.1	92-140	<0.001
TSH	1.9±1.3	0.24-6.3	2.3±1.5	0.27-7.91	0.05
FT4	1.1±0.7	0.7-2.1	1.0±0.6	0.6-2	0.245
FT3	2.6±0.7	0.65-3.47	2.7±0.7	0.58-3.15	0.321
Fasting glucose (mg)	86.2±8.2	70-100	89.4±7.3	70-100	0.109

		Non-MetS group (n=49)	MetS group (n=49)		p
Triglycerides (mg/dl)	99.5±44.7	44-218	191.3±68.2	168-387	<0.001
T-Chol (mg/dl)	186.2±35.7	111-286	187.8±33.9	121-271	0.83
HDL (mg/dl)	55.5±12.2	34-86	40.2±8.1	32-65	<0.001
LDL (mg/dl)	113.8±23.5	64-171	139.6±27.9	82-181	0.218
Systolic AP (mmHg)	115.5±12.8	81-135	114.2±12.7	90-135	0.09
Diastolic AP (mmHg)	71.8±7.7	60-93	75.9±8.8	60-94	0.08
LVEF (%)	61.3±2.3	57-68	61.3±2.0	58-65	0.944
LVM (g)	133.5±34.3	54.9-232.7	153.1±31.5	98.7-221.6	0.002
Mitral dec T (msn)	183.8±31.7	121-254	196.5±36.3	112-266	0.1
E/e'	7.1±1.5	4.2-12.4	7.7±1.8	4.6-13.2	0.033
Mitral Tei index	0.43±0.07	0.27-0.7	0.48±0.07	0.36-0.67	0.005
LAV (mm ³)	40.8±9.7	18.5-64	54.8±13.2	35.1-82.6	<0.001
TSH: Thyroid-stimulating hormone; FT3: Free triiodothyronine; FT4: Free thyroxine hormone; T-Chol: Total cholesterol; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; AP: Arterial pressure; LVEF: Left ventricular ejection fraction; LVM: Left ventricular mass; Mitral dec T: Mitral deceleration time; LAV: Left atrial volume.					

OP-121 MATERNAL AND PERINATAL OUTCOMES IN PREGNANT WOMEN WITH LEFT VENTRICULAR NONCOMPACTIONAysun Erdem Yaman¹,¹*Dr Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital*

Background: Isolated left ventricular noncompaction (LVNC) is a cardiomyopathy characterized by prominent myocardial trabeculations and deep intertrabecular recesses in the left ventricular (LV) cavity without congenital heart disease. Cardiovascular complications are increased in LVNC. Although interest in LVNC has recently increased there is still not enough information about the pregnant women with isolated LVNC. Therefore we wanted to investigate the maternal and perinatal outcomes in pregnant women with isolated LVNC.

Methods: Retrospectively 30 women with isolated LVNC who had history of pregnancy were retrospectively included to study from hospital records. Four out of 30 women were diagnosed with isolated LVNC before pregnancy while in seven women isolated LVNC was diagnosed during pregnancy. However remaining 19 women were diagnosed with isolated LVNC after pregnancy. The maternal outcomes were compared between 30 pregnant women with LVNC and 30 healthy-pregnant women. Additionally the perinatal outcomes were compared between 44 pregnancies with LVNC and 43 healthy-pregnancies.

Results: In our study arrhythmias (23.3%vs.6.7%), thromboembolic events (6.7%vs.0%) and heart failure (6.7% vs.0%) were slightly increased in pregnant women with LVNC compared to healthy-pregnant women. Moreover, miscarriage was significantly higher in pregnancies with LVNC compared to healthy-pregnancies in our study (22.7% vs. 4.7%).

Conclusions: Pregnant women with LVNC can tolerate pregnancy well, However they are at the increased risks of arrhythmias, thromboembolic events and heart failure. Additionally pregnancies with LVNC could be associated with increased risk of miscarriage.

Keywords: left ventricular noncompaction, pregnancy, cardiac events

**INTERNATIONAL
YOUNG ACADEMY OF CARDIOLOGY
E-CONGRESS**

2021

**POSTER
PRESENTATIONS**

EP-001 SUBCLINICAL HYPERCORTISOLISME AND METABOLIC SYNDROMAbderraouf Senhadji¹¹Constantine²Annaba³Guelma⁴Algeria

Subclinical Cushing is defined by the presence of biological hyper-cortisolism without the morphological modifications of Cushing's syndrome. The prevalence of infra-clinical Cushing is estimated at 0.8% in the population. The average age of the patients being 35 years, it is accompanied in a very large majority of cases of arterial hypertension in a context of metabolic syndrome. Few patients progress to a clinical Cushing. Subclinical Cushing can be supported by a cortisolic adenoma or bilateral adrenal hyperplasia which is sometimes much more marked on one side. Its pathophysiology is not fully understood. The presence of illicit receptors within adrenal tissue making the gland sensitive to stimuli other than ACTH has been shown. The first line diagnosis should be simple and very sensitive. It shouldn't cost too much and shouldn't pass up false negatives, ideally it should also be specific. The laboratory abnormalities allowing the diagnosis of Cushing's syndrome are quantitative (excessive cortisolic secretion) and qualitative (loss of the circadian rhythm of cortisol secretion and abolition of the negative feedback of exogenous corticosteroids on the production of cortisol). However, it should be emphasized that the cut-off values for the various biological parameters depend on the assay kits used and that they cannot be considered absolute and used indiscriminately. The laboratory abnormalities allowing the diagnosis of Cushing's syndrome are quantitative (excessive cortisolic secretion) and qualitative (loss of the circadian rhythm of cortisol secretion and abolition of the negative feedback of exogenous corticosteroids on the production of cortisol). However, it should be emphasized that the cut-off values for the various biological parameters depend on the assay kits used and that they cannot be considered absolute and used indiscriminately. The laboratory abnormalities allowing the diagnosis of Cushing's syndrome are quantitative (excessive cortisolic secretion) and qualitative (loss of the circadian rhythm of cortisol secretion and abolition of the negative feedback of exogenous corticosteroids on the production of cortisol). However, it should be emphasized that the cut-off values for the various biological parameters depend on the assay kits used and that they cannot be considered absolute and used indiscriminately. To assess the need for specific management of HCICs, it is necessary to determine their complication and especially the cardiovascular risk, the benefit obtained by their treatment as well as the markers correlated with the intensity of the complications making it possible to determine the population for whom a treatment would be beneficial.

Keywords: Subclinical hypercortisolisme, metabolic syndrom, cardiovascular risk

EP-002 A RARE CAUSE OF DYSPNEA: U-SHAPED PATENT FORAMEN OVALE BEHAVES LIKE AN ATRIAL SEPTAL DEFECT

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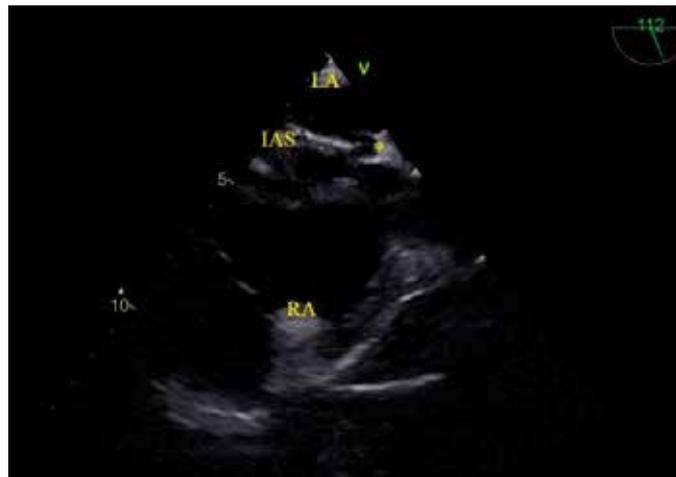
Objective: Patent foramen ovale (PFO) is the most common congenital heart abnormality of fetal origin, and it is present in approximately 25% of the adult population. PFO is the result of unsuccessful closure of the foramen ovale, which is a normal structure in the fetal circulation to direct right-to-left blood flow for bypassing the pulmonary circulation. Although PFO is often associated with atrial septal aneurysm, Chiari network, and rarely with Ebstein anomaly, there are few cases in the literature in which PFO physiologically behaves like an atrial septal defect (ASD). In this case, we present a U-shaped PFO behaving like an ASD.

Case: A 45-years-old male patient presented to the cardiology outpatient clinic with progressive dyspnea in the last two months. The patient had no known chronic diseases. Physical examination revealed a 2/6 pan-systolic murmur on the left sternal border. Electrocardiography (ECG) revealed a normal sinus rhythm and right bundle branch block. Transthoracic echocardiography (TTE) was performed, revealing a normal left ventricular systolic function and dilated right heart chambers. Therefore, transesophageal echocardiography (TEE) was planned. On TEE, there was a U-shaped PFO with a significant left atrial-right atrial shunt, which hemodynamically behaves like an ASD (Figure 1 and Figure 2). The ratio of pulmonary flow to systemic flow (Q_p/Q_s) was calculated as 1.7. The patient's dyspnea complaint was thought to be due to a significant shunt between the left atrium and the right atrium. Hence, cardiac surgery or percutaneous ASD occluder device was recommended. However, the patient refused both procedures.

Conclusion: Iatrogenic ASD can be detected after septostomy or by retraction of the left atrial flap with an interatrial septal aneurysm. However, there has been a few reported cases of U-shaped PFO that hemodynamically behaves like an ASD. With this case, we aimed to contribute to the current literature by showing a different mechanism of a PFO that acts like an ASD.

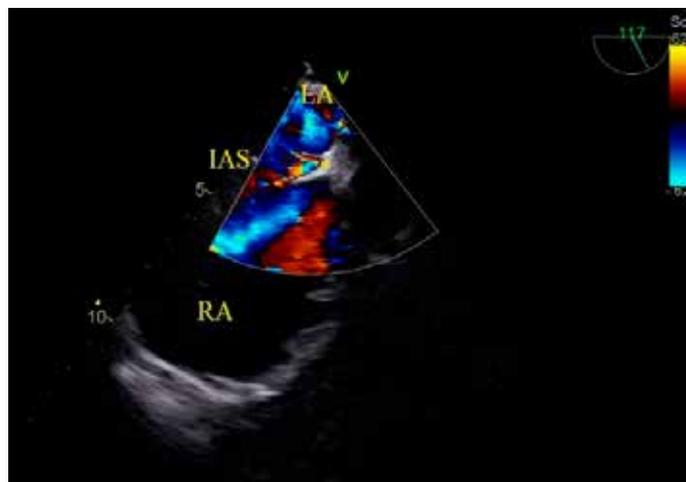
Keywords: Patent foramen ovale, atrial septal defect, behave

Figure 1



TEE image of interatrial septum showing a U-shaped PFO

Figure 2



Color Doppler TEE examination of the U-shaped PFO

EP-003 A CASE OF SPONTANEOUS CORONARY ARTERY DISSECTION RELATED TO THE USE OF AMFETAMINE

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Spontaneous coronary artery dissection (SCAD) is a rare non atherosclerotic cause of acute coronary syndromes (ACS). More rarely, almost 0.6% of SCAD cases were found to be caused by the use of amphetamine and its derivatives.(1) Intravascular imaging integrated invasive CAG is the gold standard for the diagnosis of SCAD. Conservative treatment, percutaneous coronary intervention(PCI) and surgery are treatment options for SCAD. Spontaneous coronary artery dissection may occur in patients with chronic MDMA use. Here, we present an amphetamine-associated spontaneous coronary artery dissection.

Keywords: Spontaneous coronary artery dissection, Acute coronary syndromes, Coronary angiography

Figure 1

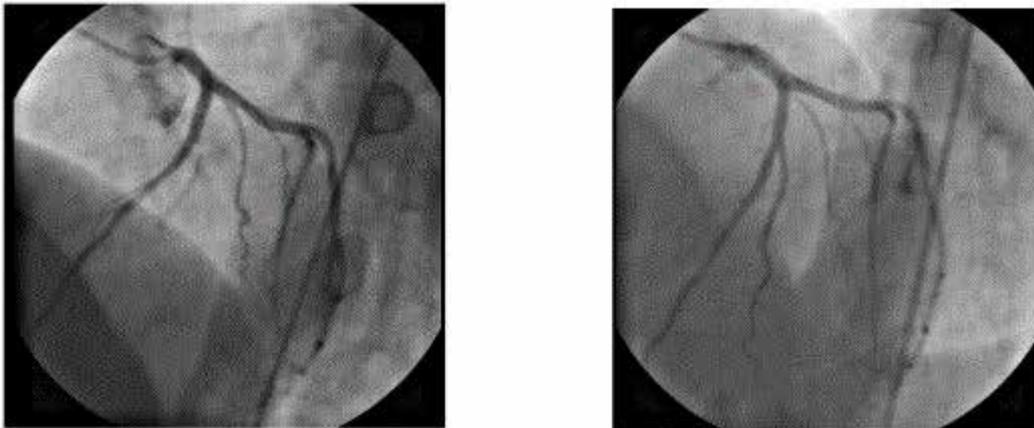


Figure 1 is showing stenting of diagonal branch of LAD artery which was performed 9 years ago after an acute coronary syndrome event.

Figure 2



Figure 2 is showing type-1 spontaneous coronary artery dissection that does not restrict flow in the amphetamine-associated LAD mid segment.

A case of spontaneous coronary artery dissection related to the use of amphetamine

Background: Spontaneous coronary artery dissection (SCAD) is a rare non-atherosclerotic cause of acute coronary syndromes (ACS). More cases are now being identified due to increased awareness and earlier use of invasive angiography in patients presenting with acute chest pain.

It frequently appears as a pregnancy-related condition in young women, fibromuscular dysplasia, systemic inflammatory diseases, connective tissue disorders, hormone therapies may play a role in etiology. More rarely, almost 0.6% of SCAD cases were found to be caused by the use of amphetamine and its derivatives.(1) Here, we present an amphetamine-associated spontaneous coronary artery dissection.

Case: A fifty- year-old male patient with a previous history of stenting of diagonal branch of left anterior descending (LAD) artery(Figure 1)was admitted emergency with pressure-like chest pain for 2 hours. He was not on regular anti-ischemic therapy. He has been smoking, drinking alcohol and abusing 3,4-methylenedioxymethamphetamine (MDMA) for 20 years. He stated that chest pain had started after the combined use of alcohol and MDMA. Except high blood pressure (210/140 mmHg), physical examination was no remarkable. Electrocardiogram (ECG) was sinus rhythm with 1 mm ST segment depression on lead V5-V6 and negative T wave on D1-aVL. After administration of iv nitroglycerin infusion, all ECG findings, blood pressure and chest pain improved. Transthoracic echocardiography was normal with an ejection fraction 60%. All blood tests were normal at the time of admission. Troponin level increased 5 times above the upper reference limit of normal after 3 hours from the first admission. So myocardial infarction without ST segment elevation (NSTEMI) diagnosis was made and 180mg ticagrelor, 300mg acetylsalicylic acid (ASA) was given to the patient as loading dose of antiplatelet therapy. Coronary angiography (CAG) revealed 80% narrowing of mid right coronary artery (RCA) as culprit

lesion and it was treated with 3.0x18mm drug eluting stent (DES). On CAG the stent in diagonal artery was open and there was also a non-obstructive type-1 SCAD in the mid segment of LAD (Figure 2).

Discussion: Although its real prevalence is still unknown, in recent studies SCAD has been found responsible for 0.1% -0.4% of all ACS cases in general population. While it is responsible for 25% of ACS cases in women under 50 age(2), 10-15% of cases are seen in men. It occurs at a younger age in men and associated with physical exercise and stress.(3) No cause was identified in 45% of all cases, highlighting that many cases of SCAD remain unexplained. The commonest identified predisposing factors were postpartum, fibromuscular dysplasia (FMD), connective tissue disease and hormonal therapy. Potential stressors include extreme physical exertion, particularly in young male patients, intense emotional stress, sympathomimetic drugs (such as cocaine, amphetamines), childbirth and Valsalva-like maneuvers (such as coughing, retching, vomiting). Triggers for SCAD are thought to increase shear stress on the coronary artery wall, often mediated by elevated catecholamine levels and intra-abdominal pressure. (1)

Almost all of the patients present with acute coronary syndrome. Approximately 26.1% of patients present with ST elevation myocardial infarction (STEMI) and 65.4% with NSTEMI-ACS, 3.6% with VT/VF. (1) Intravascular imaging integrated invasive CAG is the gold standard for the diagnosis of SCAD. (5) There are three different types of SCAD defined in coronary angiography. Type 1 which is accounted 29% of cases is the classic type characterized as double lumen appearance with radiolucent staining on the coronary artery wall. That is pathognomonic for type 1. This is the most easily recognized type by CAG. Type 2, 67% of all cases, is characterized as long (> 30 mm), diffuse and flat stenosis that can vary in severity from mild stenosis to occlusion. There is a sudden change in vessel diameter between normal and diseased parts. Other coronary vessels do not have atherosclerotic lesions. It is the most common type. Type 3, 4% of all cases, present with multiple focal long stenosis due to intramural hematoma mimicking atherosclerosis. It is the most difficult type to recognize. Optical coherence tomography (OCT) or intravascular ultrasound (IVUS) is required to distinguish Type 3 SCAD. (4)

Left anterior descending artery is the most frequently affected vessel (70-80%) and multi-vessel dissection is detected in 20-25% of cases. Coronary dissections are more common in the middle and distal segments containing lateral branches.(4-6)

Conservative treatment, percutaneous coronary intervention (PCI) and surgery are treatment options for SCAD. Coronary dissection generally heal spontaneously, so conservative treatment is recommended in uncomplicated cases. (3,9) In patients with complete occlusion, involvement of left main coronary artery (LMCA), ongoing ischemia, recurrent chest pain or hemodynamic instability are the situations which require coronary revascularization. When compared with atherosclerotic lesions, the successful PCI of SCAD is less possible.(3-6) Coronary Artery Bypass Grafting (CABG) should be considered in patients with dissection of LMCA or after failed PCI. In the patients treated with CABG, graft stenosis due to competition between native and graft vessel caused by the resorption of intramural hematoma is often. (6) In patients with non-critical luminal stenosis with TIMI-3 flow and stable clinical condition, conservative treatment is recommended. Follow-up these patients with treadmill test or CAG is not recommended. Because the expansion risk of dissection and subsequent cardiac events, these patients should be hospitalized for 1 week. (3) To maintain main lumen flow and prevent thrombosis, standard ACS treatment should be applied at the first step. Intimal rupture is pro-thrombotic after considering the risk of bleeding for every patient, DAPT should be continued for 1-12 months. After 1 year of follow up, ASA alone is recommended.(3)

Use of IVUS and OCT is useful to confirm the diagnosis and if PCI is selected for treatment, it enables to perform PCI more accurate and safely. IVUS/OCT is also important to show if the coronary wire is in the true lumen and they are also helpful in choosing the right stent size and detection of the dissected vessel area more accurately. Also the recognition of a potential arteriopathy is facilitated by these techniques.(7)

In this case, culprit lesion was the subtotal stenosis in the mid segment of RCA. Coincidentally there was a non-obstructive type-1 SCAD in the mid segment of LAD. So that the lesion in RCA is revascularized with PCI. Conservative approach is the preferred treatment option of amphetamine-related dissection in LAD mid segment

Conclusion: Spontaneous coronary artery dissection may occur in patients with chronic MDMA use. So patients presenting with ACS due to SCAD should be questioned for substance abuse.

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EP-004 BIDIRECTIONAL VENTRICULAR TACHYCARDIA DUE TO NOREPINEPHRINE INFUSION

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Introduction: Bidirectional ventricular tachycardia (BDVT) is an uncommon form of ventricular tachycardia (VT), characterised by a beat-to-beat alternation of the frontal QRS axis. It has been described in a variety of clinical settings including digitalis toxicity, herbal aconite poisoning, myocarditis, hypokalemia, cardiac coronary artery disease, metastatic cardiac tumors, cardiomyopathy, Anderson-Tawil syndrome, pheochromocytoma and at the same time, has been recognized as a hallmark of catecholaminergic polymorphic ventricular tachycardia (CPVT). In this presentation We mention a case of BDVT in a patient with Hakim Adams syndrome during nöradrinaline infüsiön.

Case presentation: A 73-year-old male patient diagnosed with Hakim Adams syndrome and previous cerebrovascular event was brought to the emergency department as a result of adding general condition disorder to complaints of fever, nausea and vomiting that began 2 days ago. Arterial blood pressure (TA) 85/65mmhg, electrocardiography (ECG) 102/ min sinus rhythm, fever 38.7 °C, white blood cell:17500/µL, consciousness was observed as samnole in the examination of patient on admission.Septic shock secondary to urinary tract infection was considered as a preliminary diagnosis in the patient. After the absence of an increase in TA values in the patient's follow-up, an infusion of neuradrenaline was started.VT occurred in patient while the infusion of neuradrenaline continued, direct current synchronized cardioversion was performed in the emergency department, and then we were consulted when there was no change in the patient's rhythm. EKG of the patient was evaluated as BDVT (Figure-2), additional cardiac examination and transthoracic echocardiography were normal.Intravenous beta blocker (single dose metoprolol tartarate 5 mg) was given to patient and the rhythm returned to the sinus. We assessed BDVT as CPVT, which occurs while the catecholamine infusion is ongoing.

Discussion: In patients with no structural heart disease and normal ECG, the diagnosis of CPVT should come to mind in the case of bidirectional or polymorphic VT caused by exercise, emociennial stress, or catecholamine infusion. Beta blockers are the most important drug in the treatment of CPVT. Although CPVT is diagnosed frequently in young patients, the presence of additional diseases of our patient and the absence of an active life-style due to its immobility may be some of the reasons that play a role in the occurrence of arrhythmia or delay in its diagnosis. Due to the appearance of BDVT as a result of ketacolamine infusion in the patient and its dramatic response to beta blockers, we considered CPVT as a preliminary diagnosis. As a result, it should be noted that CPVT can be seen not only in childhood, but also in older people.In cases of tachycardia that occur in patients as a result of catecholamine infusion, close rhythm monitoring is vital for CPVT and other fatal arrhythmias.

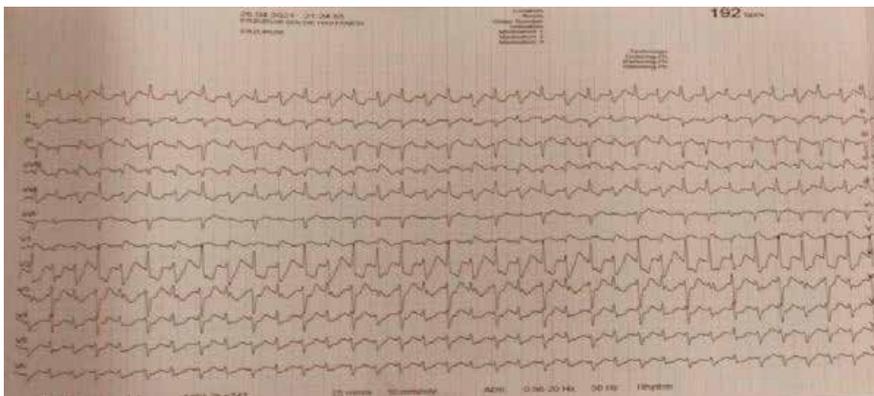
Keywords: Bidirectional Ventricular Tachycardia, Catecholamine, Catecholaminergic Polymorphic Ventricular Tachycardia

Figure 1



Figure -1a) Enlarged ventricles in patient's brain magnetic resönance image, 1b) Enlarged ventricles and accompanying old ischemic areas in brain tomography.

Figure-2



Patient's ECG during tachycardia

EP-005 TOTAL LAD OCCLUSION IN A POST-COVID-19 PATIENT

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Coronavirus disease 2019 (COVID-19) is associated with a wide spectrum of cardiovascular manifestations include acute coronary syndrome (ACS), myocarditis, and arrhythmias. We presented a patient with combination of total proximal left anterior descending artery (LAD) occlusion and recovered COVID-19 with neither troponin increase nor electrocardiography (ECG) change.

CASE REPORT: A 45 years old man presented to our out-patient cardiology department with a new onset typical angina. His medical history included recovered COVID-19 with mild symptoms 4 months ago. Physical examination and electrocardiogram (ECG) revealed normal (E).

Transthoracic echocardiography(TTE) demonstrated a left ventricular ejection fraction of % 60 with no wall motion abnormalities. Laboratory tests were within normal limits and his high sensitive troponin level was normal. We decided to perform coronary angiography because of new onset CCS class 2 angina.

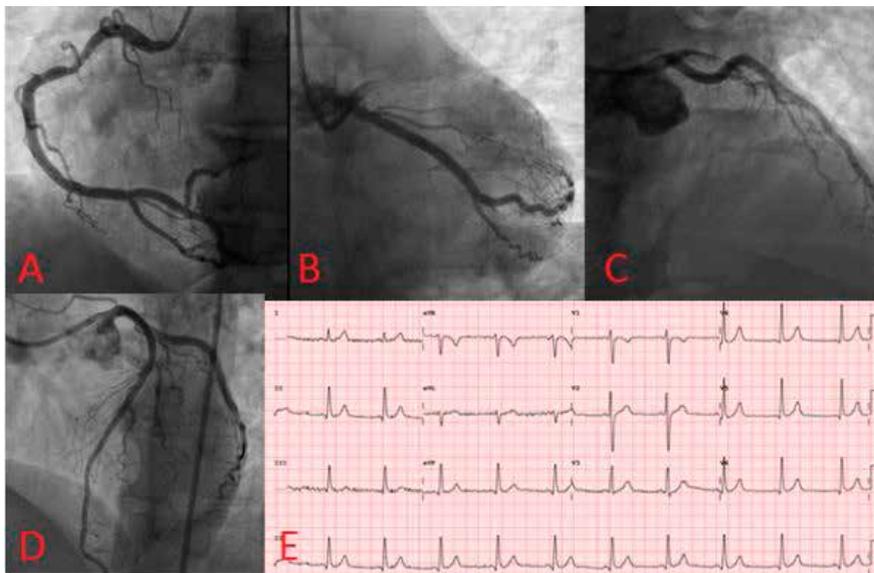
The coronary angiogram demonstrated total occlusion of proximal LAD with thrombosis and distal TIMI 0 flow. There were no significant vessel narrowing in right and circumflex coronary artery (A-B-C). Patient received glycoprotein IIb/IIIa inhibitor tirofiban (25 microgr/kg i.v. bolus over 3 min, followed by an infusion of 0.15 microgr/kg/min for 18 h) plus enoxaparin (1 mg/kg s.c b.i.d) with dual antiplatelet therapy. The control angiography performed next day and showed similar findings.

Given the proximal nature of the occlusion, percutaneous coronary intervention (PCI) was performed. The vessel was carefully wired with floppy guide wire. After multiple pre-dilatations, three drug eluting stents (2,75x18 mm,3,0x15 mm,3,0x18 mm) were deployed from distal to proximal LAD (D). The anginal symptoms were controlled and he was discharged next day. There was no remaining angina at 6th month outpatient control.

CONCLUSION: It is clear that COVID-19 pneumonia is characterized by severe endothelial dysfunction and thrombosis. As a result of the effects of COVID-19 on both thrombosis and myocardial damage, myocardial infarction is frequently seen in these patients. The presented case showed us that late occlusions in coronary vessels could be found in post COVID patients with neither ECG nor high sensitive troponin level changes.

Keywords: Acute coronary syndrome, Coranary angiography, Coronary artery disease, COVID_19

figure 1



A-B-C: patient's coronary aniiography
D: LAD after stents implantation
E: patient's ECG when he has typical angina

EP-006 CORONARY ARTERY DISEASE PRESENTING WITH DIGITAL ISCHEMIA

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Izmir Katip Celebi University Faculty of Medicine, Department of Cardiovascular Surgery

BACKGROUND: Digital ischemia is a painful and often disfiguring event. Such an ischemic condition often leads to tissue loss and can significantly affect the patient's quality of life. Digital ischemia can be secondary to a vasculopathy, vasculitis, embolic disease, trauma, or extrinsic vascular compression. In this case report, we present a patient who was admitted to the clinic with digital embolism and subsequently diagnosed with subclavian artery stenosis and underwent coronary artery bypass surgery.

CASE: A 68 year-old male ex-smoker patient with diabetes mellitus and hypertension was admitted to emergency department with cyanosis of 2.,3. and 4. distal phalanges of the fingers of left hand and difference between right and left arm blood pressures (right arm 142/88mmHg, left arm 102/56mmHg).70% left subclavian artery stenosis was detected in CT angiography. No pathology was found by transthoracic echocardiography. ECG was in sinus rhythm. Coronary angiogram revealed multi-vessel disease. Coronary artery bypass surgery was planned for the patient. He underwent 3 vessel-CABG surgery. After the operation,the patient was transferred to intensive care unit. Peripheral artery disease was treated medically.The patient was discharged on 7th postoperative day without any complication and referred to the interventional radiology department for subclavian artery stenosis. No pathology was founded in the first month outpatient clinic control of the patient.

CONCLUSIONS: In our case, examination for coronary artery disease in a patient with peripheral artery disease prevented possible cardiac complications. Coexistence of peripheral and coronary artery disease should be kept in mind. We think that performing ECG and echocardiography for patients with peripheral artery disease; and pulse examination for patients with coronary artery disease -with Doppler USG if necessary- can be useful in preoperative evaluation.

Keywords: Arterial occlusive diseases, Coronary artery disease, Peripheral angiopathies

EP-007 RACIAL INEQUALITY IN THE DISTRIBUTION OF FUNDS IN HOSPITALIZATIONS FOR CARDIOVASCULAR DISEASES IN BRAZIL

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Objectives: Previous publications have already mentioned the existence of a racial disparity in health care around the globe, and since the origin of the COVID-19 pandemic, much has been said about structural racism in health systems. Based on these premises, the present study aims to evaluate the racial disparity in the investment of the Brazilian Health System during hospitalizations for the treatment of cardiovascular diseases.

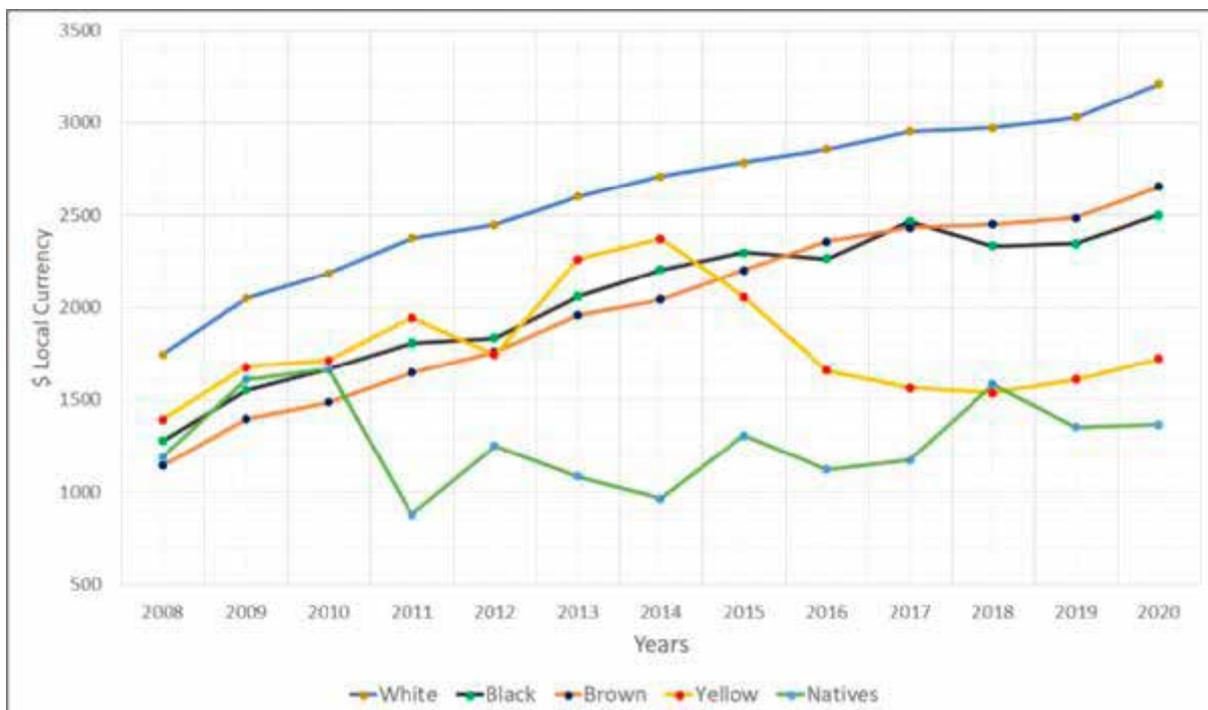
Methods: Data from medical centers participating in the Brazilian Health System were collected, and the inclusion criteria for the research was hospitalization due to cardiovascular diseases in the period from 2008 to 2020. Cases without racial identification of the patient were excluded. The variables that refer to the average amount spent on each hospitalized patient stratified into races (white, black, brown skin - "pardo" in Brazil -, orientals or native people) were analyzed. All data were collected from the public domain bank of the Informatics Department of the Brazilian Health System, previously anonymized, therefore, presenting no ethical conflicts.

Results: In all, 10,541,472 hospitalizations met the inclusion criteria and were analyzed. Comparing the average amount spent per hospitalization, we found that, in absolute values, it increased, but at a rate lower than inflation. From 2008 to 2020, investments rose 89.25%, while Brazilian inflation, according to the Brazilian Institute of Geography and Statistics, was 103.56%. Racially stratifying, the average amount spent in the white population group per patient hospitalized for cardiovascular disease was approximately US\$499.24 (\$2,608.83 in local currency), while in the natives it was US\$ 254.92 (\$1,332.13 in local currency), in the black group US\$395.24 (\$2,065.37 in local currency), in the brown skin US\$395.36 (\$2,065.99 in local currency) and the orientals US\$325.91 (\$1,703.08 in local currency). Thus, we see that the amount spent on white patients is higher than on natives, black, brown and orientals patients (95.84%, 26.31%, 26.27% and 53.18% higher, respectively). In all the years studied, the white people group presented the highest values of investments in treatment, while the group of native people was nine of thirteen times the one with the lowest investment, as we can see in the graph.

Conclusion: We demonstrate racial inequality in the allocation of investments for the treatment of cardiovascular diseases in the public health system in Brazil. The white people group received a significantly higher amount of money per hospitalization than the indigenous people, blacks, brown skin and orientals.

Keywords: Racial Inequality, Cardiovascular Diseases, Hospitalizations

Graph of values spent per hospitalization stratified by race.



Graph exposing financial resources spent by hospitalization in medical centers of the Brazilian Public Health System, stratified by race, in the period from 2008 to 2020.

EP-008 PERCUTANEOUS CORONARY INTERVENTION FOR TRUE BIFURCATION LESIONS: A SINGLE-CENTER EXPERIENCE

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Objective: Percutaneous coronary intervention (PCI) for true coronary bifurcation lesions is challenging. We aimed to describe procedural and clinical outcomes of true bifurcation PCI.

Methods: Single-center retrospective study of consecutive patients (pts) submitted to PCI for true bifurcation lesions (06/2018-06/2020). MACE comprised death, myocardial infarction (MI), stroke, restenosis and reintervention.

Results: During the study period, 1678 PCI were performed, of which 7% were for true bifurcation lesions (Medina x,x,1). 118 pts (mean age 66.4 ± 11.0 years; 74.6% male; 32.2% diabetic; mean left ventricular ejection fraction 49.8 ± 8.4%) were included.

Most pts (64.7%) were treated in the setting of an acute coronary syndrome (ACS): 26.7% ST-elevation MI, 31.9% non-ST-elevation MI, 6.0% unstable angina. 35.3% were treated for a chronic coronary syndrome. 24.8% of pts presented with 3-vessel disease and 11.1% had a left main (LM) lesion.

The most frequently diseased main branch was the left anterior descending artery: 21.2% (68), followed by the left circumflex: 17.8% (21), right coronary artery: 12.7% (15) and LM: 11.9% (14). In terms of bifurcation classification, lesions were mainly Medina 1,1,1 (71.2%), followed by 0,1,1 (13.6%); 1,0,1 (9.2%) and 0,0,1 (5.9%).

PCI was mainly performed via radial artery (73.7%). Mean number of stents: 1.4 ± 0.6. Lesions were predominantly treated with single stenting technique (80.3%). Double-stenting was used in 23 (19.7%) pts: TAP was the most used technique (10), followed by culotte (3), crush (2), double kissing crush (2) and T-stent (1). Proximal optimization technique and kissing balloon inflation were performed in 38.1% and 44.9% of cases, respectively. Rotational atherectomy was used in 10.4%. Ventricular support devices were used in 3 pts (1 Impella, 2 intra-aortic balloon pump).

Intracoronary imaging was used in 13 pts: intravascular ultrasound (IVUS) and optical coherence tomography (OCT) in 8 and 5 pts, respectively; its use was more frequent in cases of LM PCI (p=.002). OCT was associated with higher contrast doses (350 vs 224 ml, p<.001), with no increase in acute kidney injury incidence (p=.413). Fluoroscopic time (p=.684) and radiation dose (p=.916) did not differ.

Side branch occlusion occurred in 5 pts and iatrogenic coronary dissection in 7 pts. At a mean follow-up time of 17.3 ± 8.1 months, MACE and mortality rates were 13.3% and 6.9%. MACE occurred more frequently in pts with LM lesion (p<.001) and less often in cases where complete revascularization was achieved (p=.006).

Conclusion: True bifurcations represented a relatively small percentage of PCI lesions and were mainly performed in ACS setting. In line with literature, single stenting was the most used technique. MACE rate was globally low and was decreased by complete revascularization. Still, LM bifurcation PCI was associated with increased MACE.

Keywords: Percutaneous coronary intervention, Bifurcation lesions, Interventional Cardiology, Coronary artery disease

EP-009 INCIDENTAL FINDING OF CIRCUMFLEX ARTERY AGENESIS WITH MIGRATION OF ATRIAL SEPTAL OCCLUDER DEVICE AFTER AN ACUTE CORONARY SYNDROME:CASE REPORT

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Absence of circumflex coronary artery is a rare congenital anomaly, it is usually diagnosed incidentally on coronary angiography or coronary CT to rule out underlying coronary artery disease

In the other hand, Atrial septal defect is a common congenital heart defect, Percutaneous transcatheter closure become now the standard approach for ostium secundum type ASD closure. However, this procedure is not free of complications and device migration is the most common one.

In this case, a 55 year-old man was referred with acute coronary syndrome, that was successfully treated with thrombolysis therapy, than, an echocardiography was performed and showed a secundum atrial septal defect (ASD) with migration of the ASO in aortic arch confirmed by chest computed tomography.

A coronary CT scan was done and showed an agenesis of circumflex coronary artery with a superdominant right coronary artery that did not require lesion specific treatment. The patient underwent successful percutaneous ASD closure with the ASO device through a transfemoral arterial approach.

Coexistence of an ostium secundum type atrial septal defect and absence of circumflex coronary artery is exceptional, and to our knowledge, the present case is the first example of this combination.

Keywords: Migration of septal device occluder, circumflex artery agenesis, atrial septal defect

EP-010 KAWASAKI DISEASE DIAGNOSED BY ACUTE CORONARY SYNDROME

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Introduction: Coronary artery aneurysm (CAA) are rare and occurs in 0.3%-4.9% of patients undergoing coronary angiography. We present a case of coronary artery aneurysm due to undiagnosed Kawasaki disease.

Case Presentation: A 26 year old male patient admitted to emergency with headache and weakness. He had no chest pain, dyspnea or fever and doesn't have any comorbidities except smoking. He has family history of coronary artery disease.

Laboratory results were as follows; WBC:5.1 10³/uL, Plt: 152 10³/uL, Hb:13.0g/L, D-dimer: 2.1 ug/mL, CRP:133 mg/L, Hs-Troponin: 1481 ng/L, CKMB:1.3 ng/mL, Creat:0.92 mg/dL, AST:88 U/L, ALT:110 U/L, LDL:107 mg/dL, Procalcitonin: 0.49 ng/mL, ESR:49 mm/h but the blood ve and urine cultures were negative. Three times Covid-PCR test was negative and thorax CT were normal. In the follow-up troponin and acute phase reactants spontaneously regressed. Patient's electrocardiogram was sinus rhythm 88 bpm, had no abnormalities. (Figure 1) The echocardiography showed us left ventricular ejection fraction was 65% global, mild mitral regurgitation.

The patient was admitted to the ICU with a preliminary diagnosis of myocarditis. After the coronary CT angiography showed aneurysms, coronary angiography was performed. (Figure 1)

Angiography showed us giant aneurysms in coronary arteries. (Figure 2). In terms of etiology; ANA, ANCA, ENA, Hepatite serology were tested and reported negative. Cervical, abdominal doppler ultrasonography, brain CT angiography and MRI were performed and not showed any vascular aneurysm.

When his history questioned again, learned that he had a fever of 40 degrees lasting 4 days and when he was 5 years old. The patient was diagnosed with coronary aneurysm due to undiagnosed Kawasaki disease.

Kawasaki disease is a vasculitis of medium-size arteries it results in a fever lasts for more than five days and mainly affects children under 5 years of age. Coronary artery lesions develop in 15%–25% of patients with untreated KD.

Myocardial perfusion scintigraphy showed ischemia at the apex but the cardiovascular stress test was negative. After council with cardiovascular surgery treatment options were presented to the patient and medical follow-up was preferred by him. We discharged patient with aspirin, statin, beta-blocker, angiotensin converting enzyme inhibitor, rivaroxaban and recommended quit smoking to reduce risk factors. The patient has been followed six months without any symptoms.

Discussion: The options for managing coronary aneurysms are still present a therapeutic challenge to cardiologists because there are no randomized trials. Thrombosis is the main causes of myocardial infarction in CAAs. Recent studies suggested a possible advantage of anticoagulation in patients with CAAs and acute coronary syndrome.

This case is an example of Kawasaki disease manifest itself with acute coronary syndrome years later. Kawasaki related coronary artery aneurysm is rare and patient tailored treatment should be considered.

Keywords: Acute Coronary Syndrome, Coronary Aneurysm, Kawasaki Disease

Figure 1

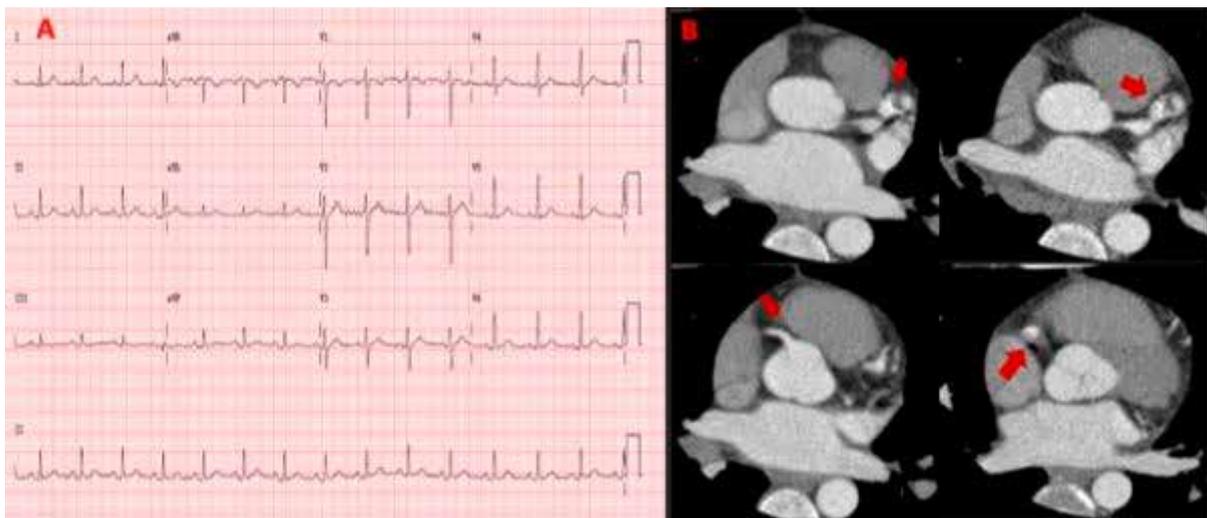


Figure 1:
A: Patient's Electrocardiogram **B:** Coronary CT angiography/ Red arrows : CAAs

Figure 2

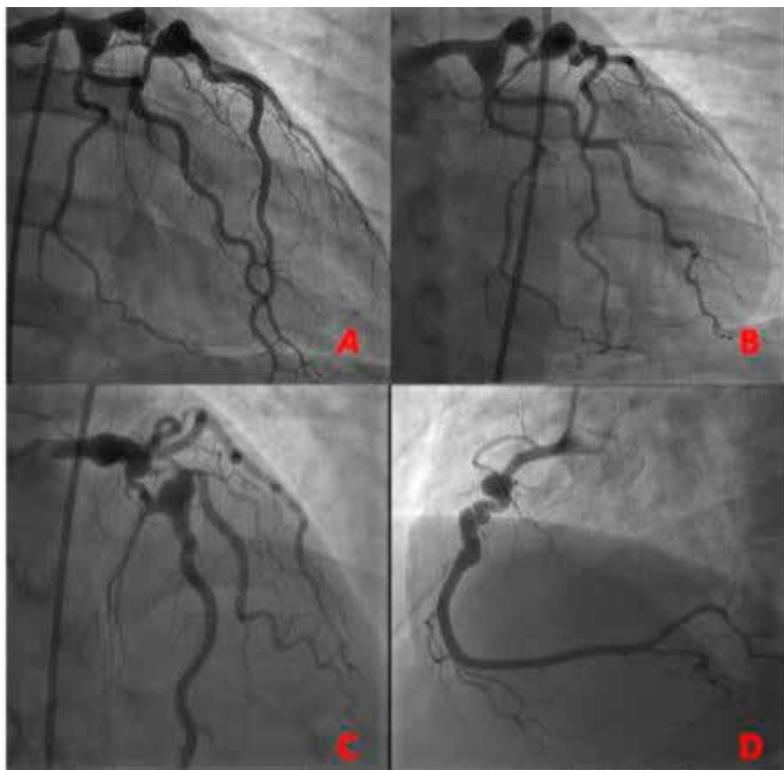


Figure2:
A,B,C:
LAD: three
consequential
giant aneurysms
in the proximal,
in the proximal
and mid region
%60-70 stenosis ,
CX: osteal
aneurysmatic
distal normal
D: RCA: proximal
and mid diffuse
aneurysma and
interaneurysm
tortiosity.

EP-011 A CASE OF CORONARY AIR EMBOLISM DURING ANGIOGRAPHY

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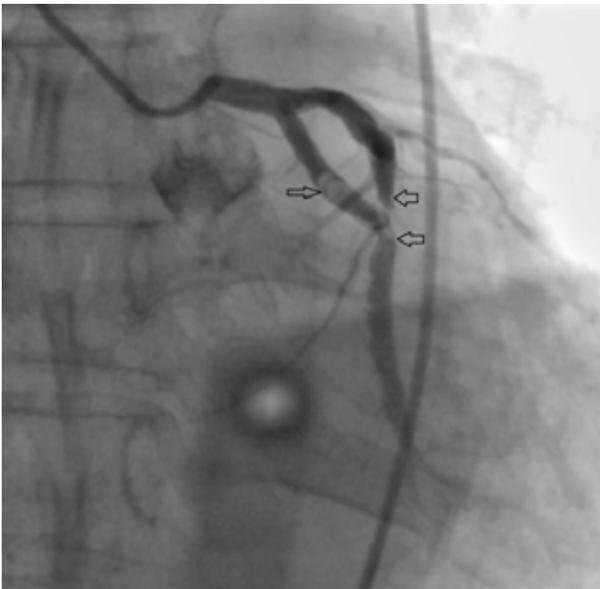
Introduction: Coronary air embolism is one of the rarely but serious complication of coronary angiography. Despite improved device and procedural safety, complications associated with PCI continue to be encountered. We report a case of catastrophic iatrogenic air embolism observed during diagnostic coronary angiography.

Case: A 62-year-old, hypertensive male patient with acute Type-A dissection was planned for coronary angiography. A standart 6F cathater, engaged at the left main coronary artery. At coronary angiography, the flow was blocked due to the full bubble from the LAD mid region and a dense bubble was observed in the Cx artery (Figure 1). When bubble air was observed in coronary arteries, patient had suddenly chest pain. In addition, the patient's hemodynamics changed rapidly with hypotension (BP:50/30) and bradycardia, and loss of consciousness. The patient was given 100% oxygen. Also approximately 200 ml force ful injection heparinized saline was given through the catheter in to the left main coronary artery. But cant provided coronary flow TIMI III. Afterwards, 0.014 floppy guidewire was advanced to both LAD and Cx. Guide wire routed back and forth, causing bubbles to suffer minör physical disintegration. In control coronary injection after 5 minute, there was no air bubbles were seen. There were not detected any lesions in coronary arteries. The chest pain of the patient completely passed. Patient's arterial tension and pulse were normalised. The further angiographic investigation of patient was uneventful and coronary artery were normal (Figure 2).

Discussion and Conclusion: Coronary air embolism is rare yet preventable complication of coronary angiography. The incidence of this complication depends on the operators experience and awareness. There is no established consensus on the adequate management of air embolism and its complications. The principal management is prevention. Operators should prepare the systems very well before the procedure, aspirate the catheters adequately, and make sure that all the connections are tightened and that manifold is always held in upright position. The clinical signs and management of coronary air embolism depend on the amount of air delivered to the coronary vessels. Various management strategies aimed at restoring blood flow in the affected coronary by resolving the air embolism are 100% oxygen, forceful injection of saline or blood aiming to dissipate the intracoronary air, air aspiration using thrombectomy catheters and use of vasodilators to address coronary slow flow. In our case, we administered forceful injection of heparinized saline. Also with the help of a guidewire and crushed with manual traction movements directed back and for the, and we ensured that the bubbles caused small physical fragmentation in LAD. Blood flow in LAD was restored. Massive coronary air embolism is a threatening complication and can be resolved with bolus saline infusion and guidewire manipulation.

Keywords: Air embolism, Coronary angiography, Saline injection

Figure 1



Air is observed in the LAD and CX arteries

Figure 2



back and forth movement of the guide wire within the LAD artery

EP-012 A RARE CASE OF CARDIAC LYMPHOMA PRESENTING AS PERICARDIAL EFFUSION AND THIRD DEGREE AV BLOK

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Abstract: Pericardial effusion is a rare presenting feature of Non-Hodgkin's lymphoma. A 75-year-old woman presented with dyspnea and was diagnosed with pericardial effusion and third-degree atrioventricular (AV) block. Transthoracic echocardiography (TTE) revealed a large pericardial effusion with swinging heart without tamponade physiology. The definitive diagnosis of Non-Hodgkin's B-Cell Lymphoma is established by postmortem pericardial biopsy specimen showing extensive infiltration of B-Cell lymphocytes to visceral pericardium.

Case Report: A 75-year-old female patient admitted to emergency department with dyspnea and tiredness. The patient had a history of coronary artery disease and hypertension. On admission, her blood pressure was 130/75 mmHg without a pulsus paradoxus, heart rate was 40 beats/min and oxygen saturation was 91%. Her physical examination was notable for mild pretibial edema and diminished heart sounds. ECG revealed third-degree AV block with low voltage in the limb leads. A TTE demonstrated 2 cm pericardial effusion with swinging heart without tamponade physiology, a left ventricular ejection fraction of 55% and mild to moderate mitral and tricuspid regurgitation.

Laboratory tests performed on admission showed polymorphonuclear dominate leukocytosis with mild anemia, elevated lactate dehydrogenase (LDH:1535.00 U/L) and brain natriuretic peptide levels. Renal, liver and thyroid functions were within normal limits. Blood cultures and viral serology didn't support an active infection.

A temporary pacemaker was inserted for AV block through transjugular approach and she was taken to the catheterization laboratory for pericardiocentesis. Biochemistry was consistent with an exudate. Laboratory findings of pericardial fluid was remarkable for elevated LDH (5567.00 U/L) and adenosine deaminase activity (ADA:156.30 U/L) and neutrophil predominancy with 74.4%. Microbiological cultures were negative. Computed tomography (CT) of the chest and abdomen showed no evidence of malignancy. Since pericardial effusion and third-degree AV block are seen together infrequently. Tuberculin skin test was negative. Positron emission tomography (PET) demonstrated extensive fluoro-deoxyglucose (FDG) uptake over pericardium and myocardium. Patient gradually developed dyspnea irresponsive to loop diuretics and second CT of the chest showed bilateral pleural effusions. Repeated TTE demonstrated diastolic dysfunction with constrictive filling pattern. Meanwhile clinical condition of the patient deteriorated gradually and she referred to cardiovascular surgery department for pericardiectomy for diagnosis. Unfortunately patient died one day after surgery. Her postmortem pericardial biopsy specimen showed extensive Cd20, Pax5, Mum1, Bcl2, Bcl6 And C-Myc positive B-cells demonstrating B-cell lymphoma.

Conclusion: Our case shows the Non-Hodgkin's lymphoma must be thought in the differential diagnosis of pericardial effusions especially with high LDH

Keywords: Non-Hodgkins lymphoma, Pericardiocentesis, Third-degree AV block

Figure 1

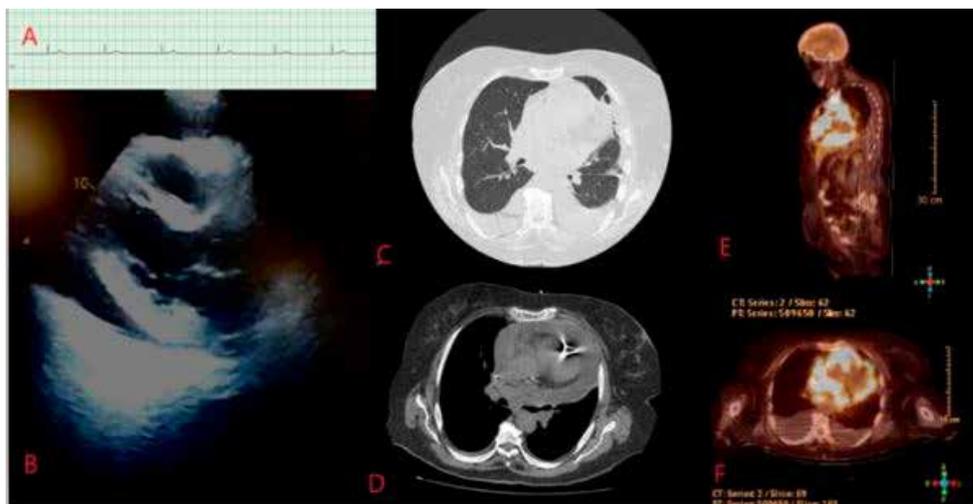


Figure 1:
 A: Showing third degree AV blok
 B: Parasternal Long Axis View showing Pericardial Effusion
 C: Second CT after Pericardiocentesis showing pleural effusion
 D: CT showing Pericardial Effusion
 E-F : PET CT Scan showing increased pericardial and myocardial FDG uptake

EP-013 STENT-IN-STENT TECHNIQUE FOR PROVIDE VASCULAR INTEGRITY IN PATIENT COMPLICATED WITH PROTRUDING SELF EXPANDABLE STENTFatih Yilmaz¹, Semih Kalkan²¹Kartal Kosuyolu Yuksek Ihtisas Egitim ve Arastirma Hastanesi²Erzurum Bolge Egitim ve Arastirma Hastanesi**Objective:** To report a rare complication of SUPERA® stent deployment and its management.**Case:** A 55-year-old male patient visited our hospital with left leg claudication (Fontaine stage III, Rutherford category 2.) over three months. After all examinations, endovascular treatment of the left superficial femoral artery was arranged. We have deployed the Supera™ (Abbott Vascular Inc) stent under fluoroscopy as required by the stent instructions for use. However, the self-expanded stent protruded into the quadriceps muscle from the intervention site. Unintended site implantation is always a common risk with self-expandable stents. We have managed the complication with an endovascular approach via deploying a covered stent into the Supera® system.**Conclusion:** Elongation and unintended site implantation are common complications of the Supera® stents. The case presented below demonstrates stent-in-stent procedure with covered stents may block blood extravasation and provide vascular integrity.**Keywords:** peripheral, vascular, disease, endovascular, intervention

EP-014 FREQUENCY AND TYPES OF ANTIHYPERTENSIVE MEDICATIONS USED IN MOTHERS PREGNANCIES OF CHILDREN WITH CONGENITAL HEART DISEASE

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Objective: To evaluate the use of antihypertensive drugs according to their known fetal risk in pregnancies of patients with congenital heart disease (CHD).

Method: The sample consisted of 198 patients who were consecutively evaluated during their first hospitalization in a cardiac intensive care unit (ICU). The patients underwent a protocol that evaluated the use of antihypertensive drugs during their pregnancy. These drugs were divided according to their fetal risk, following the classification proposed by the Food and Drug Administration (FDA). We considered high risk when the child was exposed to drugs of classes D and X.

Results: Of the total sample, 103 patients (52%) were male, with ages ranging from 1 to 4934 days. The interview for data collection was carried out with the patient's mother in 48.5% of cases and with both parents in 42.9%. The most frequently observed CHDs were ventricular septal defects (DSVs) (16.1%) and atrial septal defects (ASDs) (16.1%). One hundred twenty-four patients (62.6%) were exposed to at least one medication during pregnancy. Eighteen mothers (9%) reported the use of antihypertensive drugs during pregnancy, which included methyldopa (n=7 – 3.5%), enalapril (n=4 – 2%), hydrochlorothiazide (n=3 – 1.5%), verapamil (n=2 – 1%) and propranolol (n=2 – 1%). As for the use of enalapril (n=4), two pregnant women did it in the first trimester of pregnancy and two in the second. As for propranolol, one did it in the first trimester and the other in the third. According to the FDA classification, 3 cases (16.7%) would consist of medications belonging to class B, 12 (66.7%) to class C and 3 (16.6%) to class D (2 cases of enalapril used in the second trimester and 1 of propranolol in the third trimester of pregnancy).

Conclusions: CHDs are the most common birth defects seen at birth and represent a real public health problem. We found the use of antihypertensives belonging to class D among the pregnant women in our study, suggesting that these exposures may even play a role in the origin of the detected CHDs. This highlights the importance of preventive measures and education of health professionals and patients regarding the use of these medications during pregnancy.

Keywords: antihypertensive medications, congenital heart disease, fetal risk

EP-015 CONGENITAL HEART DISEASES AND THEIR ASSOCIATION WITH GASTROINTESTINAL TRACT MALFORMATIONS

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Objective: to determine the frequency and types of gastrointestinal tract malformations (GTI) in a sample of patients with congenital heart disease (CHD).

Method: patients with CHD hospitalized for the first time in a cardiac intensive care unit of a pediatric referral hospital in Southern Brazil were evaluated. Clinical data were obtained by completing a standard protocol.

Results: the sample consisted of 343 patients, 182 (53.1%) male, ages ranging from 1 day to 14 years and 6 months (60.1% <1 year). Malformations in the GTI were evidenced in 6 patients (1.7%) and consisted of esophageal atresia (n=2), duodenal stenosis (n=1), multiseptated gallbladder (n=1), anteriorized anus (n=1), and imperforate anus (n=1). The most observed CHD among patients with GTI malformations were septal defects (n=3), especially ventricular septal defects (n=2). Four patients were syndromic, and chromosomal alterations were observed in 5 patients.

Conclusions: extracardiac malformations (EMs) associated with congenital heart disease (CHD) can increase the risk of morbidity and mortality in the child, often making surgical intervention risky. This association between EMs and CHD may involve defects in different systems or tracts, such as the GTI. Based on the information on the presence of an associated GTI malformation, health professionals can carry out a more detailed and targeted assessment of patients with CHD, aiming at better management of these patients and preventing future complications, mainly related to their prognosis.

Keywords: congenital heart diseases, gastrointestinal tract, malformations

EP-016 REDUCING STROKE INCIDENCE IN TAVR: SHAKE IT OFF

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Background: Transcatheter aortic valve replacement (TAVR); is minimally invasive option for patients with severe aortic stenosis patients and moderate /high risk surgical aortic replacement(AVR). Stroke is the main cause of mortality and disability and also main complication in TAVI.

Stokes main sources are thrombus and other vascular debris in vascular system.

We wanted the present a method we used, to reduce the risk of stroke, especially patients with thrombus in any vascular lumen on CT angiography. This case report is typical example of it.

Case Report: A 73 year old male patient applies to our clinic with dyspnea, reduced effort capacity. History of RCA stent implantation in 2007. In preoperative coronary angiography no restenosis were observed. The patient has Hypertension, Tip 2 Diyabetes Mellitus comorbidities. Transthoracic echocardiography reported as; left ventricular ejection fraction was 50% global, AVmax: 4.4m / sec, 80 / 52mmhg gradient, aortic valve area: 0.65cm² mild aortic regurgitation, mild mitral and tricuspid regurgitation were reported. Laborutavar values; Creatinine: 1.53, BUN: 17.3, GFR: 44, CRP: 3.7, K: 4.32, BNP: 1409, Hb: 12.5, Wbc: 7.4, Plt: 167000, LDL: 95.

Widespread calcification and thrombus were observed in the right and left iliac arteries, abdominal and descending aorta in preoperative TAVI-CT. Transcatheter aortic valve replacement was planned with 34mm EvolutR Core-Valve for the patient. Procedure was started over the right femoral artery, device was taken forward on descending aorta with it's in-line sheath without any friction. At the descending aorta before the left subclavian artery we shook the TAVI catheter swinging back and forth and right-left and brushing the surface of the device with the vessel wall.

This maneuver may cause the thrombus and other materials adhering to the device surface to be directed to the periphery together with the blood flow direction, and thus it will not go to the cerebral flow.

Discussion: In metanalysis the incidence of any stroke was higher with BEV (Balloon expandable valves) than SEV (Self expandable valves) (OR 1.51, 95% CI 1.01 to 2.26). This may cause by thrombi carried out by the balloon from the periphery and combining it with device in descending aorta which creates an extra source for stroke. The use of anti-stroke devices reduces this risk by only 39% and brings vascular complications. High stroke risk in TAVR may caused by the device being rubbing through the entire aorta and collecting thrombus and other materials from the periphery.

As Dokuz Eylül University Hospital our TAVR series of 250 patients with moderate risk, the major stroke incidence in the first 30 days was %0.4 and %0.8 in the late period. We recommend and applied to our patients the maneuver (especially patients with high thrombus burden on TAVI-CT), which does not require an additional device and has a low risk of complications.

Keywords: Stoke in TAVR, Transcatheter Aortic Valve Replacement, TAVR Complications

Figure 1

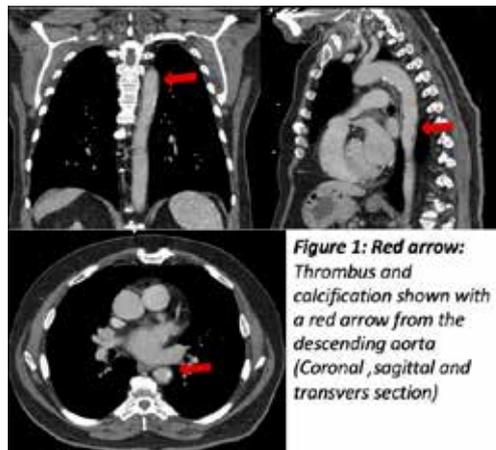
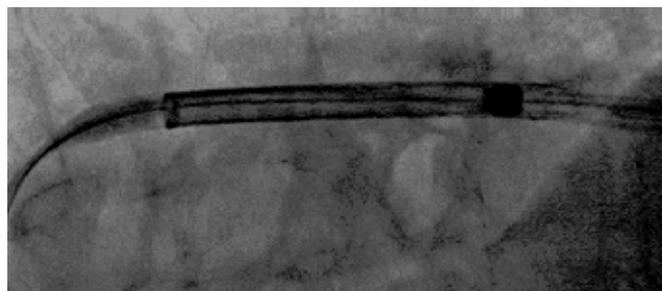


Figure 2



Shaking Maneuver

EP-017 ASSESSMENT OF PLASMA CONCENTRATION OF N-TERMINAL FRAGMENT OF PRO-BRAIN NATRIURETIC PEPTIDE AS A CARDIOTOXIC BIOMARKER IN BREAST CANCER RECEIVING ANTHRACYCLINE-BASED CHEMOTHERAPY

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Background: Breast cancer is the most common cancer in women all over the world. Anthracyclines, which are frequently used in many stages of breast cancer treatment, have well known side effects. The most important side effect of anthracycline chemotherapy (CT) regimens, which are widely used in breast cancer patients, is cardiotoxicity, which limits its use in therapeutic doses and is important for its early detection.

Aim: To assess the relationship between the early cardiotoxicity of anthracycline-containing schemes in oncology patients receiving CT and the concentration of a biomarker in the blood, which is a N-terminal fragment of the pro-brain natriuretic peptide (NT-pro BNP).

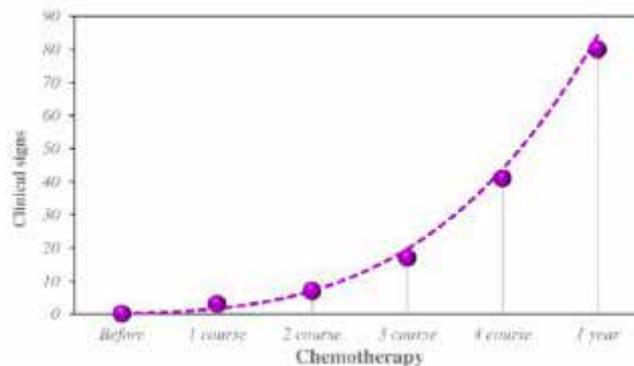
Material-Methods: 60 patients were included in the study. Inclusion criteria: Female patients aged 20-65 years who are underwent chemotherapy due to the diagnosis of breast cancer and are practically healthy from a cardiological viewpoint. Exclusion criteria: patients with structural myocardial disease, patients undergoing lifelong chemotherapy and patients with chronic renal failure. Criteria for evaluating effectiveness: a) the presence of clinical signs b) systolic function of the left ventricle (LVEFsimpson); c) diastolic function of the left ventricle; d) morphology and function of the heart valves by transthoracic echocardiography (TTE) e) the concentration of the biomarker NT-proBNP in the blood was determined. All patients underwent TTE before CT, during the course, and 1 year later to determine the concentration of the biomarker NT-proBNP in the blood.

Results: In the TTE examination: a) the left ventricular ejection fraction did not decrease by more than 15% from the lower limit of the basal value; b) diastolic dysfunction was noted in the assessment of diastolic function of the left ventricle with e' septal <7, E/e' > 15, LAVI >34 ml/m²; c) no severe change in heart valve function was noted, but mild to moderate change was noted. Clinical signs increased in those with NT-pro BNP > 300 pg / ml, and appeared in those with NT-pro BNP > 125 <300 pg / ml. And this suggests that there is a positive correlation between the clinical signs of heart failure and the level of the biomarker NT-proBNP in the blood, regardless of LVEF. There was a statistically significant increase in the value of NT-proBNP in the blood of patients after CT.

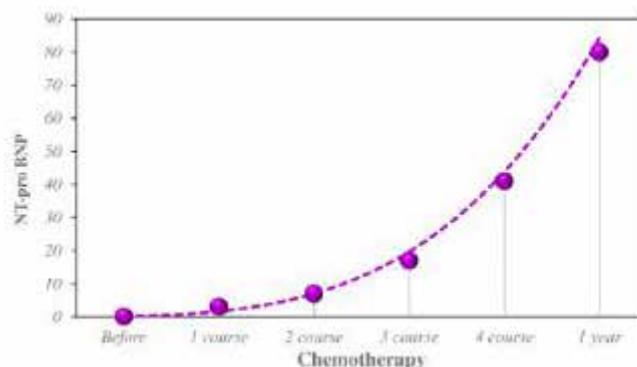
Conclusion: The determination of the NT-pro BNP biomarker in the blood is significant for the assessment of currently existing pathology in this area, as it is a sensitive method for the early assessment of anthracycline-related heart failure.

Keywords: chemotherapy, N-terminal fragment of pro-brain natriuretic peptide, cardiotoxicity

Regression analysis between the number of Chemotherapy courses and Clinical signs



Regression analysis between the number of Chemotherapy courses and NT-pro BNP



EP-018 LEFT SIDES HEMOTHORAX REVEALING THORACIC AORTIC ANEURYSM RUPTURE

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Thoracic aortic aneurysm (TAA) is a potentially fatal disease. It can cause life-threatening complications such as aortic dissection or rupture.

We report the case of a 65-year-old man admitted to our cardiology department complaining of rapidly progressive non-radiating chest pain with dyspnea. An initial examination showed a blood pressure of 140/90 mmHg bilaterally, heart rate of 90 beats/min. His cardiac enzyme levels were slightly positive, and an electrocardiogram showed sinus tachycardia with inverted T waves in anterior leads. A chest radiograph obtained on admission showed an enlargement of the mediastinum and a left pleural effusion. An acute aortic syndrome was suspected; and a chest computed tomography revealed an aneurysm of the ascending aorta, the arch, and the descending aorta ruptured in the pleural cavity causing a left abundant hemothorax. Urgently, the patient was referred to the cardiovascular surgery department. Unfortunately, he died during the operation.

TAA can be a part of complex genetic syndromes, including Marfan, Ehlers-Danlos, Loeys-Dietz, and Turner syndromes. However, increasing numbers of genetic loci linked to non-syndromic TAAs are being identified. Therefore, physicians should be cognizant of screening family members of TAA patients. Rupture of the thoracic aorta into the pleural cavity has been regularly described in the literature. For anatomical reasons, the ruptured descending thoracic aorta bleeds preferentially into the left pleural cavity. Nevertheless, 5 to 10% of these aneurysms may rupture into the right side. The radiologic assessment of patients with a suspected aortic rupture is a cornerstone of the diagnostic process. A chest radiograph reveals pathologic findings such as abnormal aortic contour, widened mediastinum, "calcium signs" or pleural effusion in 80 to 85 percent of cases. Currently, a contrast-enhanced CT scan is the gold standard for the diagnosis of patients with suspected dissection or rupture. According to current international guidelines, open repair remains the method of choice in ruptured ascending and aortic arch TAA management, whereas thoracic endovascular aortic repair has become the gold standard for ruptured descending TAA.

Keywords: hemothorax, aortic aneurysm, rupture

CT scan showing Aortic Aneurysm rupture



EP-019 CONOTRUNCAL CONGENITAL HEART DEFECTS AND 22Q11 DELETION SYNDROME (VELOCARDIOFACIAL/DIGEORGE SYNDROME)

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Objective: to verify the frequency of the 22q11.2 deletion syndrome (22q11DS) among patients with conotruncal congenital heart defects (CHDs).

Method: the sample consisted of a prospective and consecutive cohort of patients in their first hospitalization in the cardiological intensive care unit (ICU) of the Santo Antônio Child Hospital, during the period of 1 year. For each patient, an evaluation sheet was completed, with clinical data collection, and performance of high resolution karyotype and 22q11.2 microdeletion research by fluorescent in situ hybridization (FISH). Conotruncal defect classification was performed by a collaborative cardiologist based on the results of echocardiographies and catheterisms, as well as surgical descriptions.

Results: from the patients with CHD hospitalized for the first time during the study period, 52 (25.1%) had a conotruncal defect. Thirty-two were male (61.5%) and their ages ranged from 1 day to 10 years (48% < 1 month). The main reason for ICU admission was to undergo cardiac surgery (76.9%). The most frequently CHD observed was tetralogy of Fallot (TOF) (40.4%). There were no cases of aortic arch interruption. As for the karyotypic analysis, alterations were observed in 5 patients (9.6%); however, none of them had the 22q11.2 deletion. The analysis using the FISH technique could be successfully performed in 51 patients, and the 22q11 microdeletion was identified in 2 cases (3.9%) (both with TOF). Conclusions: heart malformations represent an important public health problem. Although its etiology is still little understood, SD22Q11 stands out among the known causes. The frequency of the syndrome verified in our study was similar to those studies that found values ranging from 4 to 15% and different from others that detected results between 17 and 48%. These differences appear to be related, especially, with the mode of selection adopted in the studies. The identification of these patients is fundamental for their adequate management and genetic counseling.

Keywords: Genetics, Velocardiofacial, Syndrome

EP-020 CARRIERS OF CHROMOSOMAL ANOMALIES IN SAMPLES OF PATIENTS WITH CONGENITAL HEART DISEASE: ROLE OF DYSMORPHOLOGICAL PHYSICAL EXAMINATION IN THEIR IDENTIFICATION

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Objective: Cross-sectional and observational study, which aims to verify whether the syndromic aspect can be used as a predictor of the presence of chromosomal abnormalities (CAs) among patients with congenital heart disease (CHD).

Method: The sample consisted of patients with CHD hospitalized for the first time in a cardiac and pediatric intensive care unit of a reference hospital in Southern Brazil. The patients were allocated prospectively and consecutively. They were classified as syndromic or not by a single clinical geneticist, based on the dysmorphic findings observed only on physical examination. The sensitivity and specificity of this approach was calculated. All patients underwent to high resolution karyotype and fluorescent in situ hybridization (FISH) for 22q11 microdeletion.

Results: The sample consisted of 198 patients, 103 male (52%), ages ranging from 1 to 4934 days (57% with <1 year). ACs were observed in 32 patients (16%): 23 cases of Down syndrome, 2 of Edwards syndrome, 1 of triple X, 1 of 17p duplication, 1 of add(18p) and 4 of 22q11 microdeletion. Of the 198 patients, 61 (31%) were classified as syndromic, and from these, 28 (46%) had an AC. ACS observed among non-syndromic individuals (3%) consisted of triple X and 3 of the 4 cases (75%) of 22q11 microdeletion. The sensitivity of this approach was 88% and the specificity 80%.

Conclusions: Genetic assessment through physical examination plays an important role in identifying CA carriers, which has implications for the management and genetic counseling of these CHD patients and their families.

Keywords: Congenital heart disease, Genetics, Syndromic aspect

EP-021 IMPACT OF COVID-19 PANDEMIC ON DISEASES OF THE CIRCULATORY SYSTEM IN BRAZIL

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Objectives: The COVID-19 pandemic has had a brutal impact on global health. In addition to the effects directly related to illness caused by the virus, it is important to study the consequences for the treatment of other diseases during a pandemic. In this sense, this study aims to evaluate, through the information provided by the Unified Health System, the effects of the pandemic on the treatment and mortality of diseases of the circulatory system.

Methods: Use of the national database DATASUS. The information was previously anonymized, therefore, without ethical conflicts. The data collected cover the period from six months before the pandemic hits the country (September 2019 to February 2020) until the corresponding period of the year during the pandemic (September 2020 to February 2021). The data shows the information about hospitalizations, mortality rate, and the number of deaths from diseases of the circulatory system.

Results: A total of 1,077,817 hospitalizations for diseases of the circulatory system were analyzed. Comparing the values obtained in both periods, a decrease in the number of hospitalizations associated with an increase in mortality can be observed. From the period before the pandemic, there were 593,861 hospital admissions. During the pandemic, there were 483,956 admissions, a decrease of 22,227%. Mortality increases from an average of 8.22 (from September 2019 to February 2020) to 9.46 (from September 2020 to February 2020), an 15.085% growth. It is possible to observe the result of lower demand of patients with cardiovascular diseases to be treated and hospitalized and its impact on the increase in mortality.

Conclusion: It was clear that during the covid-19 pandemic there were fewer hospitalizations due to circulatory system diseases. This fact can be generated because of the lower demand for medical care since patients avoid exposure to the virus in the hospital environment. This reality offers a delay and a consequent evolution of circulatory diseases to serious conditions. Another reason is the proportionally lower demand for mild and moderate cases concerning severe cases.

Keywords: Circulatory System Diseases, Brazil, Covid-19 Pandemic

EP-022 PERCUTANEOUS CORONARY INTERVENTION IN OCTOGENARIANS WITH ACUTE CORONARY SYNDROME

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Introduction: Percutaneous coronary intervention (PCI) are increasingly used in daily clinical practice in elderly patients with acute coronary syndrome (ACS) despite limited evidence. This study assessed the predictive factors of Major Adverse Cardiac and Cerebral Events (MACE) in this group of patients during follow-up.

Methods: We enrolled 103 patients aged ≥ 80 years admitted to Cardiology department of Charles Nicole's hospital with ACS from January 2016 to December 2018.

Results: The mean age of the patients was 82 years. 61,2% were female.

Arterial hypertension was the most prevalent cardiovascular risk factor (54,4%).

Sixty eight percent (68% had renal impairment (mean eGFR 55mL/min/m²).

53,4% were hospitalized for NSTEMI. Multivessel disease was noticed in 65% of patients. Among them, 92 had PCI.

During the first year of follow-up, there were 11,6% deaths and 37,5% MACE.

By using multivariate logistic regression model following factors were identified as independent predictors of MACE:

Incomplete revascularization ($p < 0,001$) (OR 22, 95% CI 5.23–95,1)

LEF $< 45\%$ ($p = 0,004$) (OR 9.32, 95% CI 2.01–43.02).

Discussion: Predicting risk in the elderly may be problematic given their highly variable health, social and cognitive status. Age is an independent risk factor for adverse outcomes in many conditions and is usually included in risk scores.

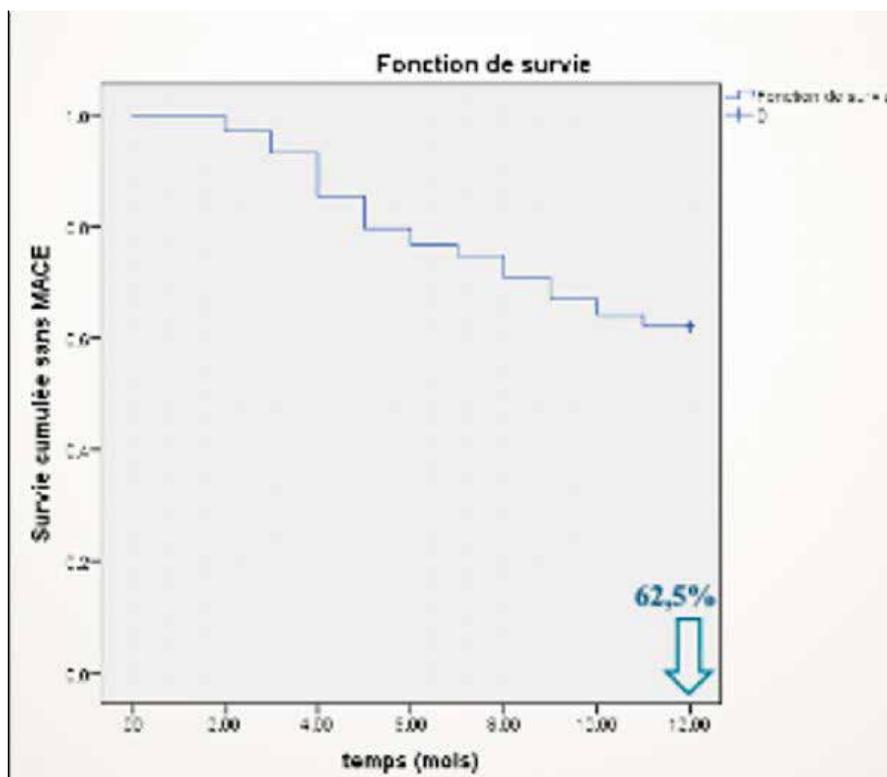
CAD in elderly patients is more extensive than in younger patients with a higher prevalence of three-vessel and left main CAD; nevertheless, two reasons make these patients less likely to undergo PCI: lower procedure success rates; and worse short-term prognosis.

Conclusion: In elderly, frailty and the complexity of coronary lesions seems to be associated with poor prognosis.

Invasive treatment seems to yield better outcomes for this group of patients.

Keywords: octagenarians, PCI, acute coronary syndrome

Kaplan-Meier survival curve



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