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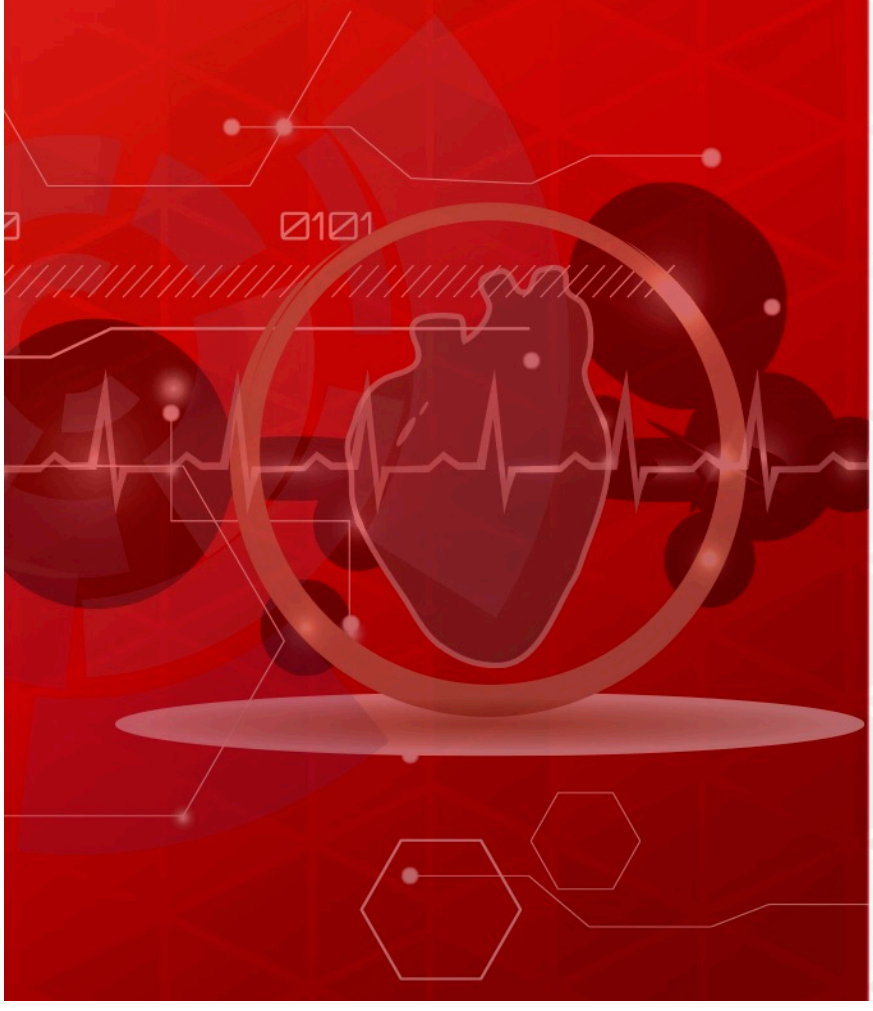

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Short communication

A comparison of drug-eluting stent versus balloon angioplasty in patients with bare-metal stent instent restenosis: 5 year outcomes☆

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ABSTRACT

Objectives: The objective of the present study was to compare the long term outcomes of balloon angioplasty (BA) versus drug-eluting stents (DES) in bare-metal stent in-stent restenosis (BMS-ISR).
Background: Coronary in-stent restenosis (ISR) remains a significant clinical problem. Long term results after management of ISR may help improve treatment strategies.
Methods: We assessed 5-year clinical outcomes in cohort of 269 patients with BMS-ISR treated with DES (n = 154) and BA (n = 115) between June 2007 and January 2010 at our institution.
Results: Clinical and demographic characteristics were similar for both groups. Mehran classification was used to classify ISR lesions. BA were used predominantly in classes I and II, whereas classes III and IV were treated with DES ($p < 0.0001$). Percentages of major adverse cardiovascular events (MACE) including death, myocardial infarction (MI) and target vessel revascularization (TVR) for 4.37 ± 1.1 years were 50.4% and 31.8% for the BA and DES groups, respectively ($p = 0.002$). Although patients in the BA group had significantly higher rates of recurrent angina (42.6% vs. 27.3%, $p = 0.009$) and TVR (37.4% vs. 20.8%, $p = 0.003$), MI (6.1% vs. 5.2%, $p = 0.752$) and cardiac death (21.7% vs. 16.2%, $p = 0.251$) were similar in both groups. MACE-free 1-year survival and 5-year survival rates were significantly higher in DES group compared to BA group (1 year survival: 91.6% vs. 71.3 $p < 0.001$, and 5 year survival: 68.2% vs. 49.6%, $p < 0.0001$, respectively).
Conclusions: Although DES were more frequently used in to treat complicated lesions in patients with ISR, follow-up MACE rates were significantly lower and MACE-free survival was significantly better in the DES treated patients.

Introduction

Development of bare metal stents (BMS) has become a major advancement in the treatment of coronary artery disease. BMS reduce restenosis rates by attenuating arterial recoil and contraction as compared to balloon angioplasty. However, in-stent restenosis (ISR) still occurs in approximately 10–20% of cases.[1] Despite high rates of restenosis, BMS are widely used for treating coronary artery disease.[2] Treatment of ISR remains a major challenge for clinicians. There are many treatment options for patients having ISR like recurrent balloon angioplasty (BA), drug-eluting stents (DES) or BMS, cutting balloon angioplasty, directional coronary atherectomy, rotational coronary atherectomy and vascular brachytherapy.[3–6] Although vascular brachytherapy is an effective treatment of ISR, it requires additional personnel, training and equipment. BA may be preferred in patients with contraindications for dual antiplatelet therapy (DAPT). Weintraub et al. showed that restenosis that developed following successful BA has no adverse effect on long-term survival.[7]

In this study, we compared the long term results of new-generaton DES with those of BA in patients presenting with BMS-ISR.

Material and methods

Patients

We analyzed clinical and angiographic data of patients who underwent PCI in our institution between June 2007 and January 2010. A total of 398 patients developed BMS-ISR during the study period. Of the 398 patients, 88 had DES-ISR, 11 patients underwent coronary artery bypass graft surgery, 9 patients underwent hybrid coronary revascularization, 3 patients refused percutanous intervention, and 18 patients were lost to follow-up. The remaining 269 patients who were treated with balloon angioplasy or DES enrolled in the study. Patients

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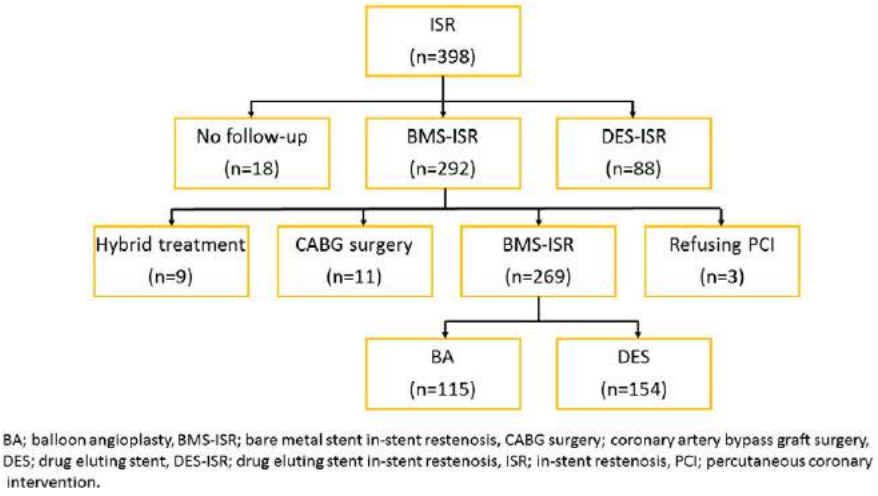

Fig. 1. Patient and treatment group profile. BMS ISR; bare metal in-stent restenosis, DES; drug eluting stent, DES ISR; drug eluting stent in-stent restenosis, ISR; instent restenosis.

were assigned to balloon angioplasty group (115 patients) or DES group (154 patients). Fig. 1 shows diagram of patients included and excluded in the study. Follow-up for all patients was continued until July 2013. The study was approved by the local ethics committee.

Clinical and laboratory evaluation

A medical history was taken from each patient, followed by a physical examination. Patient data were extracted from electronic medical records. The collected data included patient demographics, clinical characteristics, risk factors (arterial hypertension, diabetes mellitus, smoking, family history of coronary artery disease, dyslipidemia), medications, previous invasive cardiac procedures and echocardiographic findings including left ventricular ejection fraction (LVEF). Patients were excluded if they had active infection, anemia, renal failure, hepatic disease and thyroid function abnormalities. Patients received DAPT for four weeks after BMS implantation. Coronary angiography was performed to define coronary anatomy in patients who developed anginal symptoms, unstable angina, myocardial infarction (MI) and ischemic findings on nonivasive testing.

Coronary intervention

Coronary interventions were performed according to current practice guidelines and the results were recorded digitally for quantitative analysis. Degree of coronary stenosis was estimated visually by two experienced interventional cardiologists.

Definitions were based on predetermined criteria.

a- ISR was defined as >50% narrowing of the lumen diameter according to the results of follow-up coronary angiographies.
b- The Mehran and American College of Cardiology/American Heart Association classifications were used to assess lesion shape.[8] The classification is based on the length and pattern of the restenotic lesion in relation to the stented portion of the vessel. Four types of ISR have been defined: (I) focal (≤10 mm length); (II) diffuse (ISR > 10 mm within the stent); (III) proliferative (ISR > 10 mm extending outside the stent); and (IV) occlusive ISR.
c- Target vessel revascularization (TVR) was defined as repeat percutanous coronary intervention within the index procedure stent or 5 mm edge.[9–10]
d- All deaths were considered to be cardiac related unless a clear noncardiac cause could be established.
e- The diagnosis of MI required 2 of the following: 1) prolonged (>30 min) chest pain; 2) a rise in creatine kinase levels exceeding twice the local upper normal limit value (with abnormal MB fraction); and 3) development of persistent ischemic electrocardiographic changes (with or without new pathological Q waves).[11]
f- The Academic Research Consortium definition was used to assess the presence of stent thrombosis.[12]
g- Significant coronary stenosis was defined as 50% narrowing of the lumen diameter in major epicardial coronary vessels.[13]

All patients received clopidogrel (300 to 600 mg) at least 6 h before the stent implantation. They also received weight-adjusted intravenous heparin before the intervention. Procedural success was defined as reduction of stenosis to less than 10% residual narrowing, with improvement in ischemic symptoms and without major procedure related complications: death, emergency bypass surgery, or myocardial infarction (defined to be greater than twice the increase in creatine kinase-MB levels).[14]

Drug eluting sirolimus stent or drug eluting paclitaxel stent was used instent restenosis. Balloon size was selected in order to achieve a final balloon-to-artery ratio of 1.1/1. Relatively high pressures (>12 atm) were recommended.

The patients were premedicated with aspirin 100 mg/day, and were given clopidogrel (loading dose of 300 to 600 mg) at least 6 h before the intervention. The patients were advised to stay on clopidogrel for one year after stent implantation. All patients received optimal medical therapy.

The decision between BA and DES implantation as well as the choice between DES, BA or medical treatment in cases of recurrent restenosis were left to the operator. Patients in the DES group received DAPT for one year, whereas patients in the BA received aspirin only.

Intraobserver and interobserver variabilities of ISR analysis were assessed in a subset of 50 patients. Interpretations of the two investigators on the presence or absence of ISR agreed in 92% and 95% respectively. Intraobserver variability was assessed by one investigator. The concordance rate of the two readings for the presence or absence of ISR was 94% and 95% respectively.

Statistical analysis

Continuous variables are expressed as mean ± SD. Categorical variables are expressed as percentages. To compare parametric continuous variables, Student's t-test was used; to compare nonparametric continuous variables, the Mann Whitney U test was used; and to compare categorical variables, chi-squared test was used. Multivariate logistic regression analysis was carried out to identify the independent predictor of MACE. Event-free survival curves were generated by the

Table 1
Patients' clinical characteristics.

	BA	DES	p
Male, n (%)	91 (79.1%)	134 (87%)	0.08
Age, (years)	61.6 ± 11.0	61.7 ± 10.8	0.967
LVEF (%)	64.1 ± 9.3	66.4 ± 8.7	0.63
Current smoker, n (%)	54 (47%)	68 (44.2%)	0.648
Family history of CAD, n (%)	40 (34.7%)	55 (35.6%)	0.40
Diabetes mellitus, n (%)	57 (49.6%)	64 (41.6%)	0.192
Hypertension, n (%)	65 (56.5%)	77 (50%)	0.550
In-hospital medical treatment, n (%)			
ACE-I/ARB	47 (41%)	77 (50%)	0.645
Beta blocker	63 (55.1%)	91 (59.2%)	0.26
Calcium channel blocker	20 (17.4%)	26 (17.1%)	0.30
Statins	64 (56%)	83 (54%)	0.13
Fasting glucose, (mg/dL)	101 ± 10	89 ± 11	0.50
HDL-C, (mg/dL)	42 ± 13	44 ± 9.9	0.24
LDL-C, (mg/dL)	140 ± 56	135 ± 28	0.41
Hemoglobin, (g/L)	13.0 ± 1.4	13.3 ± 1.4	0.60
Creatinine, (mg/dL)	0.89 ± 0.27	0.94 ± 0.30	0.08

ACE-I; angiotensin converting enzyme inhibitor, ARB; angiotensin receptor blocker, BA; balloon angioplasty, CAD; coronary artery disease, DES: drug eluting stent, HDL-C; high density lipoprotein cholesterol, LVEF; left ventricular ejection fraction, LDL-C; low density lipoprotein cholesterol.

Kaplan–Meier method and differences in survival rates were compared using log-rank test. All variables showed significance values less than 0.05. Two-tailed P values less than 0.05 with a confidence interval of 95% were considered as significant. All statistical studies were performed by using the SPSS program (version 22.0; SPSS Inc., Chicago, Illinois, USA).

Results

Baseline clinical and demographic characteristics were similar in both groups. There was no difference between the two groups in terms of age, sex, hypertension, hyperlipidemia, diabetes mellitus, smoking, LVEF and in-hospital medical treatment. Table 1 shows the clinical characteristics of the patients.

Table 2
Patients' angiographic and procedural characteristics.

	BA	DES	p
Indication for stenting, n (%)			
Stable angina pectoris	72 (47%)	91 (59%)	0.11
Unstable angina pectoris	33 (27%)	50 (31%)	0.23
Myocardial infarction	10 (8.6%)	13 (10%)	0.45
Coronary vessel treated, n (%)			0.01
LAD	43 (40.6%)	83 (57.6%)	
LCX	26 (24.5%)	22 (15.3%)	
RCA	35(33%)	31 (21.5%)	
Number of treated coronary artery, n ≥ 2	2 (1.9%)	8 (5.6%)	
Stent length, (mm)	18.9 ± 5.9	16.0 ± 6.6	0.05
Stent diameter, (mm)	2.70 ± 0.54	3.32 ± 0.65	0.04
Lesion morphology of the previous			0.723
procedure, n (%)	15 (13%)	11 (7.1%)	
A	24 (20.8%)	37 (24.2%)	
B1	66 (57.4%)	94 (60.9%)	
B2	10 (8.6%)	12 (7.8%)	
C			
In-stent restenosis pattern by Mehran			<0.0001
classification, n (%)	42 (40.7%)	64 (33.1%)	
Pattern I: focal	46 (39.3%)	51 (32.9%)	
Pattern II: (diffuse, intrastent)	25 (22.1%)	20 (13%)	
Pattern III: (proliferative)	0 (0%)	19 (12.3%)	
Pattern IV: (occlusion)			

LAD; left anterior descending artery, RCA; right coronary artery; LCX; left circumflex artery.

Table 3
1-year follow-up results of the patients.

	BA	DES	p
MACE, n (%)	33 (28.7%)	13 (8.7%)	<0.0001
Death, n (%)	15 (13%)	11 (7.1%)	0.105
MI, n (%)	8 (7%)	10 (6.5%)	0.880
Target lesion revascularization, n (%)	23 (20%)	11 (7.1%)	0.002

MACE; major adverse cardiac events, MI; myocardial infarction.

The indications for stenting were similar in both groups. Elapsed time between stent implantation and ISR intervention was 9.8 ± 9.3 months. There were no statistically significant differences between the two groups in terms of lesion type and stent length. Compared with BA group, DES group had more LAD lesions involved and larger stent diameter (Table 2). BA was used predominantly in class I and II lesions, whereas class III and IV lesions were treated with DES. Repeat coronary angiography was performed at 9.3 ± 8.7 and 13.8 ± 10.5 months ($p = 0.03$) in BA and DES groups, respectively. Patients in the BA and DES groups were followed-up for 51.05 ± 12.7 and 54.5 ± 13.9 months ($p = 0.147$), respectively. Procedural and angiographic variables are shown in Table 2.

At one-year follow-up, patients in the DES group showed significantly lower MACE and TVR. MI and mortality rates were not significantly different between two groups. 1-year follow-up results are given in Table 3.

A mean of 5 years follow-up resulted in a significantly higher composite of MACE including death, MI and TVR, recurrent angina and TVR in BA group than those in the DES group. Cardiac mortality and MI rates were similar in both groups. TVR for recurrent restenosis occurred earlier in the BA group than that in the DES-group. Numbers of repeated target lesion and restenosis rate were both higher for the BA group than for the DES group. The incidence of definite stent thrombosis was similar in both groups. Of all six patients with stent thrombosis, four had stopped antiplatelet therapy prior to elective surgery and only two patients were on DAPT. Five-year follow-up results are given in Table 4.

Fig. 2 shows the 5-year event-free survival of the patients. MACE-free 1-year survival and 5-year survival rates were significantly higher in DES group compared to BA group. There was a progressive decrease in LVEF and renal function in BA group compared to DES group. However, these differences were not statistically significant.

Table 5 shows the results of the multivariate analysis to identify the predictors of MACE. Diabetes, lesion type, treatment device (BA or DES)

Table 4
5-year follow-up results of the patients.

	BA	DES	p
Follow-up duration, years	4.21 ± 1.03	4.49 ± 1.13	0.043
Composite MACE, n (%)	58 (50.4%)	49 (31.8%)	0.002
Cardiac death, n (%)	25 (21.7%)	25 (16.2%)	0.251
CABG surgery, n (%)	5 (4.3%)	6 (3.9%)	0.380
MI, n (%)	7 (6.1%)	8 (5.2%)	0.752
Recurrent angina, n (%)	49 (42.6%)	42 (27.3%)	0.009
Time to repeat revascularization months, n (%)	8.7 ± 4.2	12.5 ± 4.4	0.02
Target lesion revascularization, n (%)	43 (37.4%)	32 (20.8%)	0.003
Repeated target lesion, n	2.0 ± 0.8	1.38 ± 0.5	0.01
Stent thrombosis, n (%)	1 (0.9%)	6 (3.9%)	0.123
Acute, n (%)	1 (0.9%)	1 (0.6%)	
Subacute, n (%)	0 (0%)	1 (0.6%)	
Late, n (%)	0 (0%)	4 (2.5%)	
New lesion stenting, n (%)	8 (7%)	9 (%5.8)	0.711
Cardiac event free survival, n (%)	57 (49.6%)	105 (68.2%)	<0.0001
Restenosis rate	44 (38.3%)	33 (21.4%)	0.003

CABG surgery; coronary artery bypass graft surgery, MACE; major adverse cardiac events, MI; myocardial infarction.

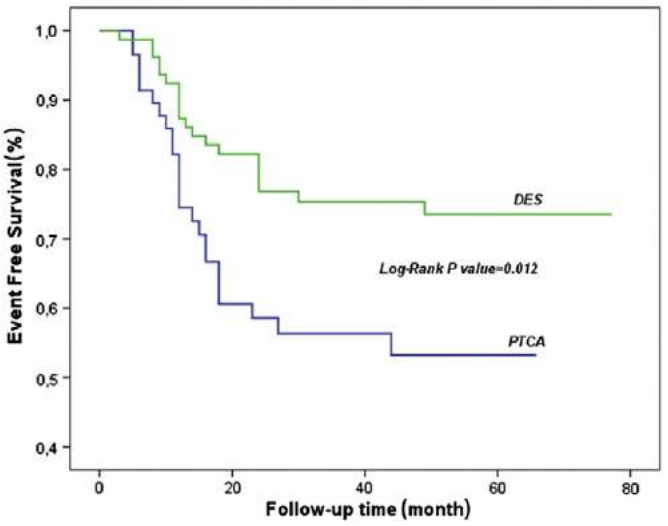

Fig. 2. Kaplan–Meier analysis of the event-free survival at five years according to treatment group.

and Mehran classification were independent predictors of MACE in multivariate analysis.

Discussion

The main finding of our study was that, at 5-year follow-up, composite MACE, recurrent angina and TVR were significantly lower and MACE-free survival was significantly better in the DES treated patients. However mortality and risk of MI did not differ between two groups. DES use was associated with lower rates of composite MACE and TVR, although it was utilized in patients with more complicated lesions.

Coronary stent implantation is frequently performed in percutanous coronary interventions. ISR is a major late complication following BMS implantation, which occurs in 10–80% of lesions treated in daily practice.[8] BMS-ISR is an independent predictor of mortality.[15,16] The use of BMS is associated with high restenosis rate (up to 25%). Although DES dramatically reduced the rate of restenosis to less than 10%,[17,18] it is associated with increased risk for late stent thrombosis and requires prolonged DAPT.[19]

Any percutaneous coronary intervention causes trauma to the vessel wall. ISR is generally viewed as a healing response after injury incurred during stent placement. This is characterized by a sequence of inflammation, granulation, extracellular matrix remodeling and smooth muscle cell proliferation. These processes lead to neointimal hyperplasia. Predictive factors of restenosis can be classified into patient-related, lesion-related and procedure-related factors. Lesion-related factors can be listed as vessel diameter, tortuous vessels, calcified lesions and previous restenosis. Patient-related factors include patient age, diabetes mellitus and genetic factors. Procedural characteristics predicting ISR are residual dissection and length of stented segment.[20]

Several therapeutic options to treat ISR have been proposed, such as repeat BA, repeat stenting, cutting balloon angioplasty, directional coronary atherectomy, rotational coronary atherectomy, brachytherapy, DES, molecular biology and genetics. BA has been used frequently, as it is relatively inexpensive and easy to perform. DES have achieved great success in treating patients with de-novo lesions. These findings provide a hope for the treatment of ISR. Sirolimus and paclitaxel inhibit smooth muscle cell proliferation and migration in vitro and in vivo.[21,22] Several studies have demonstrated that stents eluting sirolimus (Cypher, Cordis a Johnson and Johnson Company) and paclitaxel (TAXUS NIRx, Boston Scientific Corporation) reduce ISR.[23–27] It has been shown that oral therapy with sirolimus before and after repeat intervention results in a significant improvement in the angiographic parameters of restenosis.[28] Paclitaxel-coated balloon catheters reduce repeat restenosis in patients with ISR.[29]

In spite of insufficient data, interventional cardiologists commonly use the stent sandwich technique to treat ISR. Final lumen cross-sectional area is the independent predictor of subsequent ISR and TVR. Full stent expansion with sufficient lumen area (lumen area 90% or greater of the average reference lumen area preintervention) minimizes restenosis. Compared to BA, coronary stenting achieved better initial angiographic results but fails to improve restenosis rate in patients with ISR. However, patients with large vessels (≥3 mm) have a better outcome after repeat stenting.[4]

ISAR-DESIRE study randomized 300 patients with BMS ISR to treatment with SES, PES and BA.[30] At 1-year follow-up, the use of DES led to significantly lower rates of TVR. DES were markedly superior to conventional balloon angioplasty for the treatment of BMS-ISR. RIBS-II compares the results of SES with those of BMS in patients with ISR.[11] At 1 year, the event-free survival was better in the SES group. At 4 years, the event-free survival was 76% in the SES arm and 65% in the BA arm ($p = 0.019$). Same researchers published the results of 450 patients with ISR treated with BA or repeat BMS implantation.[4] In that study, binary restenosis rate, TVR and one-year event-free survival were similar in both groups.

In our study, MACE and TVR rates were consistent with other studies. Myocardial and cardiac deaths were similar in both groups. During follow-up MACE rates were significantly lower and MACE-free survival was significantly better in the DES treated patients.

Table 5
Multivariate predictors of composite MACE.

	OR	%95 CI	p
HT	1.24	0.73–2.11	0.417
DM	0.466	0.291–0.843	0.003
Number of diseased coronary arteries	1.13	0.69–1.84	0.624
Lesion type	0.66		0.001
Stent length (mm)	0.993	0.948–1.04	0.779
Mehran classification	2.82	2.09–3.89	0.000
Number of stents used	0.789	0.505–1.23	0.300
Stent diameter	0.684	0.312–1.49	0.341
Treatment with BA or DES	2.18	1.32–3.59	0.002

BA, balloon angioplasty, CI; confidence interval, DES; drug eluting stent, DM; diabetes mellitus, HT; hypertension, OR; odds ratio.

Conclusions

ISR has been a longstanding problem after percutaneous coronary intervention. The pathophysiology of ISR is multifactorial. There are several options for therapy and treatments that should therefore be individualized. Although DES have significantly reduced in-hospital complications, ISR and TVR, BA compared with DES resulted in similar rates of mortality and MI. Hence, BA may be the preferred method of treatment. Recent advances in DES technology have allowed cardiologists to treat much more complicated lesions and have dramatically reduced the rates of ISR and TVR.

Study limitations

Our study has several limitations**.** It was a retrospective analysis of data at a single center and the number of patients was small. Discontinuation of medical treatment during follow-up may affect the patient outcomes. Decision between BA and DES implantation was left up to the operator. Another limitation was that the current funding restrictions in our country affected the operator preferences and resulted in lower DES use.

Declaration of conflicting interests

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Conflict of interest

None declared.

References

1. Cutlip DE, Chauhan MS, Baim DS, et al. Clinical restenosis after coronary stenting: perspectives from multicenter clinical trials. *J Am Coll Cardiol* 2002;**40**(12):2082.
2. Díaz JF, de La Torre JM, Sabaté M, Goicolea J. Spanish Cardiac Catheterization and Coronary Intervention Registry. 21st official report of the Spanish Society of Cardiology Working Group on Cardiac Catheterization and Interventional Cardiology (1990–2011), Vol. 65 (12). Rev Esp Cardiol (Engl Ed); 2012. p. 1106–1116.
3. Pellegrini DO, Gomes VO, Lasevitch R, et al. Efficacy and safety of drug-eluting stents in the real world: 8-year follow-up. *Arq Bras Cardiol* 2014 Sep;**103**(3):174–182.
4. Alfonso F, Zueco J, Cequier A, et al. A randomized comparison of repeat stenting with balloon angioplasty in patients with restenosis after coronary stenting. *J Am Coll Cardiol* 2003;**42**(5):796–805.
5. Kastrati A, Mehilli J, von Beckerath N, et al. Sirolimus-eluting stent or paclitaxel-eluting stent vs balloon angioplasty for prevention of recurrences in patients with coronary in-stent restenosis: a randomized controlled trial. *JAMA* 2005;**293**(2):165–171.
6. Alfonso F. Treatment of drug-eluting stent restenosis the new pilgrimage: quo vadis. *J Am Coll Cardiol* 2010;**55**(24) [2717–2120].
7. Weintraub WS, Ghazzal ZM, Douglas Jr JS, et al. Long-term clinical follow-up in patients with angiographic restudy after successful angioplasty. *Circulation* 1993;**87**(3):831–840.
8. Mehran R, Dangas G, Abizaid AS, et al. Angiographic patterns of instent restenosis. Classification and implications for long-term outcome. *Circulation* 1999;**100**(18):1872–1878.
9. Alfonso F, Cequier A, Angel J, et al. Value of the American College of Cardiology/American Heart Association angiographic classification of coronary lesion morphology in patients with in-stent restenosis. *Am Heart J* 2006;**151**(3):681.e1–681.e9.
10. Al Muradi H, Mehra A, Okolo J, et al. Clinical presentation and predictors of target vessel revascularization after drug-eluting stent implantation. *Cardiovasc Revasc Med* 2012;**13**(6):311–315.
11. Alfonso F, Pérez-Vizcayno MJ, et al. A randomized comparison of sirolimus-eluting stent with balloon angioplasty in patients with in-stent restenosis: results of the Restenosis Intrastent: Balloon Angioplasty Versus Elective Sirolimus-Eluting Stenting (RIBS-II) trial. *J Am Coll Cardiol* 2006;**47**(11):2152–2160.
12. Cutlip DE, Windecker S, Mehran R, et al. Academic Research Consortium. Clinical end-points in coronary stent trials. A case for standardized definitions. *Circulation* 2007;**115**(17):2344–2351.
13. Smith Jr SC, Feldman TE, Hirshfeld Jr JW, et al. ACC/AHA/SCAI 2005 guideline update for percutaneous coronary intervention summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/SCAI Writing Committee to Update the 2001 Guidelines for Percutaneous Coronary Intervention). *Circulation* 2006;**113**(1):156–175.
14. Nebeker JR, Virmani R, Bennett CL, et al. Hypersensitivity cases associated with drug-eluting coronary stents: a review of available cases from the Research on Adverse Drug Eventsand Reports (RADAR) project. *J Am Coll Cardiol* 2006;**47**(1):175–181.
15. Schühlen H, Kastrati A, Mehilli J, et al. Restenosis detected by routine angiographic follow-up and late mortality after coronary stent placement. *Am Heart J* 2004;**147**(2):317–322.
16. Chen YL, Chen MC, Wu CJ, et al. Impact of 6-month angiographi restenosis inside bare-metal stents on long term clinical outcome in patients with coronary artery disease. *Int Heart J* 2007;**48**(4):443–454.
17. Moses JW, Leon MB, Popma JJ, et al. Sirolimus-eluting stents versus standard stents in patients with stenosis in a native coronary artery. *N Engl J Med* 2003;**349**(14):1315–1323.
18. Stone GW, Ellis SG, Cox DA, et al. A polymer-based, paclitax eleluting stent in patients with coronary artery disease. *N Engl J Med* 2004;**350**(3):221–231.
19. Kalesan B, Pilgrim T, Heinimann K, et al. Comparison of drug-eluting stents with bare metal stents in patients with ST-segment elevation myocardial infarction. *Eur Heart J* 2012;**33**(8):977–987.
20. Bhatia Vineet, Kaul Upendra. Management of in-stent restenosis after DES use. *Card Interv Today* 2011(July/August):50–55.
21. Marx SO, Jayaraman T, Go LO, Marks AR. Rapamycin-FKBP inhibits cell cycle regulators of proliferation in vascular smooth muscle cells. *Circ Res* 1995;**76**(3):412–417.
22. Poon M, Marx SO, Gallo R, Badimon JJ, Taubman MB, Marks AR. Rapamycin inhibits vascular smooth muscle cell migration. *J Clin Invest* 1996;**98**(10):2277–2283.
23. Sousa JE, Costa MA, Abizaid AC, et al. Sustained suppression of neointimal proliferation by sirolimus-eluting stents: one-year angiographic and intravascular ultrasound follow-up. *Circulation* 2001;**104**(17):2007–2011.
24. Degertekin M, Serruys PW, Foley DP, et al. Persistent inhibition of neointimal hyperplasia after sirolimus-eluting stent implantation: long-term (up to 2 years) clinical, angiographic, and intravascular ultrasound follow-up. *Circulation* 2002;**106**(13):1610–1613.
25. Morice MC, Serruys PW, Sousa JE, et al. Randomized study with the sirolimus-coated Bx velocity balloon-expandable stent in the treatment of patients with de novo native coronary artery lesions. A randomized comparison of a sirolimus-eluting stent with a standard stent for coronary revascularization. *N Engl J Med* 2002;**346**(23):1773–1780.
26. Grube E, Silber S, Hauptmann KE, et al. TAXUS I: six- and twelve-month results from a randomized, double-blind trial on a slowrelease paclitaxel-eluting stent for de novo coronary lesions. *Circulation* 2003;**107**(1):38–42.
27. Colombo A, Drzewiecki J, Banning A, et al. TAXUS II study group. randomized study to assess the effectiveness of slow- and moderate-release polymerbased paclitaxel-eluting stents for coronary artery lesions. *Circulation* 2003;**108**(7):788–794.
28. Hausleiter J, Kastrati A, Mehilli J, et al. Randomized, double- blind, placebo-controlled trial of oral sirolimus for restenosis prevention in patients with in-stent restenosis: the oral sirolimus to inhibit recurrent in-stent stenosis (OSIRIS) trial. *Circulation* 2004;**110**(7):790–795.
29. Scheller B, Hehrlein C, Bocksch W, et al. Treatment of coronary in-stent restenosis with a paclitaxel-coated balloon catheter. *N Engl J Med* 2006;**355**(20):2113–2124.
30. Kastrati A, Mehilli J, von Beckerath N, et al. ISAR-DESIRE study investigators. Sirolimus-eluting stent or paclitaxel-eluting stent vs balloon angioplasty for prevention of recurrences in patients with coronary in-stent restenosis: a randomized controlled trial. *JAMA* 2005;**293**(2):165–171.

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Case report

A different management of saphenous vein graft failure related to cardiac tamponade following coronary surgery

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ABSTRACT

Cardiac tamponade is a state of constriction of the heart with an excessive fluid or hematoma resulted from various conditions. Postoperative tamponade can occur after coronary bypass surgery. Despite it is uncommon, its results may have a high risk of mortality and morbidity. Acute postoperative cardiac tamponade reveals a vast spectrum of symptoms. Moreover, a compression over the saphenous vein graft is the worst complication that should be managed without delay. We report a rare case of saphenous vein graft failure due to the cardiac tamponade following a coronary surgery and its management with a practical measure.

Introduction

Acute postoperative cardiac tamponade is an uncommon but potentially fatal complication occurred within the seven days after cardiac surgery. [1]The incidence of this entity ranged from 0.1% to 6% and associated with preoperative or postoperative anticoagulation[2]. Cardiac tamponade often presents with the reduction in blood pressure (BP), tachycardia, increased central venous pressure, decreased urine output and respiratory distress[3]. Thus, prompt diagnosis and effective management are needed to prevent unwanted complications like circulatory and cardiorespiratory collapse.[4] However, the recent literature does not reveal enough publications related to the complications and managements of cardiac tamponade following open heart surgery. In this article, we report the management of a saphenous vein graft compression and failure due to the cardiac tamponade. Saphenous vein failure due to the external compression is a rare and life-threatening complication of cardiac tamponade encountered after coronary surgery.

Case report

A 62-year-old male was performed an urgent coronary bypass graft (CABG) operation due to acute myocardial infarction (AMI). Right coronary artery (RCA) and the circumflex artery (CxA) were revascularized with saphenous vein grafts and the left anterior descendent (LAD) artery was bypassed with internal mammary artery (IMA). The operation was accomplished with no intraoperative complication. Intensive care unit parameters were normal including chest roentgenogram (Fig. 1A). However, there was a continuous bleeding through the drain tubes. Abundant bleeding (200 ml/h) was related to the preoperative administration of 600 mg of Clopidogrel, which is a routine protocol that has been applied to all AMI patients. Transfusion of thrombocyte, fresh frozen plasma and blood products could not restore the hemoglobin levels. After 10 h of medical treatment, total bleeding reached to 2000 ml. Posteroanterior chest roentgenogram showed a widened mediastinum (Fig. 1B). Echocardiography revealed a massive global pericardial effusion that was 30 mm on the apex and 34 mm on the posterior segment in long axis examination. In apical four-chamber view, diastolic dysfunction of the right atrium was observed that was consistent with cardiac tamponade. The ejection fraction (EF) was measured as 52% with the Simpsons method (Preoperative EF was 54%). There was a mild hypokinesia in the posterobasal 2/3 of the myocardium which had also existed before the operation. Additionally, electrocardiography showed moderate depressions in D3 and AVF leads that implicated a graft failure. BP was progressively dropped to 75/35 mmHg that made us consider chest re-exploration with the diagnosis of cardiac tamponade. Coronary angiography was not performed before the reoperation in this case. Circulatory collapse necessitated an immediate reoperation without preceding angiography. The patient was reopened, and the hematoma was evacuated. No imminent surgical bleeding foci were found. However, the saphenous vein graft of the RCA was pulseless and found to be squeezed and thrombosed during both systole and the diastole due to the compression of massive hematoma. The graft of CxA was normal. The RCA graft failure was thought to be caused by the cardiac tamponade as the ECG of the patient had been otherwise normal until the cardiac

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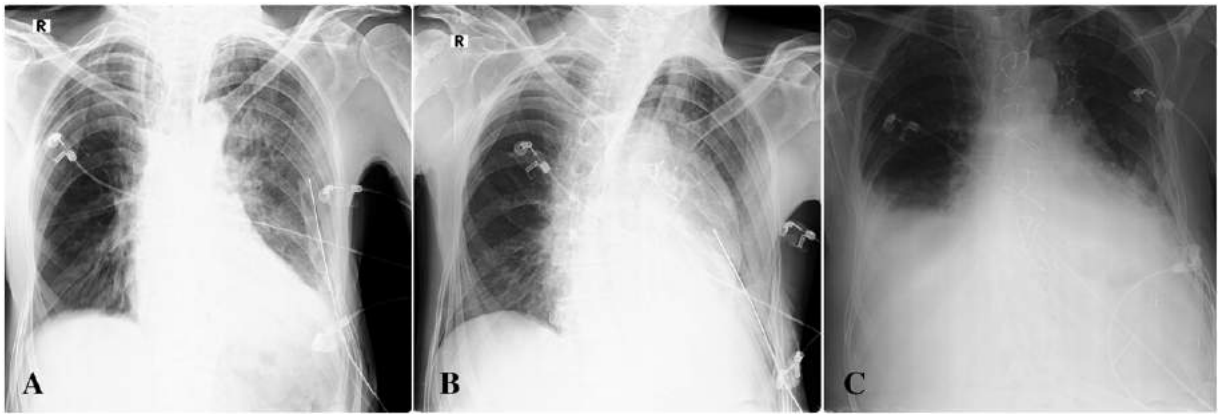

Fig. 1. Chest roentgenogram (A) Early postoperative (B) Cardiac tamponade (C) After reopening.

tamponade clinic was started. Although a thrombectomy was initially performed to the RCA graft using Fogarty catheter, circulation and pulsation could not be restored. Failed graft was decided to be replaced. A new saphenous vein graft was harvested and interposed between the proximal portion of the CxA graft and the distal part of the failed RCA graft (Fig. 2). ECG revealed a dramatic improvement in inferior leads and the BP was increased. The patient was extubated on the sixth hour postoperatively and discharged on the seventh day after surgery with a normal chest roentgenogram (Fig. 1C).

Discussion

Surgical re-exploration due to bleeding after cardiac operations accompanies with various complication affecting the postoperative course and leads to increased mortality and morbidity.[5] However, postponing the timing of reoperation despite the symptoms of cardiac tamponade also presents a risk when the delay creates a hemodynamic instability and increases the need for the excessive amount of allogeneic blood products.[6]

Cardiac tamponade is the collection of fluid in the pericardial cavity and compression of the heart causing hemodynamic instability due to the compromised contractility.[7] In all types of cardiac tamponade, including the tamponade after cardiac surgery, the mediastinum stretches to accommodate the increased volume. When the expansion capacity of the mediastinum is no longer available, intrapericardial pressure starts to rise. If this pressure exceeds the intracardiac pressure, the contractile capability of the heart diminishes.[4] This increased pressure may also cause a compression over the vein grafts as in this case. The consequences may vary from the transient collapse of the vein graft to total obstruction as a result of graft thrombosis. In the event of a graft failure after the cardiac tamponade, aortocoronary bypass should inevitably be renewed. However, conducting this reoperation under the extracorporeal circulation may cause further impairment of the myocardium. Thus, instead of the constitution of a re-arrest, the off-pump CABG choice should be taken into consideration if possible. As we present in this report, a new graft may easily be implanted between the other intact graft and the failed graft. This preference not only protects the patient from an extensive reoperation but also enables the surgeon to perform the revascularization easily, fastly and practically with a reduced risk of mortality.

Early graft failure (within the first 30 days after surgery) is seen in approximately 5 to 10% of saphenous vein grafts. These occlusions may be due to thrombosis that is related to technical problems at the anastomosis or the injury due to the manipulation during harvesting.[8]

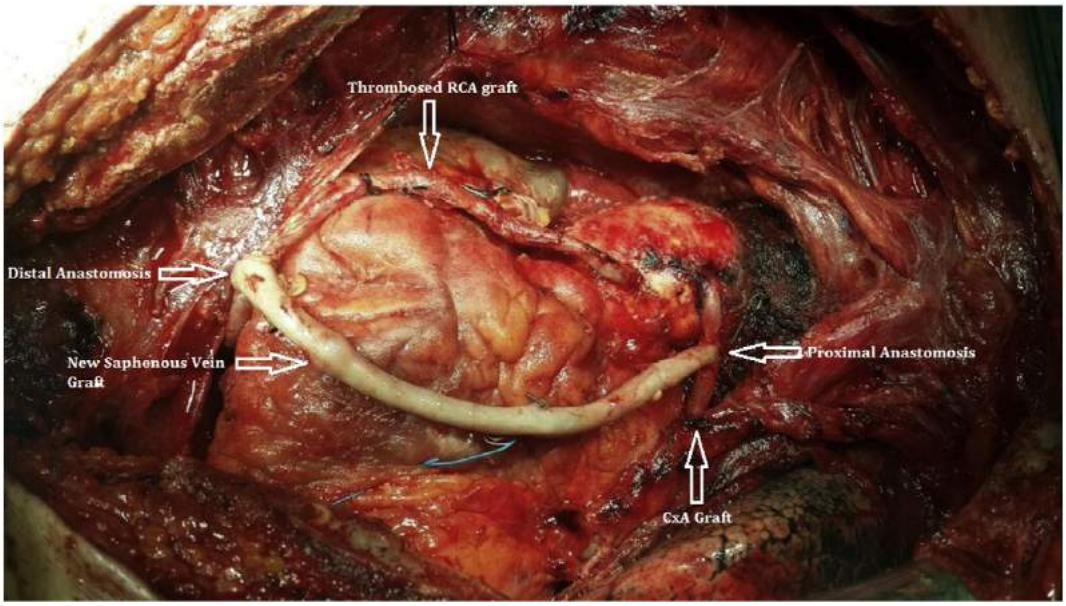

Fig. 2. A new saphenous vein graft was interposed between the two previous saphenous vein grafts.

The risk of early graft occlusion appears to be reduced by starting Acetylsalicylic acid therapy, which is typically started within six hours following surgery.

As a conclusion, this case implicates the risk of compression and failure of the saphenous vein grafts in cardiac tamponade following CABG. The management should be considered in a more practical way as in this case.

Conflict of interest

None declared.

References

1. Ranucci M, Bozzetti G, Ditta A, Cotza M, Carboni G, Ballotta A. Surgical reexploration after cardiac operations: why a worse outcome? *Ann Thorac Surg.* 2008;**86**(5):1557–1562.
2. Biancari F, Mikkola R, Heikkinen J, Lahtinen J, Airaksinen KJ, Juvonen T. Estimating the risk of complications related to re-exploration for bleeding after adult cardiac surgery: a systematic review and meta-analysis. *Eur J Cardiothorac Surg.* 2012;**41**(1):50–55.
3. Grumann A, Baretto L, Dugard A, et al. Localized cardiac tamponade after open-heart surgery. *Ann Thorac Cardiovasc Surg.* 2012;**18**(6):524–529.
4. Carmona P, Mateo E, Casanovas I, et al. Management of cardiac tamponade after cardiac surgery. *J Cardiothorac Vasc Anesth.* 2012;**26**(2):302–311.
5. Ranucci M, Bozzetti G, Ditta A, Cotza M, Carboni G, Ballotta A. Surgical reexploration after cardiac operations: why a worse outcome? *Ann Thorac Surg.* 2008;**86**(5):1557–1562.
6. Choong CK, Gerrard C, Goldsmith KA, Dunningham H, Vuylsteke A. Delayed re-exploration for bleeding after coronary artery bypass surgery results in adverse outcomes. *Eur J Cardiothorac Surg.* 2007;**31**(5):834–838.
7. Roy CL, Minor MA, Brookhart MA, Choudhry NK. Does this patient with a pericardial effusion have cardiac tamponade? *JAMA* 2007;**297**(16):1810–1818.
8. Dauerman HL, Cutlip DE, Sellke FW. Intracoronary thrombolysis in the treatment of graft closure immediately after CABG. *Ann Thorac Surg.* 1996;**62**(1):280–283.

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Case report

Idiopathic epicardial ventricular tachycardia originating from the great cardiac vein☆

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ABSTRACT

Radiofrequency catheter ablation is accepted as an effective and curative therapy for idiopathic ventricular tachycardia (IVT). Although endocardial radiofrequency (RF) ablation is the common approach for ablation of IVTs, rare patients have been reported in whom ventricular tachycardia (VT) could not be ablated from endocardium due to an epicardial origin of the tachycardia. We, herein, present a case of IVT originating from the great cardiac vein that was successfully ablated within the coronary venous system.

Introduction

Radiofrequency catheter ablation is accepted as an effective and curative therapy for idiopathic ventricular tachycardia (IVT). Although endocardial radiofrequency (RF) ablation is the common approach for ablation of IVTs, rare patients have been reported in whom ventricular tachycardia (VT) could not be ablated from endocardium due to an epicardial origin of the tachycardia. IVTs originating from the epicardial portion are remote from the left ventricular (LV) endocardium and are not amenable to standard approach via endocardial RF ablation.[1–3] Several recent reports have demonstrated that the left coronary veins are potential routes for mapping and ablating VT originating from an epicardial site.[4,5] IVT/premature ventricular complexes (PVCs) originating from the great cardiac vein constitute only a small number of cases. We, herein, present a case of IVT arising from the great cardiac vein that was successfully ablated within the coronary venous system.

Case report

A 25-year-old male first admitted to our hospital with palpitation and atypical chest pain for six months. His physical examination was unremarkable. A 12-lead electrocardiogram (ECG) revealed PVCs with left bundle branch block (LBBB) and inferior axis (Fig. 1A). Echocardiography showed a normal examination with a global ejection fraction of 65% and no chamber enlargement or valve disease. 24-hour Holter monitorization showed frequent monomorphic PVCs and several times of sustained and nonsustained VTs (Fig. 1B). In the cardiovascular stress test, the frequency of PVCs increased with exercise, and the test was terminated because of a sustained VT in stage 3 (Fig. 2A). The patient underwent electrophysiologic study (EPS) using conventional mapping technique. But no early ventricular activation site was found. Epicardial origin was suspected on ECG that showed inferior axis and LBBB with a pseudo delta wave (PdW), precordial R-wave transition in V3 and tall R wave in the inferior leads. In the mapping of the coronary sinus in EPS, the earliest epicardial activation preceding the onset of the QRS complex by 32 ms was found in the great cardiac vein (Fig. 2B). Also, pace mapping provided an identical (12/12) match with the clinical PVCs morphology. Coronary angiography showed that the distance from the catheter tip to coronary arteries was enough for RF application (Fig. 3A, 3B). One application of RF energy at this site (55 °C, 20 W, temperature control) for 30 s by Marinr multicurve ablation catheter terminated spontaneous PVCs/VT. No VT or PVCs were inducible after RF at that site by ventricular or atrial stimulation. After the procedure, monitorization in coronary care unit showed normal sinus rhythm (Fig. 3C). The patient was discharged from the hospital without any medication and remained completely asymptomatic during 12 months follow-up.

Discussion

Idiopathic ventricular tachycardia and PVCs mainly originate from the right ventricular outflow tract (RVOT). However, uncommon sites of origin are seldom encountered. The incidence of an epicardial origin in IVT may be as high as 9%.[6] There is little data regarding the

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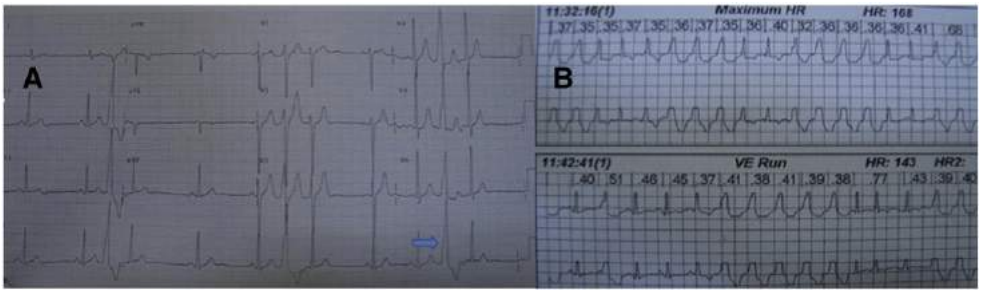

Fig. 1. 12-lead ECG revealed PVCs with LBBB and inferior axis (A). 24-hour Holter monitorization showed nonsustained VT (B). ECG: electrocardiogram, LBBB: left bundle branch block, PVCs: premature ventricular complexes, VT: ventricular tachycardia.

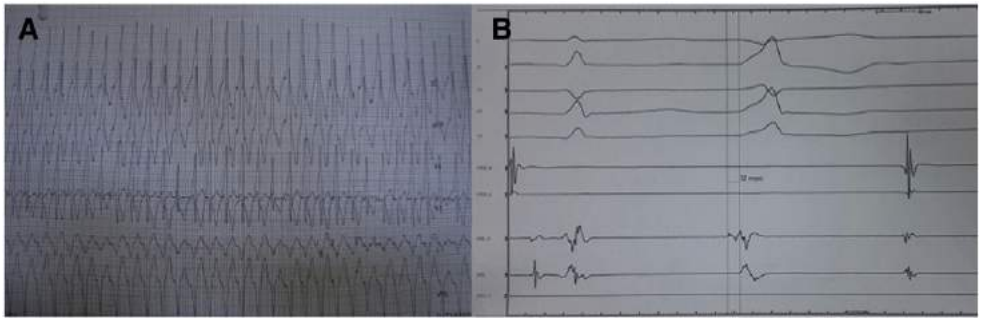

Fig. 2. The cardiovascular stress test in stage 3 demonstrated a sustained VT (A). Electrophysiologic tracing showed that the earliest epicardial activation preceding the onset of the QRS complex was 32 ms (B). VT: ventricular tachycardia.

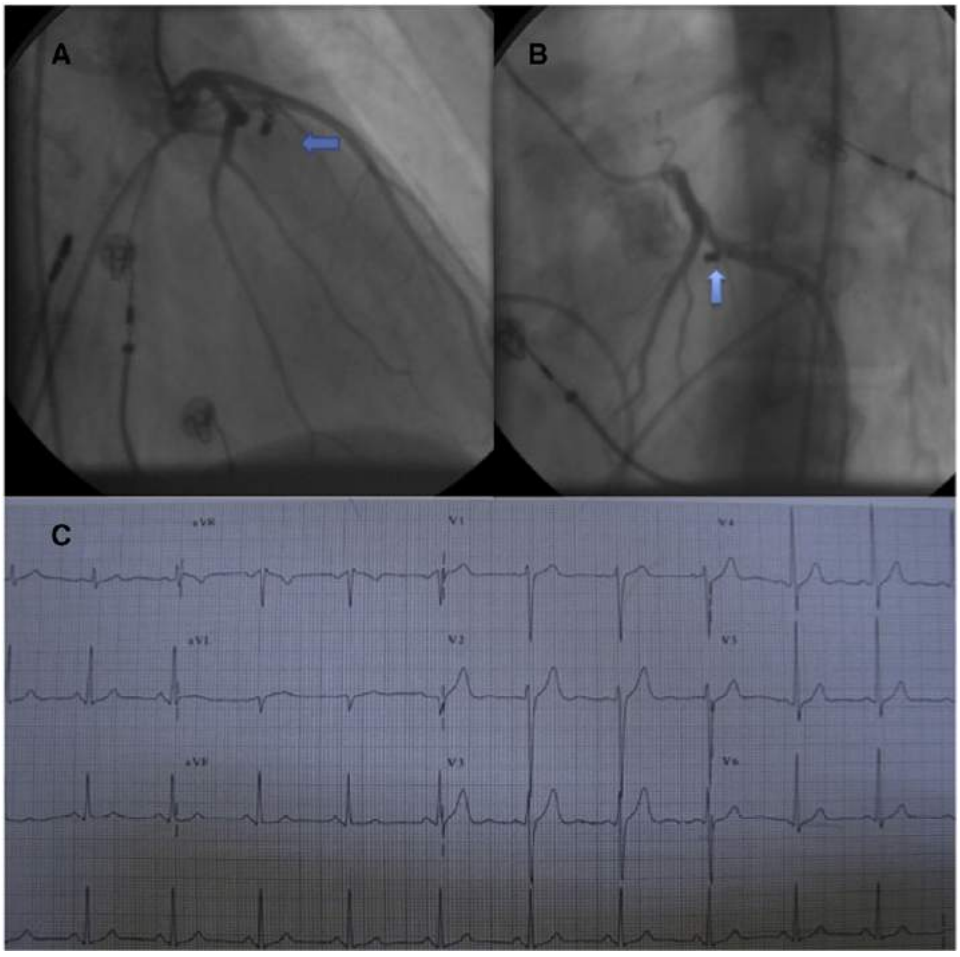

Fig. 3. Coronary angiography from RAO (A) and LAO (B) views revealed the distance from the catheter tip (arrow) to coronary arteries. Post-procedural ECG showed normal sinus rhythm (C). ECG: electrocardiogram, LAO: left anterior oblique, RAO: right anterior oblique.

prevalence, ECG characteristics, and common sites of tachycardia origin around the coronary sinus and especially its branches. Also, the efficacy of RF catheter ablation of these cases are unclear.[6] ECG is an essential guide to locate the origin of the tachycardia and to plan the optimal catheter ablation before the patient is brought to the electrophysiology laboratory. An S wave in lead I, deep Q wave in lead aVL, tall R wave in the inferior leads, and precordial transition zone near the leads V1–3 have been reported as the ECG findings of IVTs arising from the LV epicardial portion.[7] Berruezo et al.[8] also described the ECG criteria that identified an epicardial origin of VT including PdW ≥ 34 ms in precordial leads, intrinsicoid deflection time (IDT) ≥ 85 ms in lead V2, and shortest RS complex duration ≥ 121 ms in any precordial leads. As is seen in this case, the ECG characteristics have shown almost the same diagnostic criteria related to epicardial origin. Medical therapy and ablation are the options for management of these arrhythmias. Frequency and severity of symptoms play a critical role in determining the treatment strategy.[6,7] Ablative therapy of epicardial VT/PVCs includes percutaneous and transthoracic epicardial approaches. Several recent reports have demonstrated that the left coronary veins are potential routes for mapping and ablating VT originating from an epicardial site. This procedure can successfully treat the majority of the patients. The transthoracic epicardial approach is recommended in cases in which standard ablation methods have failed.[6] RF energy applications within the coronary venous system can be challenging because they can potentially cause complications, such as venostenosis, vein rupture, venous thrombosis, cardiac tamponade, or coronary artery injury, even if they are performed with relatively low power.[9]

Conclusion

Although idiopathic VT and PVCs mainly originate from the RVOT, uncommon sites of origin are seldom encountered. If the idiopathic VT/PVCs are not localized in the most common origin, epicardial foci including coronary sinus and its branches should be sought by pace-mapping. Also, physicians should keep in mind that a detailed analysis of the ECG is indispensable for diagnosing and ablating IVTs arising from the LV epicardium.

References

1. Tada H, Nogami A, Naito S, et al. Left ventricular epicardial outflow tract tachycardia: a new distinct subgroup of outflow tract tachycardia. *Jpn Circ J* 2001;**65**:723–730.
2. Ito S, Tada H, Naito S, et al. Development and validation of an ECG algorithm for identifying the optimal ablation site for idiopathic ventricular outflow tract tachycardia. *J Cardiovasc Electrophysiol* 2003;**14**:1280–1286.
3. Aksu T, Erdem GT, Yalin K. Successful ablation of an epicardial ventricular tachycardia by video-assisted thoracoscopy. *Europace* 2015;**17**:1116.
4. Obel OA, d'Avila A, Neuzil P, Saad EB, Ruskin JN, Reddy VY. Ablation of left ventricular epicardial outflow tract tachycardia from the distal great cardiac vein. *J Am Coll Cardiol* 2006;**48**:1813–1817.
5. Wright M, Hocini M, Ho SY, Haissaguerre M. Epicardial ablation of left ventricular outflow tract tachycardia via the coronary sinus. *Heart Rhythm* 2009;**6**:290–291.
6. Daniels D, Lu YY, Morton J, et al. Idiopathic epicardial left ventricular tachycardia originating remote from the sinus of Valsalva: electrophysiological characteristics, catheter ablation, and identification from the 12-lead electrocardiogram. *Circulation* 2006;**113**:1659–1666.
7. Kaseno K, Tada H, Tanaka S, et al. Successful catheter ablation of left ventricular epicardial tachycardia originating from the great cardiac vein. *Circ J* 2007;**71**:1983–1988.
8. Berruezo A, Mont L, Nava S, Chueca E, Bartholomay E, Brugada J. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. *Circulation* 2004;**109**:1842–1847.
9. Giorgberidze I, Saksena S, Krol RB, Mathew P. Efficacy and safety of radiofrequency catheter ablation of left-sided accessory pathways through the coronary sinus. *Am J Cardiol* 1995;**76**:359–365.

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Case report

Acute myocarditis mimicking myocardial infarction can misdirect the diagnostic approach☆

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ABSTRACT

Acute myocarditis is a well-recognized but rare manifestation of mostly viral infections. It can present with various clinical manifestations and may mimic myocardial infarction (MI) since patients usually present with chest pain, and the electrocardiographic changes similar to those observed in acute ST-elevation MI. We, herein, present such an extreme case of acute myocarditis characterized by dynamic ST segment elevation with reciprocal changes in the electrocardiogram.

Introduction

Acute myocarditis is a well-recognized but rare manifestation of mostly viral infections with a broad spectrum of symptoms and clinical features.[1] The diagnosis of acute myocarditis is one of the most challenging issues in cardiology due to the nonspecific pattern of clinical presentation. It can present with various clinical manifestations and may mimic myocardial infarction (MI), since patients usually present with chest pain, and the electrocardiographic changes similar to those observed in acute ST-elevation MI.[2–4] In this report, we present such an extreme case of acute myocarditis characterized by dynamic ST segment elevation with reciprocal changes in the electrocardiogram (ECG).

Case report

A 25-year-old man with no previous cardiac history presented to the emergency department with intermittent retrosternal crushing chest pain radiating to back. On admission, he was pain-free and had a two-day history of symptoms of acute upper respiratory infection including fever, cough, sore throat and nausea. There was no history of smoking, diabetes, hypertension, dyslipidemia or family history of coronary artery disease. On examination, initial heart rate was 84 bpm, temperature 37.4 °C, and arterial blood pressure 115/75 mm Hg. Cardiac examination revealed normal findings, with no additional sounds, murmurs or pericardial rub. ECG on admission revealed sinus rhythm, normal QRS axis and minimal ST-segment elevation in leads II, aVF, I, aVL, V5, and V6 with reciprocal ST segment depression at lead V1 (Fig. 1). An initial echocardiogram showed mild hypokinesia in the apical region of the left ventricle with an ejection fraction of 55%. Cardiac biomarkers revealed serum Troponin I of 37.7 ng/mL (normal < 0.04 ng/mL), creatine kinase (CK) of 2250 U/L (normal 20–171 U/L), and CK-MB of 181 IU/L (normal <0–24 IU/L). White blood cell count was 13,100/mL, and erythrocyte sedimentation rate was 28 mm/h. Other routine biochemical blood tests were within normal limits. With all these findings, our initial diagnosis was acute myocarditis, and we decided to start angiotensin-converting-enzyme inhibitor and beta-blocker therapy because of the mild ventricular dysfunction. After 30–35 min of admission, the patient's chest pain increased progressively. The ECG taken at that time showed 4–5 mm ST-segment elevation in inferior and lateral leads with ST segment depression in right precordial leads (Fig. 2A). Posterior ECG showed that the ST-segment depression in right precordial leads was due to the reflection of ST elevation of the posterior wall (Fig. 2B). Concurrent echocardiography showed an apparent regional hypokinesia in the middle and basal segments of the inferior and posterior walls with an ejection fraction of 45%, whereas

☆ There is no conflict of interest.

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Fig. 1. ECG on admission revealed sinus rhythm and minimal ST-segment elevation in leads II, aVF, I, aVL, V5, and V6 with reciprocal ST segment depression at lead V1. ECG, electrocardiogram.

the ST-segment elevation in especially leads I, aVL was concave seen in pericarditis. Therefore, we performed coronary angiography due to the acute changes. Coronary angiography showed normal epicardial coronary arteries (Fig. 3A) and we observed mild hypokinesia in the apical region of the left ventricle. Over the following days, ST-segment elevations decreased gradually, and ECG revealed biphasic T waves in the leads I, II, aVL, V5 and V6 seen in the subacute phase of myocarditis. The patient discharged on perindopril 5 mg q.d. and metoprolol succinate 50 mg q.d. (Fig. 3B). Follow-up echocardiography at two weeks revealed normal findings without wall motion abnormality.

Discussion

The symptoms and clinical features of myocarditis range from an asymptomatic state to fulminant cardiogenic shock and sudden death. The diagnosis of acute myocarditis, especially when it mimics MI, is challenging due to wide variation in the clinical presentation such as our patient. Although endomyocardial biopsy (EMB) is the gold standard for diagnosis, current guidelines recommend the EMB only in a limited number of clinical scenarios such as life-threatening clinical presentations of myocarditis.[5] In addition, selective coronary angiography is recommended in patients with suspected myocarditis presented with an acute coronary syndrome-like findings. CT coronary angiography may also be an alternative to conventional angiography.[6] Non-invasive imaging techniques such as cardiac magnetic resonance (CMR) imaging can be useful for diagnosis and delineating the extent of the disease.[5,6] However, its usage may be limited regarding availability in acute cases. In the present case, history and clinical presentation (young age, low coronary risk profile, concomitant flu-like symptoms in the few days before admission) were consistent with myocarditis. But the localized ST-segment elevation along with the "reciprocal-like" changes in addition to segmental left ventricular dysfunction and worsening chest pain raised the possibility of an acute MI. Then, the patient underwent coronary angiography for eliminating an acute coronary syndrome. As is seen in our case, confirming the diagnosis with coronary angiography in patients with typical symptoms of acute MI is of great importance especially in young cases with myocarditis or other non-coronary reasons (such as Takotsubo syndrome).[7]

Conclusion

The diagnosis of acute myocarditis is one of the most challenging issues in cardiology. A detailed history, examination and ECG interpretation are usually adequate for diagnosis. However, there may be an extreme similarity regarding ECG findings in cases mimicking acute MI. Therefore, the clinicians should keep in mind that coronary angiography is necessary before taking an action with treatment modalities such as thrombolysis due to serious side effects.

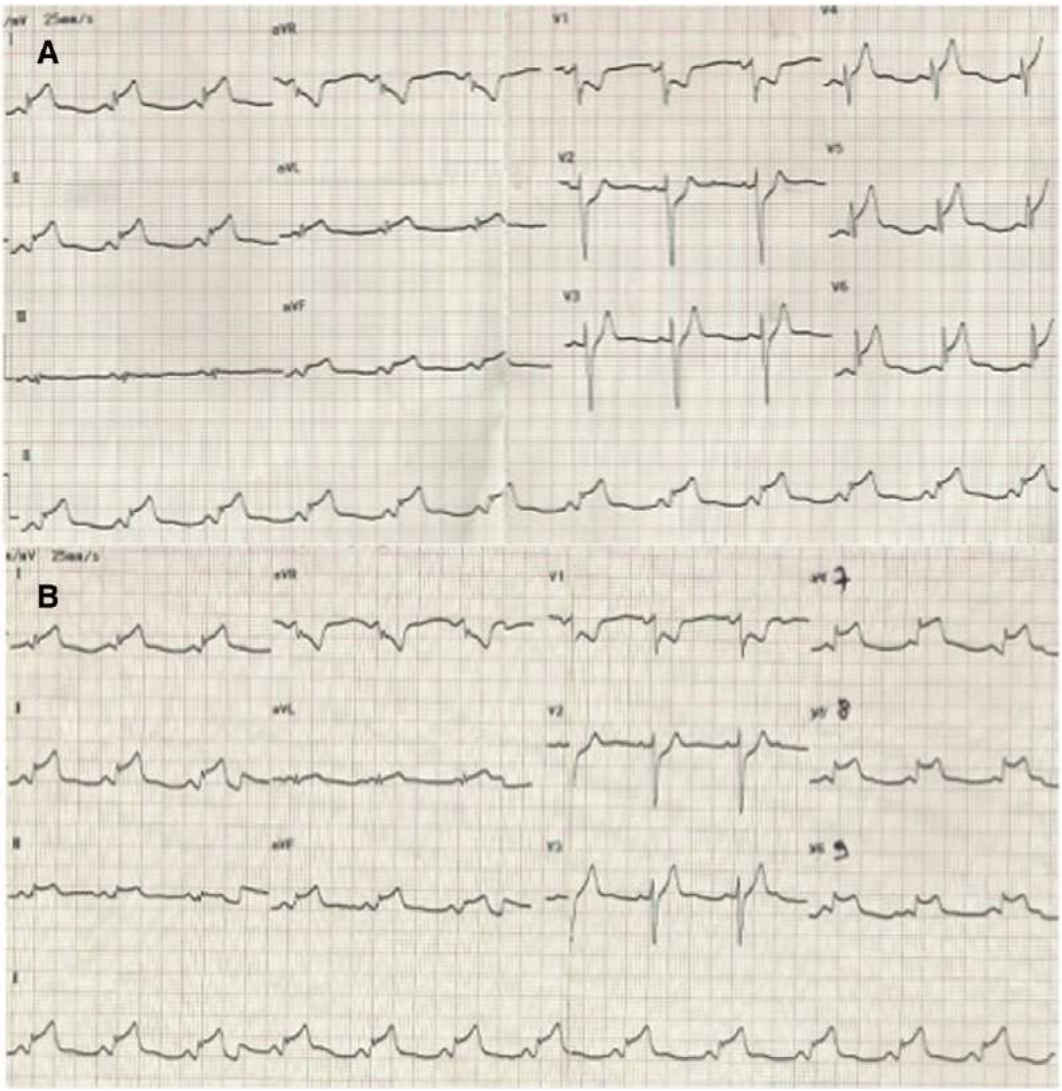

Fig. 2. ECG taken when the patient had chest pain showed 4–5 mm ST-segment elevation in leads I, II, III, aVF, aVL, and V4–V6 with ST segment depression in right precordial leads (A). Posterior ECG showed that the ST-segment depression in right precordial leads was due to the reflection of ST elevation of the posterior wall (B). ECG, electrocardiogram.

References

1. Lieberman EB, Hutchins GM, Herskowitz A, Rose NR, Baughman KL. Clinicopathologic description of myocarditis. *J Am Coll Cardiol* 1991;**18**:1617–1626.
2. Moitard N, Mewion N, Bonnefoy E, Abdellaoui M, Revel D, Kirkorian G. Acute myocarditis mimicking lateral myocardial infarction. *Anaesth Intensive Care* 2008;**36**(5):739–742.
3. Basic D, Gupta S, Kwong RY. Parvovirus B19-induced myocarditis mimicking acute myocardial infarction. *Circulation* 2010;**121**(7):e40–e42.
4. Testani JM, Kolansky DM, Litt H, Gerstenfeld EP. Focal myocarditis mimicking acute ST-elevation myocardial infarction. *Tex Heart Inst J* 2006;**33**(2):256–259.
5. Fridrich MG, Strohm O, Schulz-Menger J, Marciniak H, Luft FC, Dietz R. Contrast media enhanced magnetic resonance imaging visualizes myocardial changes in the course of viral myocarditis. *Circulation* 1998;**97**(18):1802–1809.
6. Caforio ALP, Pankuweit S, Arbustini S, et al, European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J* 2013;**34**(33):2636–2648.
7. Buchholz S, Rudan G. Tako-tsubo syndrome on the rise: a review of the current literature. *Postgrad Med J* 2007;**83**(978):261–264.

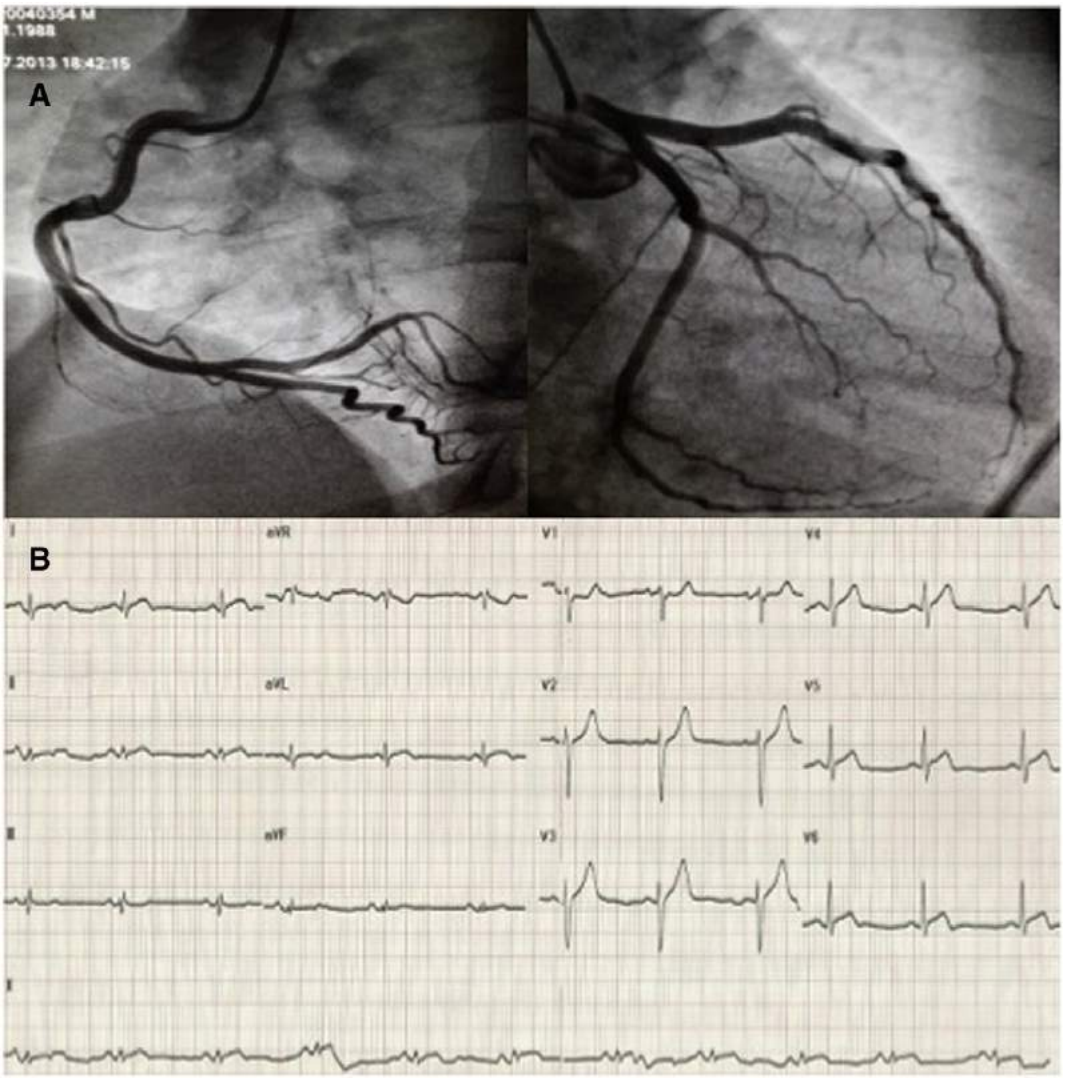

Fig. 3. Coronary angiography showed normal epicardial coronary arteries (A). ECG on discharge revealed biphasic T waves in the leads I, II, aVL, V5 and V6 (B). ECG, electrocardiogram.

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Case report

External jugular vein aneurysm in a young woman: An uncommon cause of neck mass

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ABSTRACT

External jugular vein aneurysms are extremely rare pathologies compared to arterial ones. The patients often present with a painless mass in the neck that becomes visible while coughing and straining. Palpation of a soft and compressible swelling over the external jugular vein is a diagnostic hallmark. Doppler ultrasound examination is considered as the golden standard for the radiological diagnosis that allows a precise determination and confirmation of an aneurysm. Surgical excision is performed mostly for cosmetic concerns and symptomatic aneurysms. In this article, we present the clinical aspects, radiological and microscopic findings, diagnosis and surgical treatment of an external jugular vein aneurysm in a young female patient to emphasize the typical clinical presentation of this rare entity.

Introduction

Venous aneurysms were initially described by Harris in 1928 after the presentation of an infant with a congenital venous cyst of the mediastinum.[1] The terminology of a venous aneurysm was then suggested by Hilscher in 1995 to create a similarity to arterial aneurysms.[2] In contrary to the arterial aneurysms, venous aneurysms are rarely seen. They can be presented in any veins throughout the body including thoracic, cervical, visceral and lower extremity veins. However, due to the low-pressure system of the superior vena cava, the venous aneurysms of the head and neck are rarely encountered than the deep veins of the abdomen and lower limbs.[3] In this paper, diagnosis and the surgical treatment of an external jugular vein aneurysm in a young woman were reported.

Case report

A 19-year-old female was referred for a right neck mass that has been noticed for five years. The mass was non-tender, soft, compressible and bluish and located in the right supraclavicular area. It was clearly visible in rest, but becoming more prominent with Valsalva maneuver (Fig. 1). There was no pulsation or a murmur over the mass that could be heard. The mass has been growing gradually in time and had no association with breathing or swallowing. In her anamnesis, there was a history of a lipoma excision in the right supraclavicular region eleven years ago when she was eight.

The initial diagnosis was a right jugular vein ectasia to explain the increase in size with Valsalva. A cystic lymphangioma extending into the mediastinum was also considered. Ultrasound (US) imaging revealed a fusiform venous structure measuring 2.5 × 3.5 × 1.5 cm on the right anterolateral neck with an existing flow by Doppler US. The mass was over the trace of the right external jugular vein and had no relation to the internal jugular vein. The flow pattern and the morphology of the right common carotid artery were normal. Magnetic resonance angiography imaging showed a round shaped lesion above the right clavicula with a high contrast uptake consistent to a vascular structure (Fig. 2).

Surgical treatment was planned after the diagnosis of a venous aneurysm and the anamnesis of gradual enlargement of the mass. The patient was operated electively with a simple excision of the mass (Fig. 3). It was found to be a right external jugular venous aneurysm consistent with the preoperative diagnosis and measurements (Fig. 4). The pathologic investigation with a microscope revealed an aneurysmatic dilatation of the vein with a focal thinning of smooth muscle wall consistent with phlebectasia. The patient was discharged the next day with no complication and scheduled for a routine follow-up.

Discussion

Venous aneurysms are uncommon causes of the cervical masses when compared to arterial ones.[2] Aneurysms of the neck veins are extremely rare because of the low intravascular pressure in the superior vena cava system.[4] A venous aneurysm can be either primary (congenital) or secondary (acquired). Congenital aneurysms are

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Fig. 1. External jugular vein aneurysm of a 19-year-old woman.

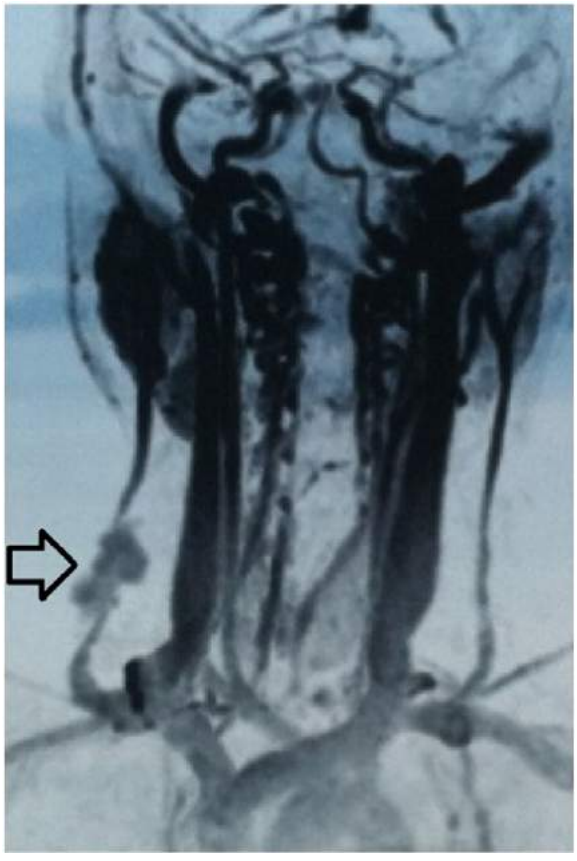

Fig. 2. Magnetic resonance angiography image reveals a venous aneurismatic dilatation on the right external jugular vein.

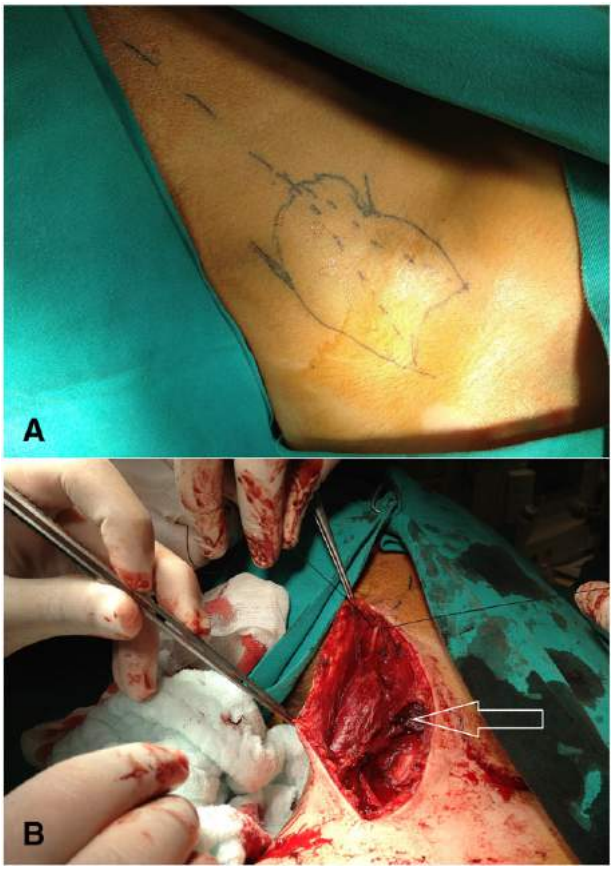

Fig. 3. A. Mapping before the surgical intervention. B. Exploration and delineation of the mass before excision.

considered as true aneurysms as they have an intact venous wall with a weakness of elastic fibers.[2,5] Various etiological mechanisms have been proposed for the development of acquired venous aneurysms. These include trauma, chronic inflammation, degeneration, and increased pressure.[5]

In the differential diagnosis of a cervical mass, cystic hygroma, cavernous hemangioma, a lymphocele, a laryngocele, an enterogenous cyst, thyroid swelling, lymphadenopathy, a thyroglossal cyst, a dermoid cyst and a branchial cleft cyst should be considered.[4,5] Engorgement of the neck swelling during strain eliminates others than a laryngocele and jugular vein aneurysm. The absence of air inside the lesion on plain roentgenography further eliminates the laryngocele.[4]

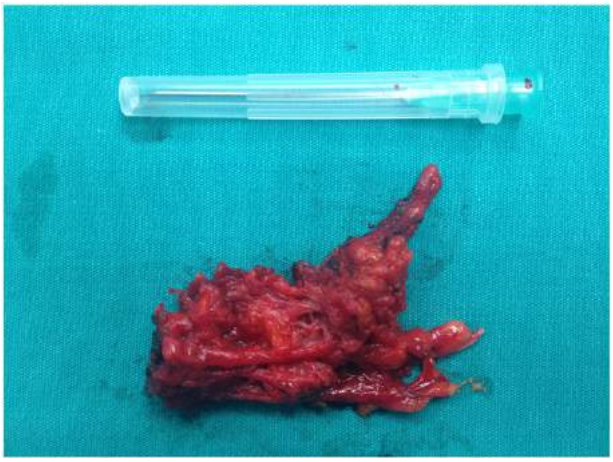

Fig. 4. Macroscopic aspect of the excised external jugular vein aneurysm.

Diagnosis of jugular vein aneurysms can be achieved by color Doppler US imaging (with and without Valsalva), computed tomographic angiography, magnetic resonance angiography imaging, and venography.[6]

Histopathologic examination of the specimen may show a broad spectrum of findings varying from normal venous wall to those unique to phlebectasia.[5] The term phlebectasia has been increasingly recognized in recent years indicating the venous dilatation without tortuosity.[7] Phlebectasia is a condition including the thinning of the venous wall with the loss of smooth muscle cells and their replacement with a fibrous layer as well as the disruption of the elastic layer.[5,8]

Asymptomatic external jugular vein aneurysms can be monitored with regular follow-up. However, surgical excision should be considered for the cosmetic reasons or the presence of a painful aneurysm secondary to phlebitis or thrombosis.[9] Surgical removal also eliminates the theoretical risk of aneurysmal rupture and pulmonary embolism.[10] It also allows surgeons to get the exact histopathological diagnosis. Saccular type of external jugular venous aneurysms can be safely treated by excision and ligation. Whereas, an exclusion and bypass may be needed in a fusiform aneurysm.[9,10] The rarity of this type of venous aneurysms may be related to the asymptomatic nature of them or the tendency of reporting only surgical results.[11]

Conflict of interest

None declared.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

References

1. Harris RI. Congenital venous cyst of the mediastinum. *Ann Surg* 1928;**88**(5):953–957.
2. Ekim H, Özen S. Primary venous aneurysm of the external jugular vein. *EJM* 2002;**7**: 24–25.
3. Regina G, Cardia G, Squeo MA, Lillo A, Latorre M, Odero A. Aneurysm of the internal jugular vein—a case report. *Vasc Surg* 1988;**22**(3):169–171.
4. Kirmani S, Rashid M, Ali I, Badar F. External jugular vein aneurysm: a rare cause of neck swelling. *J Ultrasound Med* 2011;**30**(8):1157–1158.
5. Al-Shaikhi A, Kay S, Laberge JM. External jugular venous aneurysm: an unusual cause of a neck mass in a young child. *J Pediatr Surg* 2003;**38**(10):1557–1559.
6. Karapolat S, Erkut B, Unlu Y. Multiple aneurysms of the left external jugular vein. *Turk J Med Sci* 2005;**35**(1):43–45.
7. Paleri V, Gopalakrishnan S. Jugular phlebectasia: theory of pathogenesis and review of literature. *Int J Pediatr Otorhinolaryngol* 2001;**57**(2):155–159.
8. Cadariu F, Avram J, Enache A, et al. External jugular vein aneurysm: case report. *J Exp Med Surg Res* 2010;**17**:271–274.
9. Ascher E, Salles-Cunha S, Hingorani A. Morbidity and mortality associated with internal jugular vein thromboses. *Vasc Endovasc Surg* 2005;**39**(4):335–339.
10. Mohanty D, Jain BK, Garg PK, Tandon A. External jugular vein aneurysm: case report. *J Nat Sci Biol Med* 2013;**4**(1):223–225.
11. Sander S, Elicevik E, Unal M, Vural O. Jugular phlebectasia in children: is it rare or ignored? *J Pediatr Surg* 1999;**34**(12):1829–1832.

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Case report

Left atrial band confused with cor triatriatum sinister

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A 22-year-old female patient was admitted with complaints of dyspnea on exertion for 3 years. Cardiac examination of the patient in physical examination was normal. ECG was normal. Transthoracic echocardiography (TTE) revealed normal for valve structures, heart chambers, and the LV systolic and diastolic parameters. However, a band-like structure was observed in left atrium (Fig. 1). A band-like structure was observed extending from interatrial septum to the lateral wall of the left atrium in the transesophageal echocardiography (TEE) performed for a more detailed analysis of the left atrium (Fig. 2). A defect was not detected on this structure which was considered as differential diagnosis of a membrane seen in cor triatriatum sinister. In addition, colour Doppler examination did not show turbulent flow in the left atrium (Fig. 3), and there were no gradients in CW and PW examinations. Left atrial band (LAB) is a rare congenital anomaly. Although the incidence of congenital LAB was reported to be 2% in clinico-pathologic study,[1] the number is quite low for echocardiographic examinations. LAB has been studied and shown to be in association with Chiari's network, patent foramen ovale, and mitral valve prolapse; however, it is commonly recognized as a benign entity.[2] The band appearance can also be seen because of suture line after operations like transplantation with biatrial anastomosis. Our patient had no history of surgery. LAB is not uncommon in TTE examination in which left atrium band and cor triatriatum sinister are confused. In discrimination of the two, the defect on this structure with TEE; turbulent flow in the left atrium in colour Doppler; and gradients in CW examination are important discriminants between the two.

Case report

In our case, 22-year-old female patient was admitted to our department with complaints of dyspnea. We detected LAB with TTE incidentally and then confirmed with TEE. It can be confused with cor triatriatum sinister, if it does not examine carefully.

Differential diagnosis

Cor triatriatum sinister.

Imaging diagnosis

LAB can be diagnosed with TTE and TEE.

Treatment

LAB is an anomaly that does not require treatment. However, the treatment of cor triatriatum sinister is surgery.

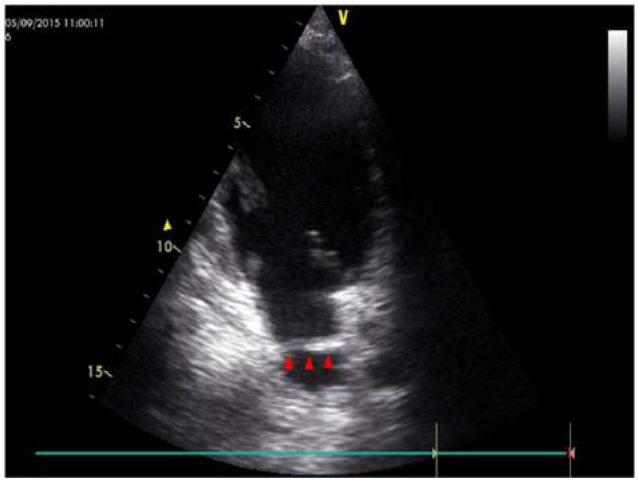

Fig. 1. Transthoracic echocardiogram showing left atrial band (arrowheads).

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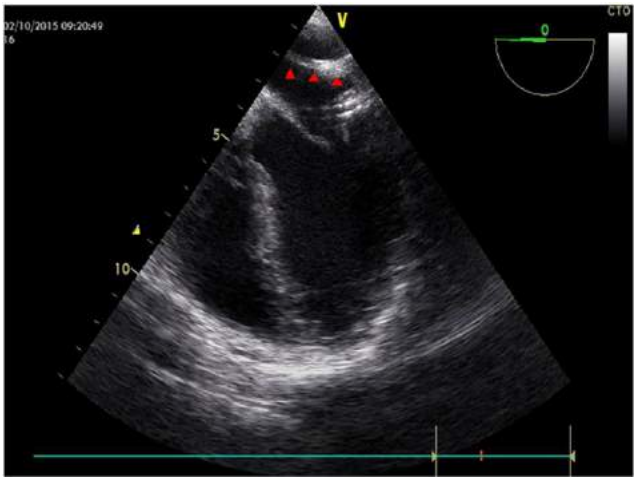

Fig. 2. Transesophageal echocardiogram demonstrating left atrial band extending from interatrial septum to the lateral wall of the left atrium (arrowheads).

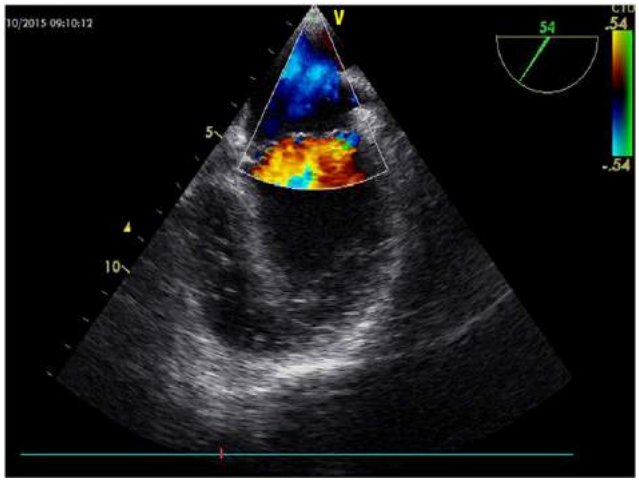

Fig. 3. Colour doppler examination is not showing turbulent flow in the left atrium.

Related reports

Few cases have been reported in the literature about the LAB. But, there is no article about discrimination by cor triatriatum sinister.

Experiences and lessons

It is not uncommon in TTE examination that left atrium band and cor triatriatum sinister are confused. In discrimination of the two, the defect on this structure with TEE; turbulent flow in the left atrium in colour Doppler; and gradients in CW examination are important discriminants between the two.

References

1. Yamashita T, Ohkawa S, Imai T, et al. Prevalence and clinical significance of anomalous muscular band in the left atrium. *Am J Cardiovasc Pathol* 1993;**4**:286–293.
2. Ozer O, Sari I, Davutoglu V, et al. Cryptogenic stroke in two cases with left atrial band: coincidence or cause? *Eur J Echocardiogr* Mar 2009;**10**(2):360–361.

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Case report

Myocardial reinfarction with simultaneous occlusions of two major coronary arteries one of which is due to the early stent thrombosis

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ABSTRACT

Coronary artery stent thrombosis is a rare but often fatal complication associated with percutaneous coronary intervention. Although strict adherence to dual anti-platelet therapy minimizes this risk, stent thrombosis will still occur in rare patients, leading to acute, subacute, or late life-threatening coronary syndromes. Intracoronary thrombosis is a usual finding in acute coronary syndrome but simultaneous formation of the thrombi in two different coronary arteries is very rare. Acute coronary syndrome is not simply the result of isolated local plaque disruption and thrombosis, but rather global coronary vessel inflammation, leading to weakening of atherosclerotic plaques in multiple sites nearly simultaneously. We report a case of myocardial reinfarction with simultaneous occlusion of two major coronary branches one of which is due to the early stent thrombosis.

Introduction

Coronary atherosclerosis and thrombosis are major factors contributing to myocardial infarction. However, simultaneous formation of the thrombi in two different coronary arteries is very rare [1]. Patients with ST segment elevation myocardial infarction (STEMI) frequently have obstructive non-culprit lesions [2]. Stenoses in noninfarct arteries may cause serious adverse cardiac events that could be avoided by performing preventive percutaneous coronary intervention (PCI) during the initial procedure. In patients with STEMI and multivessel coronary artery disease undergoing infarct artery PCI, preventive PCI in noninfarct coronary arteries with major stenoses significantly reduced the risk of adverse cardiovascular events, as compared with PCI limited to the infarct artery [3]. We report a case of myocardial reinfarction with simultaneous occlusion of two major coronary branches one of which is due to the early stent thrombosis. In our case, prior primary PCI for inferior STEMI has been limited to the right coronary artery (RCA) which is infarct artery, and preventive PCI has not been performed to significant stenosis in left circumflex artery (LCX).

Case report

A 69-year-old woman was admitted to emergency department with a sensation of tightness in the chest for 3 h. She had a risk factor of hypercholesterolemia and no other coronary risk factors of smoking, diabetes, hypertension, or positive family history were documented. An electrocardiogram showed ST-segment elevation in leads DII, DIII and aVF (Fig. 1). She underwent cardiac catheterization and emergency angiography revealed significant stenosis in LCX and a total occlusion of RCA after the right ventricular branch (Fig. 2A, B). Left anterior descending artery showed no stenosis. The results of biochemistry laboratory at admission are as follows: Troponin I: 0.56 μg/l (normal range 0–0.023), glucose: 177 mg/dl, creatinine: 0.9 mg/dl, HbA1c: 6.3%, total cholesterol: 236 mg/dl, triglyceride: 106 mg/dl, low density lipoprotein cholesterol: 164 mg/dl, high density lipoprotein cholesterol: 51 mg/dl, hemoglobin: 12.2 g/dl, hematocrit: 36.9%, and platelet: 333×10^3/ml. Balloon angioplasty of the RCA lesion was performed and bare-metal stent was then employed to treat the lesion (Fig. 2C). For anticoagulation, unfractionated heparin was used during the procedure and the patient was loaded with clopidogrel 600 mg orally. There were no complications and the patient tolerated the procedure well. Preventive PCI has not been performed to significant stenosis in LCX. After an uncomplicated 74-hours hospital course, she was discharged with acetylsalicylic acid, clopidogrel, atorvastatin, perindopril, and metoprolol treatment. Four days after discharge, the patient was admitted to the emergency department with chest pain similar to previous. The initial electrocardiogram demonstrated minimal ST elevation and T wave inversion in leads DII, DII and aVF (Fig. 3). The results of

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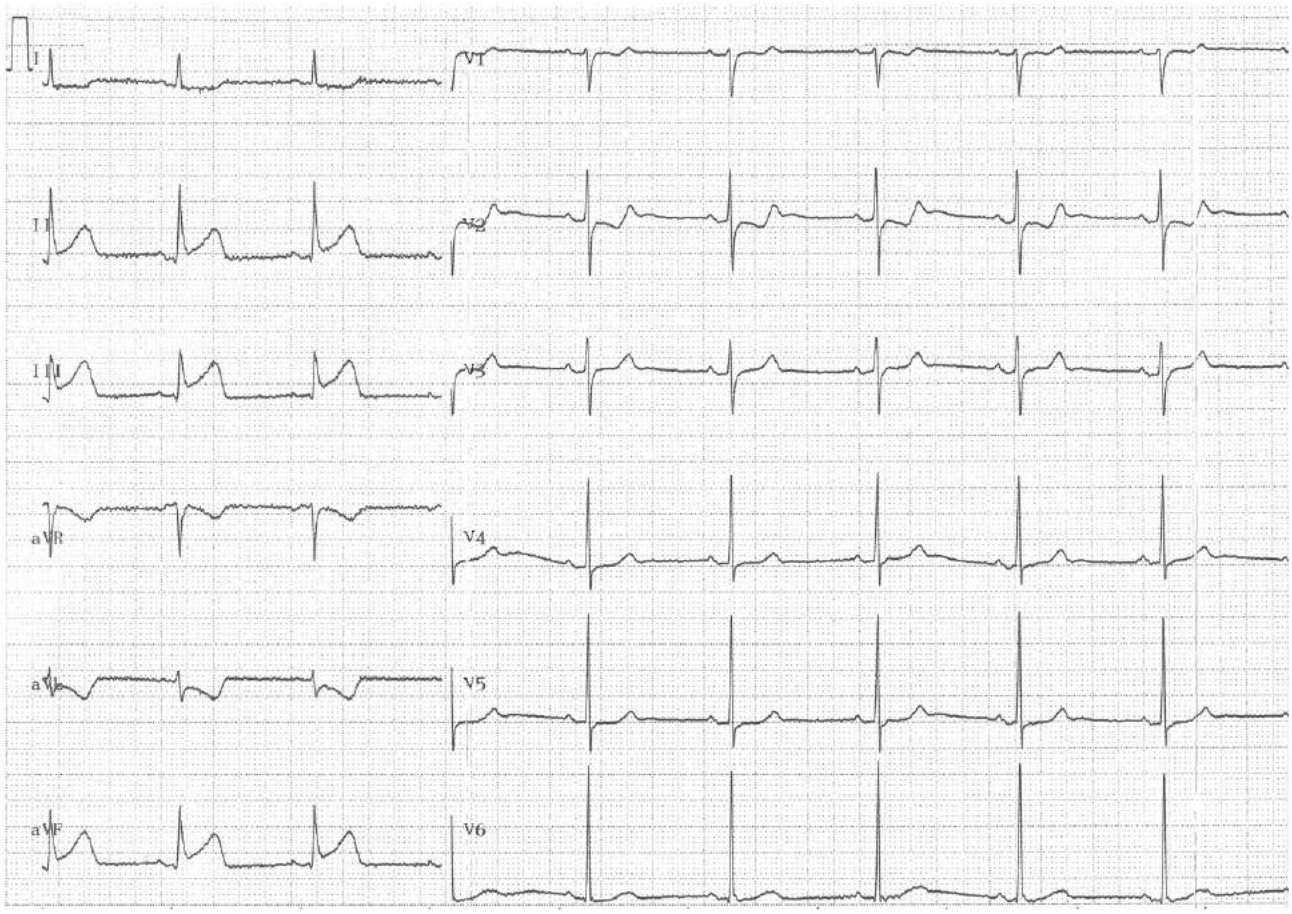

Fig. 1. ECG of the patient at first admission.

biochemistry laboratory at admission are as follows: Troponin I: >80 μg/l (normal range 0–0.023), glucose: 132 mg/dl, creatinine: 0.79 mg/dl, hemoglobin: 11.6 g/dl, hematocrit: 35.2%, and platelet: 370 × 10^3/ml. She was adherent to dual antiplatelet therapy. The patient was diagnosed with myocardial reinfarction probably due to stent thrombosis. A slightly reduced response to clopidogrel (260 P2Y12 resistance units) was shown in platelet function test (target range for sufficient platelet inhibition, <240 P2Y12 resistance units). The platelet inhibition response to aspirin was sufficient. Stent thrombosis – at least partly – was thought to be due to slightly reduced response to clopidogrel. Therefore, the patient was loaded with ticagrelor 180 mg orally before the procedure. Emergency angiography revealed a simultaneous total occlusion of the LCX and an in-stent total thrombotic occlusion of RCA (Fig. 4A, B). Stent in RCA showed no evidence of migration or malposition. The stent occlusion was treated with balloon angioplasty followed by a drug-eluting stent placed distal to the initial stent due to intimal dissection. The LCX occlusion was also treated with balloon angioplasty followed by an adjacent bare-metal stent. For anticoagulation, unfractionated heparin was used during the procedure. She was observed as an inpatient for 3 days and discharged with acetylsalicylic acid, ticagrelor, atorvastatin, perindopril and metoprolol treatment. Recent 1-year follow-up also revealed no significant symptoms and she has been compliant with her dual antiplatelet medication regimen.

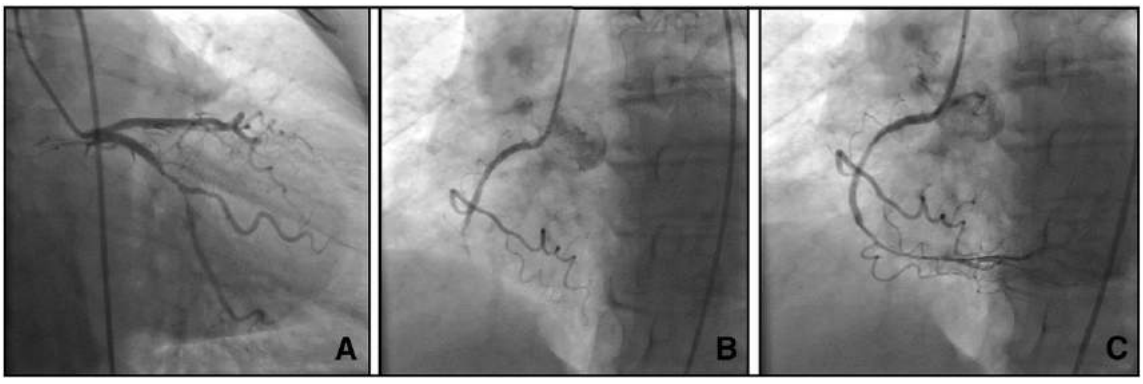

Fig. 2. (A) Significant stenosis in left circumflex artery. (B) Total occlusion of right coronary artery after the right ventricular branch. (C) Right coronary artery after the bare-metal stent implantation.

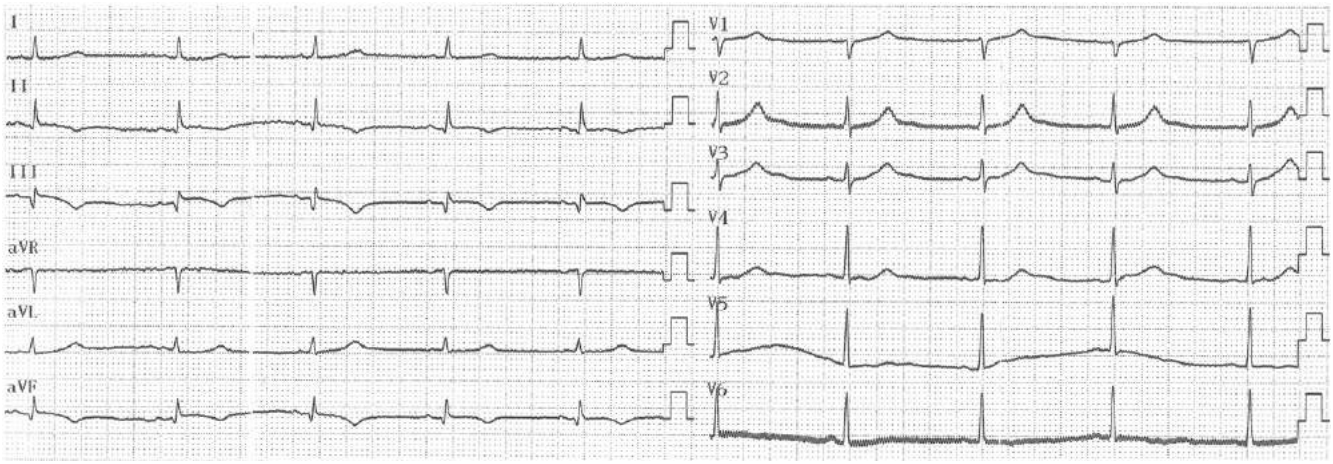

Fig. 3. ECG of the patient at second admission.

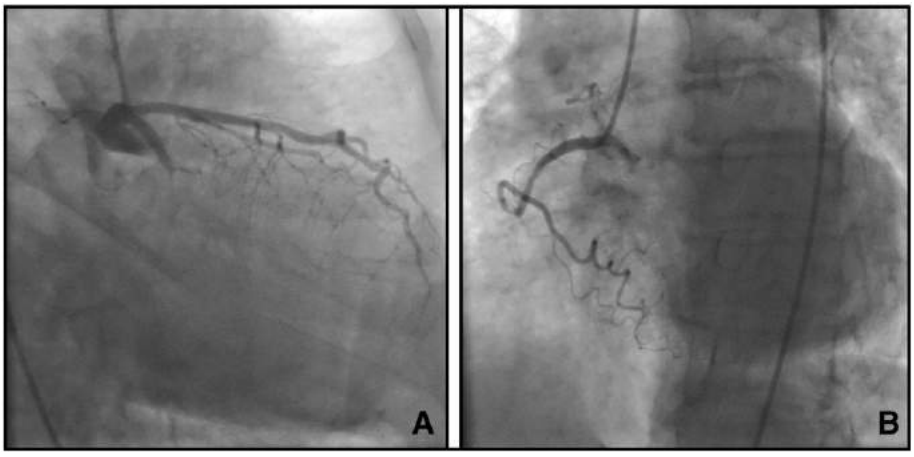

Fig. 4. (A) Total occlusion of left circumflex artery before large obtuse marginal branch. (B) In-stent total thrombotic occlusion of right coronary artery.

Discussion

About 40–50% of people presenting with STEMI have multivessel disease [4,5]. Patients with acute STEMI and multivessel coronary disease have a worse prognosis compared with individuals with single-vessel disease. Compared with those with single-vessel disease, patients with multivessel disease had a higher frequency of recurrent ischemia at 30-days [6]. Also they have higher mortality rates and a greater incidence of non-fatal reinfarction [6–8]. It is unclear whether this is attributable to an increased disease burden or because relevant lesions in other areas are left untreated.

Coronary artery stent thrombosis is a rare but often fatal complication associated with PCI. Stent thrombosis can occur within days of stent placement (acute), up to 1 month post-procedure (subacute), or later (late). Although strict adherence to dual anti-platelet therapy minimizes this risk, stent thrombosis will still occur in rare patients, leading to acute, subacute, or late life-threatening acute coronary syndromes. Acute stent thrombosis is generally caused by the following mechanisms: antithrombotic drug resistance, hypercoagulable states, stent malposition/underdeployment, or coronary arterial damage during initial PCI like intimal dissection. The exact etiology of stent thrombosis, however, is often multi-factorial and difficult to comprehend.

Several studies have demonstrated the pathogenetic role of local thrombus formation in coronary arteries at the site of a ruptured plaque. Plaque disruption leads to platelet activation and to thrombin generation. Although intracoronary thrombosis is a usual finding in STEMI, simultaneous formation of the thrombi in two different coronary arteries is very rare. In our case, RCA was occluded by stent thrombosis and LCX was occluded by thrombus formation in significant stenotic lesion. The state of hypercoagulability and vasospasm caused by acute stent thrombosis may lead to thrombus formation and acute occlusion in LCX lesion. Atherosclerotic plaques in multiple sites may be weakened nearly simultaneously by global coronary vessel inflammation caused by acute coronary syndrome. Of course, the exact opposite is also possible. A new isolated local plaque disruption and thrombus formation in LCX lesion may have triggered the stent thrombosis in RCA via hypercoagulability. Inflammatory mediators in acute coronary syndromes could serve to facilitate a generalized multi-vessel prothrombotic state. Therefore, in patients with STEMI and multivessel coronary artery disease undergoing infarct artery PCI, preventive PCI in noninfarct coronary arteries with significant stenoses seems to be a rational approach.

Conflicts of interest

None.

References

1. Nakagawa T, Yasuno M, Tanahashi H, et al. A case of acute myocardial infarction. intracoronary thrombosis in two major coronary arteries due to hormone therapy. *Angiology* 1994;**45**(5):333–338.
2. Goldstein JA, Demetriou D, Grines CL, Pica M, Shoukfeh M, O'Neill WW. Multiple complex coronary plaques in patients with acute myocardial infarction. *N Engl J Med* 2000;**343**(13):915–922.
3. Wald DS, Morris JK, Wald NJ. et al.; Randomized trial of preventive angioplasty in myocardial infarction. *N Engl J Med* 2013;**369**(12):1115–1123.
4. Kelbaek H, Terkelsen CJ, Helqvist S, et al. Randomized comparison of distal protection versus conventional treatment in primary percutaneous coronary intervention: the drug elution and distal protection in ST-elevation myocardial infarction (DEDICATION) trial. *J Am Coll Cardiol* 2008;**51**(9):899–905.
5. Rasoul S, Ottervanger JP, de Boer MJ, et al. Zwolle Myocardial Infarction Study Group. predictors of 30-day and 1-year mortality after primary percutaneous coronary intervention for ST-elevation myocardial infarction. *Coron Artery Dis* 2009;**20**(6):415–421.
6. Sorajja P, Gersh BJ, Cox DA, et al. Impact of multivessel disease on reperfusion success and clinical outcomes in patients undergoing primary percutaneous coronary intervention for acute myocardial infarction. *Eur Heart J* 2007;**28**(14):1709–1716.
7. Halkin A, Singh M, Nikolsky E, et al. Prediction of mortality after primary percutaneous coronary intervention for acute myocardial infarction: the CADILLAC risk score. *J Am Coll Cardiol* 2005;**45**(9):1397–1405.
8. Muller DW, Topol EJ, Ellis SG, Sigmon KN, Lee K, Califf RM. Multivessel coronary artery disease: a key predictor of short-term prognosis after reperfusion therapy for acute myocardial infarction. Thrombolysis and angioplasty in Myocardial Infarction (TAMI) Study Group. *Am Heart J* 1991;**121**(4 Pt 1):1042–1049.

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Case report

A practical solution for coil migration and coronary artery dissection in the same patient with coronary artery fistula

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Migration

ABSTRACT

Coronary artery fistulas (CAF) are defined as a direct communication between a coronary artery and any cardiac chamber or vessel. They are generally congenital and the majority occurs in the right coronary artery (RCA) almost always draining to the right cavities and exact incidence of CAF is unknown. The treatment options for CAF are surgery or catheter closure. Catheter closure of the CAF is now considered to be an effective and safe method alternative to surgery. We report the first case with coil migration and coronary dissection to occur simultaneously in the same patient and successful treatment of these two complications with stent implantation.

A 68-year-old man was admitted to our department with complaints of exertional dyspnea and chest pain. Coronary angiography did not show significant lesions in the epicardial coronary arteries and a fistula was observed from the proximal portion of RCA draining into the pulmonary artery (Fig. 1). For embolisation a 0.014î and 300 cm hydrophilic guidewire was moved up to the distal segment of the fistula and a microcatheter was then loaded over this and a 2 × 25 mm coil (Balt, Montmorency, France) was placed. For total occlusion achievement a second 2 × 50 mm coil (Balt, Montmorency, France) was placed (Fig. 2). Unfortunately soon after the insertion of the second coil, during contrast injection this coil detached and migrated to proximal portion of RCA. At the same time coronary dissection was observed in the osteal part of RCA (Fig. 3). Therefore, 4.0 × 9 mm bare metal stent (Simchrome CoCr, Simeks, Turkey) was successfully implanted at 14 atm to cover the dissected segment and migrated coil was jailed to side branch to prevent any embolization to the distal portion of RCA or aorta immediately (Fig. 4). Electrocardiogram and Troponin levels were normal and patient was discharged after a 72-hour follow-up without any complication.

The clinical symptoms of CAF depend on the severity of the left–right shunt and the coronary steal. Closure is indicated for symptomatic

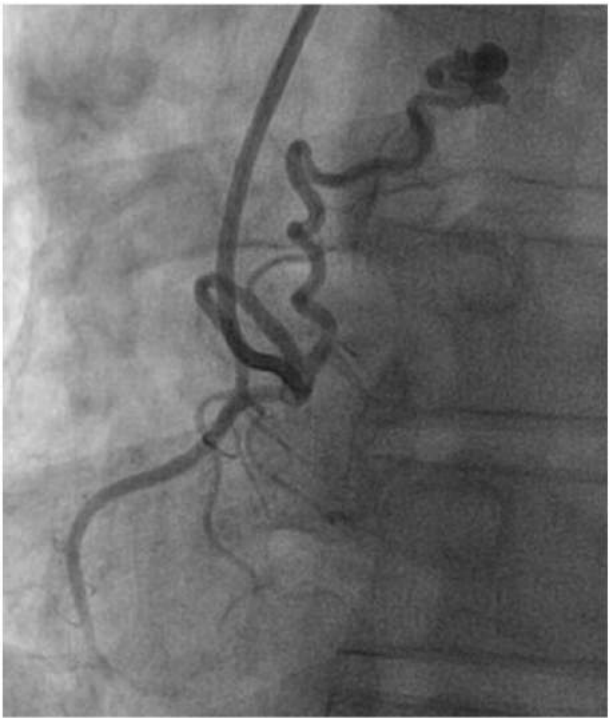

Fig. 1. A fistula originating from the proximal portion of RCA draining into the pulmonary artery.

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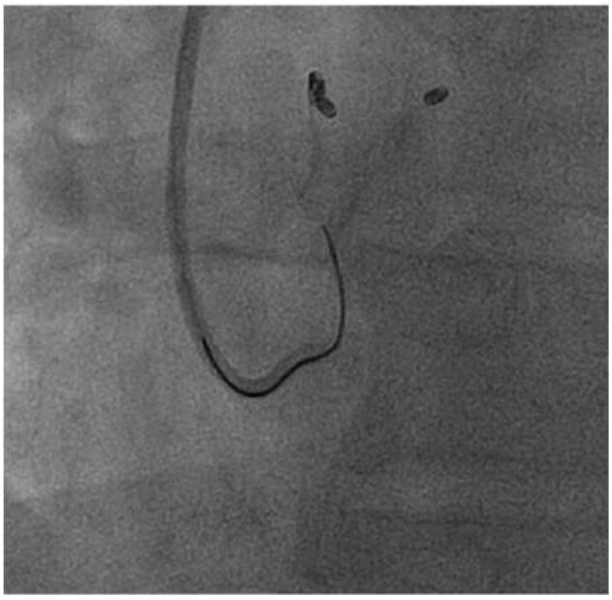

Fig. 2. Second coil placement.

patients and fistulas with a high flow rate. Percutaneous closure is the preferred technique, in particular in the absence of multiple fistulas and large fistula branches, and in cases where the fistula can be easily accessed.[1,2] With catheter closure techniques, complete occlusion of the fistula may be achieved in >95% of the patients. The main complications include either premature deflation of a detachable balloon, inadvertent coil embolisation, transient T-wave changes, transient bundle branch block and myocardial infarction. All the complications are rare, except for inadvertent coil migration, which may occur as a result of high flow in the large fistulas or with undersized coils.[3,4] In our patient migration of the coil was due to fast contrast medium injection. Even if the coils do migrate, they can be pulled back with snares. In our knowledge this is the first case with coil migration and coronary dissection to occur simultaneously in the same patient.

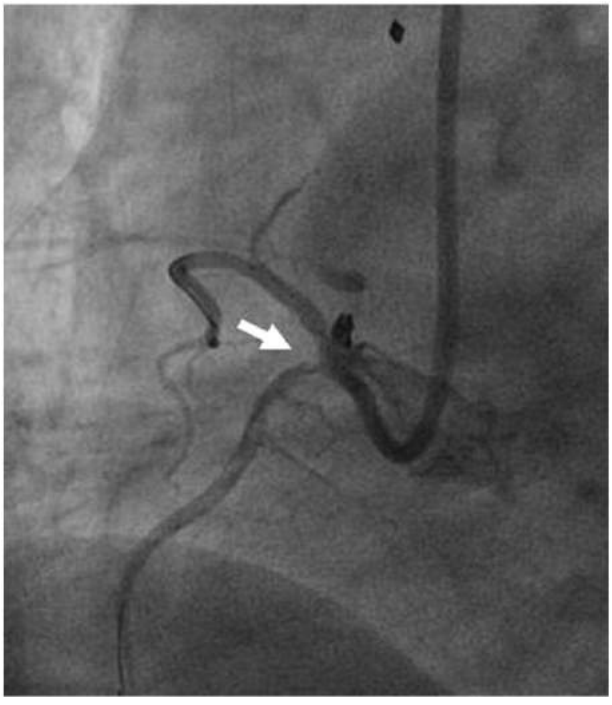

Fig. 3. Coil migration and coronary dissection in the osteal part of right coronary artery (arrow).

Fig. 4. Bare metal stent implantation to cover the dissected segment and to jail migrated coil (arrow).

This case shows the unusual complication of percutaneous closure of coronary artery fistulas and successful management of this complication using stent implantation.

Conflict of interest

None.

References

1. Gowda RM, Vasavada BC, Khan IA. Coronary artery fistulas: clinical and therapeutic considerations. *Int J Cardiol* 2006;**107**:7–10.
2. Jiang Z, Chen H, Wang J. Right coronary artery fistula to left ventricle treated by transcatheter coil embolization: a case report and literature review. *Intern Med* 2012;**51**: 1351–1353.
3. Reidy JF, Anjos RT, Qureshi SA, Baker EJ, Tynan MJ. Transcatheter embolization in the treatment of coronary artery fistulas. *J Am Coll Cardiol* 1991;**18**:187–192.
4. Qureshi SA, Reidy JF, Alwi MB, et al. Use of interlocking detachable coils in embolizationof coronary arteriovenous fistulas. *Am J Cardiol* 1996;**78**:110–113.

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Review

The effect of knowledge about hypertension on the control of high blood pressure

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ABSTRACT

Objective: The aim of this study was to search the effect of knowledge about hypertension and socio-demographic characteristics on controlling high blood pressure levels among patients diagnosed with hypertension.
Methods: This is a cross-sectional study. The study population was obtained from subjects diagnosed with hypertension and applied to primary health care centers in Yozgat province center, in 2013. The subjects with informed consent were enrolled into the study (n = 485). The data were collected via 15-item hypertension knowledge questionnaire and personal information survey prepared in accordance with the literature. The knowledge level was classified as follows: low (<8 points); moderate (8–11 points); adequate (≥12 points).
Results: Frequencies of low, moderate and high level of knowledge about hypertension were 31.3%, 62.1% and 6.6% respectively. The effects of other socio-demographic parameters on the knowledge level were not significant even after multivariate analysis. Knowledge level was positively related to ratio of subjects with blood pressure under control but not significant ($p > 0.05$).
Conclusion: Majority of the subjects had inadequate knowledge about hypertension, two third of the subjects did not imply significant life style modification for hypertension.

Contents

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Introduction

High blood pressure (HBP) is a leading major risk factor for chronic diseases and deaths. The prevalence of patients with hypertension (HT) had reached from 600 million in 1980 to one billion in 2008.[1] The prevalence of HBP was approximately 40% among adults of 25 years and above in 2008. Approximately 7.5 million people (12.8% of all-cause deaths) die every year due to HBP.[2] It is estimated that HT is responsible for 45% of deaths due to heart diseases and 51% of deaths due to stroke. HBP consists of 3.7% of Disability Adjusted Life Years (DALY).[1] Even prehypertension (PreHT) increases mortality risk due to cardiovascular and stroke-related diseases.[3]

According to Turkey Burden of Disease Study (TBDS) 2004, the leading factor for DALY was found to be HBP among seven most frequent factors. It was estimated that the prompt control of this factor would prevent approximately one of every four (25.2%) deaths.[4] According to the data derived from Turkish Statistics Institute (TSI) 2013, 39.8% of all deaths stem from circulation-related disorders and 12.8% of these deaths were related to HT.[5] The prevalence of hypertension is 32.2% among males and 30.5% among females in developing countries while these values are 40.8% and 33.0% respectively in developed countries.[6] In our country HT prevalence is found to be 35.1% according to the results of SALTurk trial held in 2008, it was 31.3% according to TURDEP II trial held in 2010 and it was 30.3% according to Turkish Society of Hypertension and Kidney Diseases[7] made PatentT2 trial held in 2012.[7–9] It seems that increasing trend in HT prevalence is stopped in recent years in Turkey.

The ratio of taking BP under control among hypertensive patients is 28.7% in our country while it is 29.6% among males and 34% among females in developing countries and 33.2% among males and 38.4% among females in developed countries respectively.[6,7] Chronic Diseases and Risk Factors Survey (CDRFS) in Turkey 2011 revealed that 85% of patients with hypertension used medication and 72% of hypertensive patients under medication had blood pressure values within targeted levels.[10]

The studies held in Pakistan revealed that hypertensive patients had inadequate knowledge about HT and the frequency of patients with BP under control was higher among the patients with high level of knowledge about HT.[11,12] Similarly, the study held in USA also showed that hypertensive patients had the low level of knowledge about the health issues.[13] In Turkish population, it was also found that the participants had the low level of knowledge about HT.[14]

WHO calls attention to importance of primary health care in struggle against HT and it pointed that health professionals, especially nurses, should take part in creating awareness among the society members and they should take active role in organizing educational meetings about risk factors.[1,15]

Purpose

The aim of this study was to search the relation between knowledge about HT, socio-demographic characteristics and obtaining controlled BP levels among patients diagnosed with HT.

Conceptual framework of this study

In this study, effects of HT knowledge level and socio-demographic factors on HT control were made by multivariate analysis that examined which variables were more important.

Materials and methods

Study design

This was a cross-sectional study aimed at identifying the knowledge about HT which impacts the controlled BP levels of patients with HT.

Setting and samples

The study population was obtained from subjects diagnosed with HT and applied to primary health care centers in Yozgat province center between January 2013 and December 2013. Random cluster sampling method was used to determine sample size. There had been 7 Family Health Centers (FHC) in the province center at the time of sampling. Of them, three FHCs (1, 3, and 5) were selected randomly for sampling. According to TSI 2012 data,[16] Yozgat province center has 78,328 residents and the number of people aged above 18 years old was approximately 56,000. SaltTurk trial reported the frequency of HT prevalence among adults over 18 years old as 35%.[8] Considering all these data, the expected number of people with HT above 18 years old was 19,600 people. Size of the sample was determined as follows: The estimated sampling universe (people with HT) = 19,600; the ratio of subjects with BP under control = 30% (according to CDRFS results)[10]; deviation of this ratio = 0.05; and the probability of a making type I error (α) = 0.05. As a result, the estimated sample size was found to be 343 people. Since multivariate analyses were planned for BP levels, level of knowledge about HT and socio-demographic characteristics, it was decided to increase the number of subjects 50% more than the estimated sample size. Finally, 485 subjects were enrolled into the study.

Ethical consideration

Informed consent of each subject, ethical and official approval from the local authorities were obtained for the study accordingly and the investigation was performed in accordance with the principles outlined in the Declaration of Helsinki.

Measurement

A questionnaire was prepared by the researchers in accordance with the literature to measure the level of knowledge about HT. The questionnaire had 15 items as follows: three questions about BP classification; four questions about HT complications, four questions about treatment and BP control; and four questions about signs and follow-up of HT.[17] And also a survey was formed to get data about socio-demographic characteristics of the subjects. Total scores for the questionnaire about HT knowledge ranged from 0 to 15 at maximum. The subjects were classified further in respect to the scores obtained from the questionnaire as follows: low level of knowledge <8 points; moderate level of knowledge 8 to 11 points; and adequate level of knowledge ≥12 points.[17]

Procedure

The questionnaire and survey were fulfilled by the subjects under the observation of an independently trained interviewer. Height and weight of all subjects were measured with automatic digital scale at morning times between 08:00 to 11:00 o'clock under casual clothes without shoes. The blood pressure (BP) levels were measured from the right and left arms of the subjects in a sitting position by one trained observer blind to the study at the place of interview. BP was measured twice with 10 min interval. The systolic BP (SBP) and diastolic BP (DBP) were recorded at the first and fifth Korotkoff phases respectively using a mercury sphygmomanometer. The average of the four BP measurements was used for analysis. BP levels were classified in accordance with Joint National Committee Report-7 as follows: normal: SBP <120 mmHg and DBP <80 mmHg; PreHT: SBP 120–139 mmHg and/or DBP 80–89 mmHg; HT: ≥140 mmHg and/or ≥90 mmHg.[18] The Joint National Committee Report-8 recommend, in the general population aged 60 years or older, initiate pharmacologic treatment to lower BP at SBP of 150 mmHg or higher or DBP of 90 mmHg or higher and treat to a goal SBP lower than 150 mmHg and goal DBP lower than 90 mmHg.[19] The subjects who have BP measurement of SBP <140 mmHg (≥60 age,

SBP <150 mmHg) and/or DBP <90 mmHg at the time of interview were classified as subjects with BP under control.[20] Body Mass Index (BMI) was calculated by the formula (BMI = weight in kg/(height in m)^2) defined according to the WHO criteria.[21] Patients were classified as normal weight (BMI 18.5–24.9 kg/m^2), overweight (BMI 25.0–29.9 kg/m^2), obese class I (BMI 30.0–34.9 kg/m^2), obese class II (BMI 35.0–39.9 kg/m^2) or obese class III (BMI 40 kg/m^2 or more).

Statistical analysis

The data were analyzed with SPSS package program. Statistical analyses were carried out by the help of simple correlation tests and backward LR model of multiple variables binary logistic regression (BLR).[22] In the BLR analysis, the state of being under the control of BP (coded; controlled: 1, uncontrolled: 0) was used as the dependent variable. The independent variables; HT knowledge score, age, BMI, and disease duration as a continuous variable, education level, perception of income level, and level of satisfaction in life as an ordinal variable, gender, marital status and employment status were included in the model as a categorical variable. Continuous variables were expressed in mean ± SD, categorical variables were shown in percentage, p-values of <0.05 was used to show statistical significance.

Results

The study included 485 subjects. 57.7% of the subjects were female and 85.5% of study population were aged 50 years old and above with a range of 18 to 90 years old. Median age was 63 years old with the average age of 61.5 ± 10.9 years. 83.5% of the study population were married, 28.2% of them had education duration of 5 years or less, however prevalence of subjects graduated from secondary school or above was 12.8%. 10.1% of the subjects were employed. 75.1% of them claimed that they satisfied with quality of their life. 26% of the study population reported that their economic status were well enough while 6.4% of them claimed that they had low economical status (Tables 1, 2).

It was found that 36.5% of the subjects with HT consumed enough fruit/vegetables (≥3 times a day), 31.3% of them exercised sufficiently (at least 3 days and 150 min per week) while 52.0% of the study population did not exercise at all. 25.6% of all hypertensive subjects generally did not consume salt while 34.4% of them claimed usage of low level salt on their diet. 10.5% of hypertensive subjects were a current smoker (Table 3), 65.2% of the study population had one of the concurrent comorbidities (such 6.7% with diabetes). Ratio of parental HT was 47.4% (Table 2).

According to results of the questionnaires, we found that frequencies of poor, moderate, and adequate level of knowledge about HT among subjects with HT were 31.3%, 62.1%, and 6.6% respectively. Level of knowledge was correlated significantly with educational status but not with other variables (Table 1). Among the study population, BP value and HT duration did not show any statistical correlation between score of the HT knowledge. Also, the knowledge score mean of the subjects did not significantly differ in respect to gender, marital status, and economic status however HT knowledge scores mean was statistically different with age clusters and educational status (Table 1). HT knowledge score of old subjects aged 70 years and above had significantly lower than other age groups' scores (posthoc LSD, $p < 0.05$). Other age clusters didn't differ in respect to each other ($p > 0.05$). Average knowledge score of subjects without formal education had significantly lower than that of subjects with primary school and above graduate (posthoc Tamhane, $p < 0.05$) while other educational states did not show any significant difference in respect to each other ($p > 0.05$). In multivariate regression analyses, considering age (in year) and educational states (in ordinal value) as independent variables, the contribution of educational states on HT knowledge score was statistically significant ($B = 0.408$, $p < 0.001$) while contribution of age did not significantly contribute to HT knowledge score ($B = -0.013$, $p = 0.167$). Educational status contributed only 4.5% of changes in HT knowledge score ($R^2 = 0.045$).

Table 1
Hypertension knowledge distribution according to socio-demographic properties in the study population.

Risk factors	HT knowledge level [a]				
Gender	n (%) [b]	X ± SD	Poor n (%)	Moderate n (%)	Adequate n (%)
Male	205 (42.3)	8.5 ± 2.1	59 (28.8)	133 (64.9)	13 (6.3)
Female	280 (57.7)	8.3 ± 2.4	93 (33.2)	168 (60.0)	19 (6.8)
Age groups	t = 1.24, p = 0.22			$X^2 = 1.23$, P = 0.540	
<50	69 (14.2)	8.7 ± 2.1	22 (31.9)	41 (59.4)	6 (8.7)
50–59	124 (25.6)	8.5 ± 2.0	39 (31.5)	76 (61.3)	9 (7.3)
60–69	177 (36.5)	8.5 ± 2.3	48 (27.1)	120 (67.8)	9 (5.1)
≥70	115 (23.7)	7.8 ± 2.5	43 (37.4)	64 (55.7)	8 (7.0)
Education	F = 3.42, p = 0.02			$X^2 = 5.33$, P = 0.503	
Illiterate	137 (28.2)	7.6 ± 2.6	58 (42.3)	75 (54.7)	4 (2.9)
Primary school	234 (48.2)	8.6 ± 2.1	69 (29.5)	150 (64.1)	15 (6.4)
Secondary school	52 (10.7)	8.8 ± 2.0	12 (23.1)	36 (69.2)	4 (7.7)
High school or more	62 (12.8)	9.1 ± 1.8	13 (21.0)	40 (64.5)	9 (14.5)
Marital status	F = 8.7, p < 0.001			$X^2 = 19.44$, P = 0.003	
Married	405 (83.5)	8.5 ± 2.2	121 (29.9)	255 (63)	29 (7.2)
Not married	80 (16.5)	8.0 ± 2.5	31 (38.8)	46 (57.5)	3 (3.8)
Economic level	t = 1.73, p = 0.085			$X^2 = 3.18$, P = 0.204	
High	126 (26.0)	8.5 ± 2.1		33 (26.2)	10 (7.9)
Middle	328 (67.6)	8.4 ± 2.3	107 (32.6)	200 (61.0)	21 (6.4)
Low	31 (6.4)	7.8 ± 2.8	12 (38.7)	18 (58.1)	1 (3.2)
Current BP level	F = 1.1, p = 0.341			$X^2 = 3.11$, P = 0.540	
Normal	105 (21.6)	8.3 ± 2.4	35 (33.3)	65 (61.9)	5 (4.8)
PreHT	224 (46.2)	8.5 ± 2.2	61 (27.2)	144 (64.3)	19 (8.5)
HT	156 (32.2)	8.2 ± 2.3	56 (35.9)	92 (59.0)	8 (5.1)
HT duration	F = 1.2, p = 0.292			$X^2 = 8.48$, P = 0.076	
<5 years	157 (32.4)	8.5 ± 2.1	49 (31.2)	96 (61.1)	12 (7.6)
5–9 years	120 (24.7)	8.3 ± 2.5	36 (30.0)	76 (63.3)	8 (6.7)
10–14 years	98 (20.2)	8.5 ± 2.1	29 (29.6)	63 (64.3)	6 (6.1)
≥15 years	110 (22.7)	8.1 ± 2.4	38 (34.5)	66 (60.0)	6 (5.5)
	F = 0.8, p = 502			$X^2 = 1.24$, P = 0.975	
Total	485 (100.0)	8.4 ± 2.3	152 (31.3)	301 (62.1)	32 (6.6)

[a] HT knowledge score <8 is poor, 8–11 is moderate, ≥12 is adequate. [b] Percentages are sum of columns t: Independent Student t test, F: One way ANOVA.

Only some of subjects knew that HBP does not show sign (7.4–30.7%) and HT does not lead any cancer (23.5%). When asked about the definition of normal BP, preHT and HT, respectively 65.2%, 49.7% and 72% of the subjects answered the relevant BP classification correctly. Most of the subjects knew HBP lowering methods correctly (71.3–80.6%) and the necessity of regular intake of antihypertensive drugs (95.7%).

According to blood pressure measurement results of the subjects, 21.6% of the subjects had normal BP, 46.2% with preHT and 32.2% with hypertensive level of BP. BP level of the subjects did not reveal any significant relation to age, gender, educational, marital, employment and economic states ($p > 0.05$ for all comparisons). Although subjects with hypertensive level of BP measurement had lower knowledge score compared to normal and preHT levels it did not reach the level of statistical significance ($p > 0.05$). Subjects with higher BMI value had significantly higher frequency of having hypertensive BP level ($p < 0.05$). 25.5% of subjects with BMI less than 25 kg/m^2 and 35.5% of subjects with BMI 30 kg/m^2 and more had hypertensive level of BP measurement (Table 2). The case of classifying the subjects into hypertensive BP measurement and the others, BMI values did not show significant difference between the groups ($X^2 = 2.64$, $p = 0.267$).

It was found that HT duration of the subjects was less than 5 years in 32.4% of the subjects and ≥15 years in 22.7% of the subjects. Subjects with HT duration of less than 5 years had significantly higher frequency of hypertensive BP measurement compared to subjects with HT duration of ≥15 years (35.0% vs. 30.9% respectively, $p < 0.05$).

Table 2
Blood pressure levels distribution according to socio-demographic properties in the study population.

Characteristics	Current blood pressure level			
	Normal	PreHT	HT	Total
Gender	n (%)[a]	n (%)[a]	n (%)[a]	n (%) [b]
Male	47 (22.9)	94 (45.9)	64 (31.2)	205 (42.3)
Female	58 (20.7)	130 (46.4)	92 (32.9)	280 (57.7)
Age groups (year)	$X^2 = 0.37$ P = 0.829			
< 50	15 (21.7)	35 (50.7)	19 (27.5)	69 (14.2)
50–59	24 (19.4)	49 (39.5)	51 (41.1)	124 (25.6)
60–69	42 (23.7)	87 (49.2)	48 (27.1)	177 (36.5)
≥70	24 (20.9)	53 (46.1)	38 (33.0)	115 (23.7)
Education	$X^2 = 7.51$ P = 0.276			
Illiterate	26 (19.0)	69 (50.4)	42 (30.7)	137 (28.2)
Primary school	56 (23.9)	97 (41.5)	81 (34.6)	234 (48.2)
Secondary school	11 (21.2)	24 (46.2)	17 (32.7)	52 (10.7)
High school or more	12 (19.4)	34 (54.8)	16 (25.8)	62 (12.8)
Marital status	$X^2 = 5.15$ P = 0.525			
Married	86 (21.2)	187 (46.2)	132 (32.6)	405 (83.5)
Not married	19 (23.8)	37 (46.3)	24 (30.0)	80 (16.5)
Parental HT	$X^2 = 0.34$ P = 0.846			
Have not	60 (23.5)	120 (47.1)	75 (29.4)	255 (52.6)
Have	45 (19.6)	104 (45.2)	81 (35.2)	230 (47.4)
BMI (kg/m^2)	$X^2 = 2.23$ P = 0.327			
<25	21 (41.2)	17 (33.3)	13 (25.5)	51 (10.5)
25–29.9	45 (22.5)	95 (47.5)	60 (30.0)	200 (41.2)
≥30	39 (16.7)	112 (47.9)	83 (35.5)	234 (48.2)
Economic level	$X^2 = 15.57$ P = 0.004			
High	27 (21.4)	60 (47.6)	39 (31.0)	126 (26.0)
Middle	70 (21.3)	157 (47.9)	101 (30.8)	328 (67.6)
Low	8 (25.8)	7 (22.6)	16 (51.6)	31 (6.4)
HT knowledge level	$X^2 = 8.16$ p = 0.068			
Poor (<8)	35 (23.0)	61 (40.1)	56 (36.8)	152 (31.3)
Moderate (8–11)	65 (21.6)	144 (47.8)	92 (30.6)	301 (62.1)
Adequate (≥12)	5 (15.6)	19 (59.4)	8 (25.0)	32 (6.6)
		$X^2 = 5.04$	p = 0.283	
Total	105 (21.6)	224 (46.2)	156 (32.2)	485 (100.0)

[a] Percentages are sum of rows, [b] percentages are sum of columns.

Table 3
Blood pressure levels of the subjects with hypertension according to the factors affecting blood pressure levels.

Characteristics	Current blood pressure level			
	Normal	PreHT	HT	Total
HT medication	n (%)[a]	n (%)[a]	n (%)[a]	n (%) [b]
Using regular	94 (22.9)	188 (45.9)	128 (31.2)	410 (84.5)
Using irregular	11 (14.7)	36 (48.0)	28 (37.3)	75 (15.5)
HT training	$X^2 = 2.80$ P = 0.247			
Not received	73 (23.4)	140 (44.9)	99 (31.7)	312 (64.3)
Received	32 (18.5)	84 (48.6)	57 (32.9)	173 (35.7)
Alternative or complementary medicine	$X^2 = 1.61$ P = 0.447			
Not admitted	65 (22.4)	126 (43.4)	99 (34.1)	290 (59.8)
Admitted	40 (20.5)	98 (50.3)	57 (29.2)	195 (40.2)
Exercise level	$X^2 = 2.24$ P = 0.327			
Not exercising	52 (20.6)	116 (46.0)	84 (33.3)	252 (52.0)
Inadequate	17 (21.0)	40 (49.4)	24 (29.6)	81 (16.7)
Adequate	36 (23.7)	68 (44.7)	48 (31.6)	152 (31.3)
Fruit and vegetable consumption	$X^2 = 0.96$ P = 0.916			
Not eat every day	23 (20.4)	51 (45.1)	39 (34.5)	113 (23.3)
One meal per day	19 (18.1)	55 (52.4)	31 (29.5)	105 (21.6)
Two meals per day	17 (18.9)	35 (38.9)	38 (42.2)	90 (18.6)
≥3 meals per day	46 (26.0)	83 (46.9)	48 (27.1)	177 (36.5)
Salt consumption habits	$X^2 = 9.17$ P = 0.164			
Normal/more salty	47 (24.2)	84 (43.3)	63 (32.5)	194 (40.0)
Less salty	33 (19.8)	81 (48.5)	53 (31.7)	167 (34.4)
Salt less	25 (20.2)	59 (47.6)	40 (32.3)	124 (25.6)
How to continue BP	$X_2 = 1.61$ p = 0.807			
Normal	84 (25.8)	163 (50.0)	79 (24.2)	326 (67.2)
High	11 (8.7)	43 (34.1)	72 (57.1)	126 (26.0)
Unstable	10 (30.3)	18 (54.5)	5 (15.2)	33 (6.8)
HT duration	$X_2 = 52.69$ P < 0.001			
<5 years	33 (21.0)	69 (43.9)	55 (35.0)	157 (32.4)
5–9 years	38 (31.7)	47 (39.2)	35 (29.2)	120 (24.7)
10–14 years	20 (20.4)	46 (46.9)	32 (32.7)	98 (20.2)
≥15 years	14 (12.7)	62 (56.4)	34 (30.9)	110 (22.7)
	$X_2 = 14.43$ p = 0.025			
Total	105 (21.6)	224 (46.2)	156 (32.2)	485 (100.0)

[a] Percentages are sum of rows, [b] percentages are sum of columns.

The subjects were questioned about their BP levels according to their experience, 67.2% of the subjects answered that they had a normal level of BPs while 26% of them had HBP levels. However, the BP measurement results of the subjects who claimed normal BP during their lifetime revealed that only 25.8% of them had normal BP in reality while 24.2% of them had hypertensive BP measurement. Oppositely, BP measurement results of the subjects who claimed HBP during their lifetime revealed that only 57.1% of them had hypertensive BP in reality.

Substantial number of the subjects (84.5%) reported regular intake of antihypertensive medication, 10.3% of the subjects claimed drug intake in case of high BP measurement, 5.2% of the subjects did not take their prescribed medication at all. Measured BP levels of subjects with regular intake of drugs were normal in 22.9% of subjects and hypertensive in 31.2% of the subjects. These rates were 14.7% and 37.3% respectively among subjects who did not take their medication or take irregularly (Table 3).

In the study, 40.2% of the subjects reported the usage of alternative methods to control BP, 4.5% of the subjects reported regular jogging, and 2.7% of the subjects reported diet modification. 52.6% of the subjects claimed that they use only the prescribed drugs to control BP. 34.1% of only drug users had hypertensive BP measurements while 29.2% of alternative method users had hypertensive BP measurements (Table 3). This ratio was 22.7% among the subjects performing regular jogging. 35.7% of the study population noted that they got an education about HT (Table 3). 28% of all subjects (78.4% of educated subjects) got this education from medical doctors, 4.3% of nurses and 3.3% of both medical doctors and nurses. 18.6% of educated subjects reported that they got this education once, 5.6% of them twice, 6.8% of them three times, and 4.7% of them four or more times during their lifetime. There was not any significant relation between education about HT and degree of BP levels measured at the time of interview (Table 3).

Since age vs. BMI ($r = -0.146$, $p < 0.001$), age vs disease duration ($r = 0.301$, $p < 0.001$) and age vs. educational status (Spearman's rho $= -0.304$, $p < 0.001$) had significant correlation, these parameters were included in the analyses. Among the factors affecting BP level statistically with the p-value < 0.10, significant independent variables as BMI, HT duration and use of alternative method and not significant independent variables as age, educational status and knowledge level about HT were all included in multivariate BLR analyses. HT knowledge score, gender, age, BMI, disease duration, education level, and perception of income level as independent variables were included in the BLR analyses. All independent variables did not show statistical significance, thus relevant table showing the results of this analyses were not expressed further in another table.

Discussion

In this study, the relation between knowledge about HT, sociodemographic characteristics and obtaining controlled BP levels were evaluated among patients diagnosed with HT with the help of

multivariate analyses. As known, increased knowledge about the disease promotes the compliance of the patient with prescribed medications. However, there have been few studies searching the relation between awareness, knowledge and real-time BP measurements in Turkish population. Thus, we designed this study to evaluate the socio-demographic factors affecting outcomes of hypertensive patients.

We found that approximately one-third of patients with HT had a poor level of knowledge about HT and very few numbers of patients (6.6%) had adequate level knowledge (Table 1). In USA, the frequency of HT patient with the poor level knowledge about HT was 22%.[23] In same study, 86% of the subjects defined BP categories properly, that value was 72.4% in our study. In Pakistan, only 0.8% of hypertensive patients claimed that they had adequate knowledge about HT.[11] All these data indicates that patients in developed countries have more knowledge about HT compared to people in underdeveloped or developing countries. It means that there may be a correlation between industrialization level and awareness for HT.

The ratio of subjects with BP under control was 75% among subjects with adequate level of HT knowledge and this was higher compared to the ones with moderate (69.4%) and poor levels (63.2%). But it was not statistically significant (Table 2). The study from Pakistan also revealed similar results. Average knowledge score of subjects with BP under control was found to be higher compared to that of subjects with uncontrolled BP (21.8 vs. 18.7 with $p > 0.05$) as found in our study.[12]

Educational status and level of knowledge about HT were shown to promote positively the control of HBP, but the additional effects of these factors were found to be minimal.[24] Similarly, Xu LJ, Meng Q[25] found that educating patients about HT enabled reduction in SBP of 19.03 mmHg and DBP of 10.33 mmHg. However, in our study, these variables were not significant (Table 1).

Another important issue in management of HT is to know asymptomatic nature of having HBP. In our study, most of the subjects (63.3–92.6%) did not know that HBP can course without any sign and symptoms. It was 67.3% for Turkish population living around İzmir while it was 20.6% in USA.[14,23] The patients assumed that they experienced headache (92.6%) and dizziness (82.9%) in case of HBP. Due to these false perceptions, the patients think their BP levels are under control in the absence of these complaints whereas HBP usually does not give any sign and symptoms. This false understanding adversely affects the HT control. The knowledge about HT is very important for patients to evaluate themselves-their BP measurements at home and to comply with their treatment and they can easily be aware of their general status and take precaution promptly.

In our study, we found that level of knowledge about HT increased proportionally with higher degree of educational states. However, the effect of age on the HT knowledge was not statistically significant. Thus, educational status contributed only 4.5% ($R^2 = 0.045$) of changes in HT knowledge score. In a study from USA, it was also shown that medical knowledge level increased proportionally with increment in degree of education and decrement in age and educational contributed 17.7% of changes in medical knowledge while age variable affected less (6.1%).[13] This conflicting result can be due to the different effects of educational status on the medical knowledge of the countries with different level of industrialization. Another reason for this kind of result is the difficulty in standardization of education and medical knowledge.

Most of the patients (84.5%) reported regular intake of prescribed drugs (Table 3). In other studies, the ratio of regular drug intake was ranging between from 78.8% to 98.1%.[14,26] In CDRFS, it was 85%.[10] Thus, our finding was inconsistent with the literature. Among subjects taking their antihypertensive medicine promptly, two third (68.8%) had BP level under control. This ratio differs in different studies ranging from 53.9% to 72%.[7,10,27] In a hospital-based study, 78% of regular drug users had BP under control.[28] All these different results imply that there are different rates of controlling BP in spite of regular intake of prescribed medications. The reason for such result may be due to different sampling universe and varied frequency of planned admissions for the diseasesince regularity of outpatient admissions can change the course of treatment and use of prescribed medications.

Age, educational status, HT knowledge score, BMI, HT duration and use of alternative or complementary medicine methods were not found to be significant in controlling BP among hypertensive patients after performing multivariate FLR analyses. 40.2% of the subjects reported the use of alternative medicine method to control BP. HT control rate was 70.8% among alternative method users and 65.9% among subjects who do not use any alternative method, but the difference was not statistically significant. Still this difference can be explained in a way that patients using alternative medicine methods were more motivated to control their BP compared to the others. In the study from İzmir, salt restriction, diet, weight loss, quitting smoking, exercise and alcohol restriction were found to promote positively the treatment of HT.[14] Our study results revealed that the subjects' knowledge to keep BP under control was moderate, but they did not apply promptly what they knew.

Approximately one-third of the study population noted that they got education about HT (Table 3). Most of the educated subjects (78.4%) got this education from medical doctors, 4.3% of them from nurses and 3.3% of them from both medical doctors and nurses. Oskay, Onsuz[14] also described the source of knowledge as mostly doctors, then social environment, television and newspapers in order. Nurses can be a source of knowledge about HT and are expected to the important role in educating subjects in future.

There are some limitations to our study. Firstly, the study population consisted of residents in Yozgat province, thus the results may not be extrapolated to general population of Turkey. Secondly, the study enrolled only subjects from primary health centers, thus the data in hand can't reflect hypertensive subjects applied to secondary or tertiary health centers. Thirdly, this is a cross-sectional study based on claims of subjects, thus the answers of subjects may be biased.

Conclusions

Increase in number of deaths due to cardiovascular diseases in recent years diverted researchers' attention to prevention and controlling of HBP which is a leading cause of cardiovascular diseases. According to the results of our study, (1) there was no relationship between HT knowledge level and controlling high blood pressure, (2) majority of subjects did not have sufficient knowledge about HT, and (3) two third of the subjects did not perform important lifestyle modifications to control BP.

Practice implications

Nurses should follow patients with HT, should replace lack of knowledge, promote behavioral changes, and should be motivated to prepare educational programs for the patients.

Conflicts of interest

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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Ethical and official approvals from the local authorities were obtained for the study accordingly and the investigation was performed in accordance with the principles outlined in the Declaration of Helsinki.

References

1. WHO. A Global Brief on Hypertension: Silent Killer, Global Public Health Crisis. Genava: World Health Organization; 2013.
2. A. Alwan. Global Status Report on Noncommunicable Diseases 2010. World Health Organization; 2011.
3. Huang Y, Su L, Cai X, et al. Association of all-cause and cardiovascular mortality with prehypertension: a meta-analysis. *Am Heart J* 2014;**167**:160–168 [e1].
4. Turkey Health Ministry. Turkey Burden of Disease Study (TBDS) 2004. Ankara: Aydoğdu Ofset Matbaacılık San. ve Tic. Ltd. Şti.; 2007.
5. TSI. Causes of Death Statistics. Ankara: Turkish Statistical Institute (TSI); 2013;2014.
6. Pereira M, Lunet N, Azevedo A, Barros H. Differences in prevalence, awareness, treatment and control of hypertension between developing and developed countries. *J Hypertens* 2009;**27**:963–975.
7. TSHKD. Turkish hypertension prevalence study 2012 (PatenT2). Turkish Society of Hypertension and Kidney Diseases (TSHKD); 2012.
8. Erdem Y, Arici M, Altun B, et al. The relationship between hypertension and salt intake in Turkish population: SALTURK study. *Blood Press* 2010;**19**:313–318.
9. Satman I, TURDEP-II SG. Turkey Diabetes Prevalence Studies: TURDEP-I and TURDEP-II. 47 National Congress of Diabetes. Antalya, Turkey: Rixos Sungate Hotel; 2011.
10. Ünal B, Ergör G, Dinç-Horasan G, Kalaça S, Sözmen K. Chronic Diseases and Risk Factors Survey in Turkey. Ankara: Anıl Matbaa Ltd. Şti; 2013.
11. Saleem F, Hassali M, Shafie A, Awad A, Bashir S. Association between knowledge and drug adherence in patients with hypertension in Quetta, Pakistan. *Trop J Pharm Res* 2011;**10**.
12. Almas A, Godil SS, Lalani S, Samani ZA, Khan AH. Good knowledge about hypertension is linked to better control of hypertension; a multicentre cross sectional study in Karachi, Pakistan. *BMC Res. Notes* 2012;**5**:579.
13. Levinthal BR, Morrow DG, Tu W, Wu J, Murray MD. Cognition and health literacy in patients with hypertension. *J Gen Intern Med* 2008;**23**:1172–1176.
14. Oskay EM, Onsuz M, Topuzoglu A. Assesment of hypertension knowledge, attitude and throught of policyclinic patients at a primary health care center in Izmir. *J Adnan Menderes Univ Med Fac* 2010;**11**:3–9.
15. Hong W-HS. Evidence-based nursing practice for health promotion in adults with hypertension: a literature review. *Asian Nurs Res* 2010;**4**:227–245.
16. TSI. Address Based Population Registration System (ABPRS) Results. Population of localities: Turkish Statistical Institute; 2013.
17. Gazmararian JA, Williams MV, Peel J, Baker DW. Health literacy and knowledge of chronic disease. *Patient Educ Couns* 2003;**51**:267–275.
18. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. *J Am Med Assoc* 2003;**289**:2560–2571.
19. James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *J Am Med Assoc* 2014;**311**:507–520.
20. Rodriguez CJ, Swett K, Agarwal SK, et al. Systolic blood pressure levels among adults with hypertension and incident cardiovascular events: the atherosclerosis risk in communities study. *JAMA Intern Med* 2014;**174**:1252–1261.
21. Amine E, Baba N, Belhadj M, et al. Diet, Nutrition and the Prevention of Chronic Diseases: Report of a Joint WHO/FAO Expert Consultation. World Health Organization; 2002.
22. Meyers LS, Gamst G, Guarino A. Applied Multivariate Research: Design and Interpretation. Sage; 2006.
23. Viera AJ, Cohen LW, Mitchell CM, Sloane PD. High blood pressure knowledge among primary care patients with known hypertension: a North Carolina Family Medicine Research Network (NC-FM-RN) study. *J Am Board Fam Med* 2008;**21**:300–308.
24. Pandit AU, Tang JW, Bailey SC, et al. Education, literacy, and health: mediating effects on hypertension knowledge and control. *Patient Educ Couns* 2009;**75**:381–385.
25. Xu LJ, Meng Q, He SW, et al. The effects of health education on patients with hypertension in China: a meta-analysis. *Health Educ J* 2014;**73**:137–149.
26. Öztürk A, Aykut M, Günay O, et al. Prevalence and factors affecting of hypertension in adults aged 30 years and over in Kayseri province. *Erciyes Tıp Derg* 2011;**33**:219–228.
27. Kılıç M. Hypertension prevalence, awareness, and control, and affecting factors among the primary care clients in Yozgat city center. *TAF Prev Med Bull* 2013;**12**:79–86.
28. Göçgeldi E, Babayiğit MA, Hassoy H, Açıkel CH, Taşçı İ. Evaluation of the quality of life perceived by hypertensive patients and of the factors affecting the quality of life. *Gülhane Tıp Derg* 2008;**50**:172–179.

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Case report

Transcatheter aortic valve implantation with either Edwards SAPIEN valve and core valve in patients with horizontal aorta

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Introduction

TAVI has been becoming a mainstay of treatment of severe aortic stenosis in patients who have a high estimated risk of surgical valve replacement.[1,2] Unfavorable anatomical structures can induce technical difficulties like high or low valve deployment which can also occur following implantation appear to be more common with the CV. The low or high positioning may be associated with paraprosthetic regurgitation, device embolization and impact atrioventricular node, resulting hemodynamic instability.[3] Herein, we aimed to delineate pre-procedural root angulation prediction and performing maneuvers for optimal device deployment and abstain from complications.

Case report 1

An 80 year old man admitted with increasing dyspnea (New York Heart Association functional Class-III) and palpitation for last 1 month. On physical examination, the patient had signs of pulmonary congestion and cardiovascular auscultation revealed aortic stenosis murmur. Baseline transthoracic echocardiography revealed a calcified aortic valve, mean velocity of 3.9 m/sn, mean transvalvular gradient of 42 mm Hg. Left ventricular ejection fraction was 40%. His coronary angiography revealed calcified plaque in the epicardial coronary arteries. His calculated Logistic Euro Score was 14.2% and Society of Thoracic Surgeons (STS) score was 5%. Due to the patient had low risk, the heart team decided to perform surgical aortic valve replacement (SAVR) but the patient refused SAVR and decided to proceed with TAVI. So, the heart team approached for off-label TAVI. Multidetector computed tomography (MDCT) documented bilateral ilio-femoral arteries which had minimum diameter of 10.1 mm in right side, aortic annulus was measured as 25 mm in diameter and aortoventricular anatomy within horizontal lying (Fig. 1A, B). In cardiac catheterization especially left anterior oblique 10° cranial, serial aortic root injections also revealed the aortoventricular angle. Transfemoral route didn't fit for purpose because of large degree of aortic root angulation. Because of specific anatomic consideration, transfemoral TAVI performed as off-label indication. TAVI was performed under local anesthesia the patient evaluated one month later. A Novaflex+ (Edwards Lifesciences, Inc.) deployment catheter was inserted through the right common femoral artery. Pre-implantation balloon valvuloplasty was done with a 25 * 40 mm balloon with good expansion. The 29 mm ESV alignment and deployment was done under fluoroscopic guidance with "two step deployment technique" including pull back Amplatz Extra-Stiff J-tip guidewire (COOK, Bjaeverskov, Denmark) by about a centimeter and then, floppy tipped guidewire was rotated clockwise providing coaxial alignment. After the deployment, fluoroscopic control showed normal bioprosthetic valve function and there is no leakage (Fig. 2).

Case report 2

A 71 year old woman admitted to arising from gradual onset of exertional dyspnea for last 1 year. The patient had a past medical history of coronary artery bypass graft. On cardiovascular auscultation, there was a grade 3/6 systolic murmur in aortic focus. Baseline transthoracic echocardiography revealed several calcified changes of aortic valve with mean transvalvular gradient of 38 mm Hg and mean velocity of 4.1 m/sn. Left ventricular ejection fraction was 60%. Cardiac catheterization revealed severe coronary artery disease with 70% narrowed diffuse lesion of left anterior descending artery and with 100% occluded left internal mammary artery. The Logistic Euro Score and STS were calculated to be 32% and 9%. Because of high surgical risk, the heart team approached for TAVI. MDCT evaluation prior to TAVI revealed minimal bilateral ilio-femoral luminal diameter of 8.2 mm, aortic annulus was measured as 26 mm and changes in anatomical structures of aortic root which had broad aortoventricular angle (Fig. 3A, B). In cardiac catheterization, left anterior oblique 10° cranial position revealed large degree of aortic root. Transfemoral TAVI was performed under general anesthesia as off-label indication due to very calcified bilateral subclavian arteries. A right femoral cutdown was performed and then, the 18 Fr CV delivery catheter was indwelled into the right common femoral artery. Balloon aortic valvuloplasty was performed under rapid

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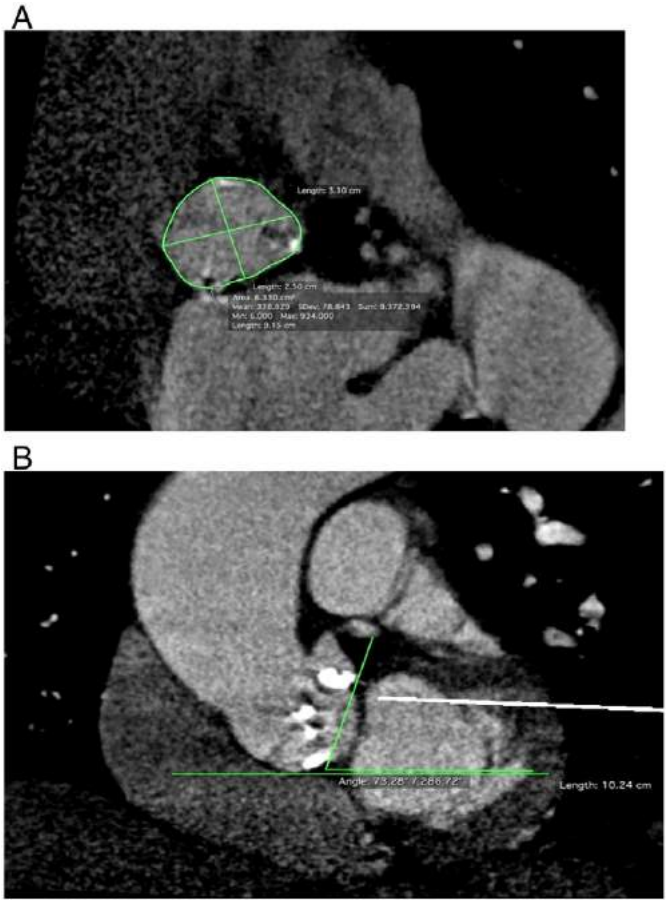

Fig. 1. A. The maximum and minimum aortic annulus diameter and annulus area. B. Calcified aortic valve and aortoventricular angle was measured as 73.28° in coronal plane of multidetector computed tomography.

ventricular pacing using 23 mm balloon. The 31 mm CoreValve Evolut™ positioning was based on fluoroscopy guidance with "two step deployment technique" including pull back Amplatz Super-Stiff J-tip guidewire by about a centimeter and then, floppy tipped guidewire was rotated clockwise providing coaxial alignment. Then, CV was deployed under supra-annular aortography during right ventricular pacing. Post-procedural aortography showed suitable position of the CV with no valvular and paravalvular leakage (Fig. 4).

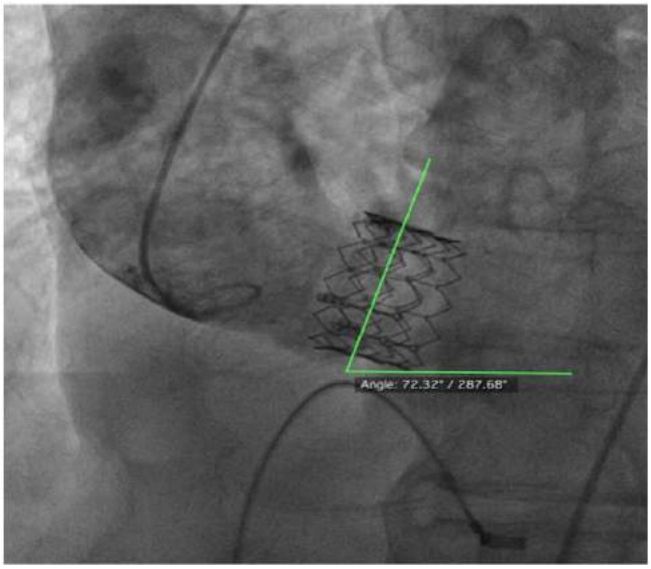

Fig. 2. Left anterior oblique image of the Edwards Sapien Valve had normal function.

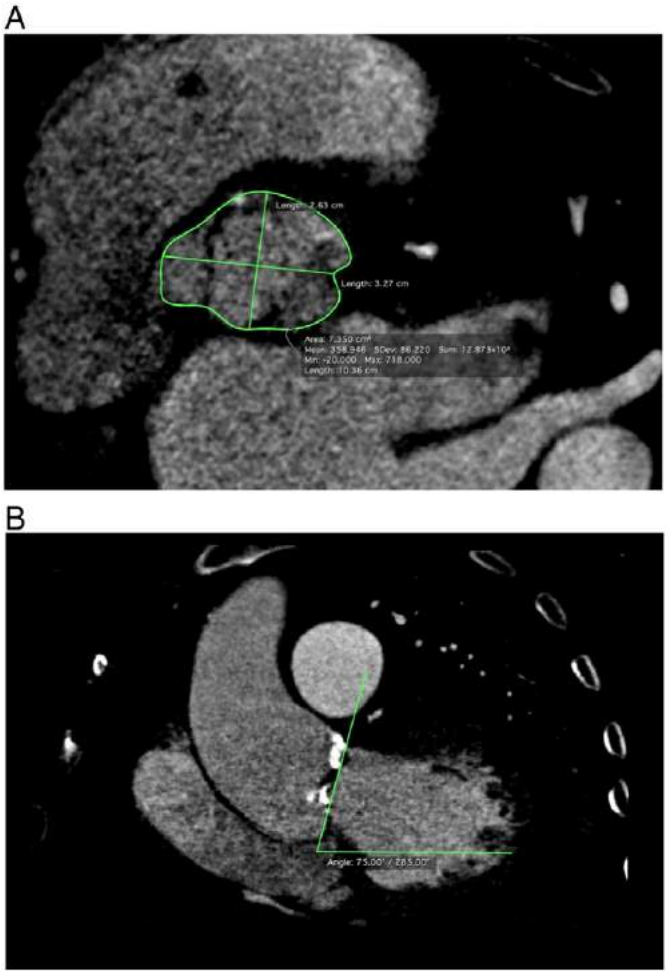

Fig. 3. A. The maximum and minimum aortic annulus diameter and annulus area. B. Calcified aortic valve and aortoventricular angle was measured as 75° in coronal plane of multidetector computed tomography.

Discussion

Horizontal aortic root with a vertical annulus is ascribed technical challenge in TAVI. Preprocedural MDCT offers detailed aortic root anatomy. Understanding of root anatomy plays key role for certain alignment and accurate positioning of the bioprosthesis valve.[4] Coronal oblique projection in the MDCT provides aortoventricular angulation defined as the angle between left ventricle and ascending aorta 40 mm above the aortic annulus.[5] If angulation is >70°, transfemoral and left subclavian approaches are contraindicated. Especially transfemoral approach within excessive angulation limits the capability to control the tension on delivery catheter during device release may lead to implant failure. Alternative routes such as transaortic, transaxillar accesses are utilized to ensure more effortless device manipulation in these cases.[3,4] Edwards NovaFlex's steerable catheters can facilitate controlled position and following route in challenging anatomies including horizontal aorta.[6] Furthermore, U.S. Food and Drug Administration approves for the CV Evolut R transcatheter valve and the EnveoR delivery system in June 2015. EnveoR's capsule flare and flexible catheter offer navigation of unfavorable anatomical structures. This catheter provides the option of repositionability.[7] Finally, Chan et al. performed several different maneuvers in a patient with horizontal aorta implanted CV

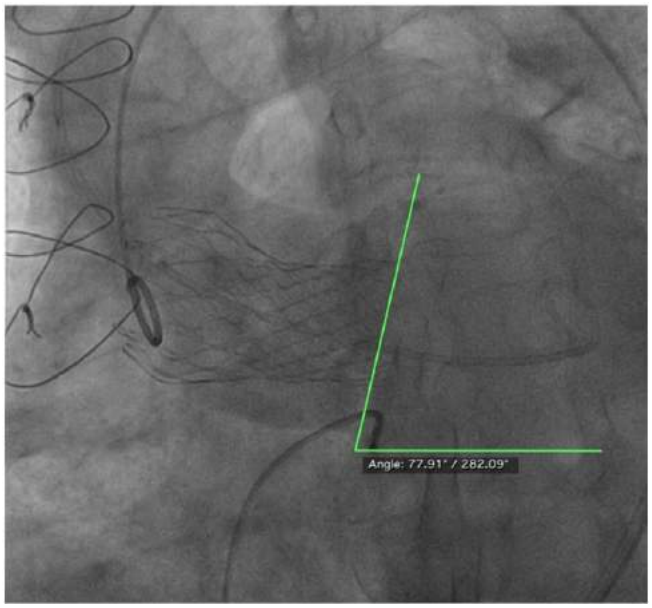

Fig. 4. Left anterior oblique image of the Core Valve had normal function.

successfully including taking back the Super-Stiff guidewire. Firstly, introducing of back-end of the guidewire, retracting the delivery system without guidewire and the last maneuver is re-introducing soft tip of the guidewire and pushing the delivery system towards the ventricular side gradually.[4]

In conclusion, preprocedural imaging techniques especially MDCT plays as a central role in a planning prior to procedural approaching and deciding to apply suitable technical management. In this manner, the number of the patients with horizontal aorta implanted bioprosthesis valve may be increased, transfemoral TAVI may apply in this condition more commonly.

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this manuscript.

References

1. Leon MB, Smith CR, Meck M, et al. Partner trial investigators: transcatheter aortic valve implantation for aortic stenosis in patients who cannot undergo surgery. *N Engl J Med* 2010;**363**:1597–1607.
2. Cabau JR, Webb JG, Cheung A, et al. transcatheter aortic valve implantation for the treatment of severe symptomatic aortic stenosis in patients at very high or prohibitive surgical risk: acute and late outcomes of the multicenter Canadian experience. *J Am Coll Cardiol* 2010;**55**(11):1080–1090.
3. Sarkar K, Ussia GP, Tamburino C. Transcatheter aortic valve implantation with core valve revalving system in uncoiled (horizontal) aorta. Overcoming anatomical and technical challenges for successful deployment. *Catheter Cardiovasc Interv* 2011;**78**(6):964–969.
4. Chan PH, Alegria-Barrero E, Mario C. Difficulties with horizontal aortic root in transcatheter aortic valve implantation. *Catheter Cardiovasc Interv* 2013;**81**:630–635.
5. Schultz C, Moelker A, Tzikas A, et al. The use of MSCT for the evaluation of the aortic root before transcutaneous aortic valve implantation: the Rotterdam approach. *EuroIntervention* 2010;**6**(4):505–511.
6. Edwards Lifesciences Corporation.
7. Medtronic,CNBC;June 23rd 2015.

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Case report

Cleft in aortic valve

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ABSTRACT

Congenital aortic valve cleft is a rare entity with scanty literature available. Cleft in aortic valve can be congenital, degenerative or age related. Sudden rupture of cusps can lead to severe aortic insufficiency requiring surgical intervention. With this we acknowledge a case where patient had aortic insufficiency due to cleft in right coronary cusp with perimembranous ventricular septal defect.

Case detail

A 9 year old boy, who has been diagnosed recently as suffering from congenital ventricular septal defect and moderate aortic valvular insufficiency, has been admitted with complains of dyspnea NYHA class II and failure to thrive with no history suggestive of rheumatic heart disease or infective endocarditis. Two dimensional echocardiography was suggestive of moderate size perimembranous ventricular septal defect with left to right shunt, moderate aortic insufficiency. At surgery, the findings were confirmed; there was tricuspid aortic valve with cleft in right coronary cusp with no cusp prolapse, without any additional commissures (Fig. 1). The ventricular septal defect was closed with GORE-TEX® patch, continuous PROLENE® suturing while cleft in right coronary cusp was repaired with interrupted direct PROLENE® sutures (Fig. 2). Post-operative transesophageal echocardiography was suggestive of no flow across interventricular septum with trivial aortic insufficiency.

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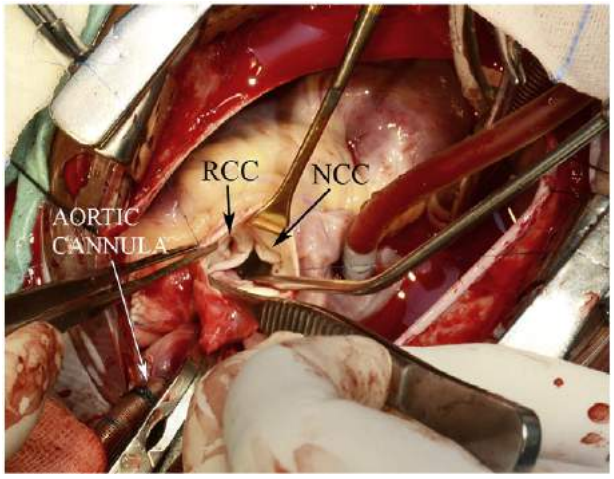

Fig. 1. Perioperative photograph demonstrating cleft in aortic valve right coronary cusp.

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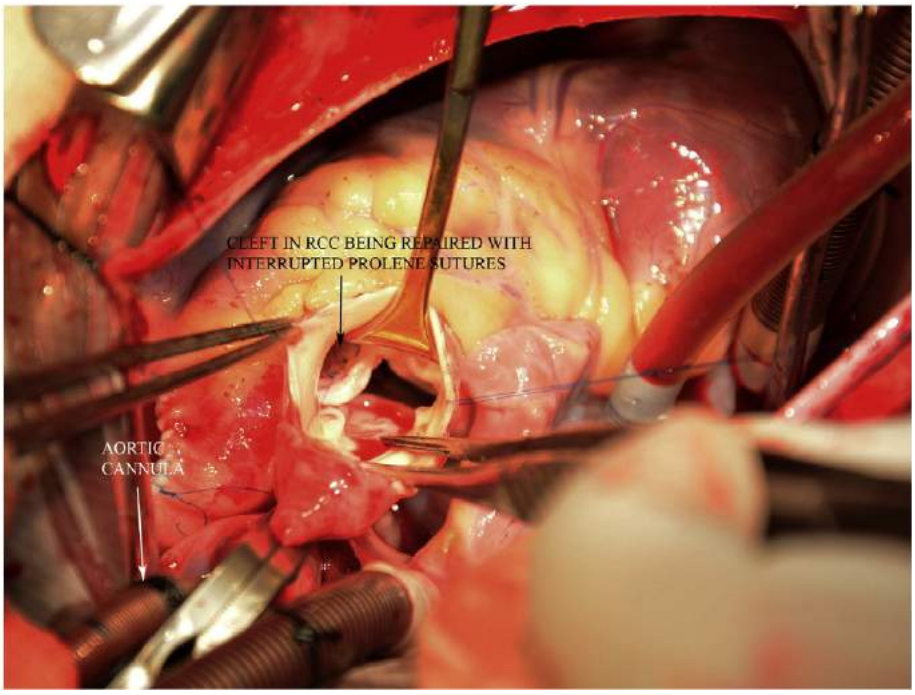

Fig. 2. Perioperative photograph demonstrating cleft being repaired with direct PROLENE® sutures.

Conflict of interest

None declared.

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Case report

Percutaneous extraction of implantable cardioverter defibrillator electrode with mechanical dilator sheath

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ABSTRACT

For removing the electrodes, manual simple traction may be useful and various methods have been used in extraction for chronically implanted electrodes. We report the case of successful percutaneous extraction of implantable cardioverter defibrillator electrode with Evolution mechanical dilator sheath.

A 66-year-old man with ischemic cardiomyopathy who received a dual-chamber implantable cardioverter defibrillator (ICD) 7 years ago was referred to our hospital for the skin erosion causing protrusion of the lead portion secondary to infection. After detailed questioning, it was discovered that he had ICD pocket infection 3 years ago. ICD generator was removed and a new single-chamber ICD was implanted to contralateral pectoral region. Atrial and ventricular electrodes could not be removed due to fibrous adhesions at the contact points between electrodes and venous or cardiac walls. Proximal tips of these electrodes were fixed to the pectoral region. 2 months ago proximal tips of these electrodes were protruding after the skin erosion (Fig. 1).

Transvenous lead extraction procedure was performed in the cathlab under moderate sedation. After the skin dissection electrodes were separated from the scar tissue by blunt dissection. Atrial electrode was removed with manual traction. A locking stylet (Liberator Universal Locking Stylet, Cook Medical) and mechanical dilator sheath were used for ventricular lead. The Evolution mechanical dilator was positioned over the electrode. The dilator sheath was moved along the electrode body by cutting fibrous adhesion via the distal metal tip. Once the fibrous attachment is cut the outer sheath is advanced until another area of attachment is encountered. After the release of the electrode from fibrous tissue the electrode was pulled back into the sheath and removed (Figs. 2 and 3).

Electrodes of pacemaker or ICD usually undergo fibrotic encapsulation by activation of humoral and cellular immune response mechanisms after transvenous implantation.[1] For removing these electrodes, manual simple traction may be useful, however chronically implanted electrodes develop fibrous attachments to surrounding structures and require more advanced extraction systems.[2,3] Various methods have been used in electrode extraction including manual traction, extended weight or forcep-assisted traction, radiofrequency or laser devices, mechanical extraction systems, and open-chest surgery.[3,4] The mechanical method with a hand-powered sheath marketed as the Evolution can

Fig. 1. Protrusion of the electrodes after skin erosion.

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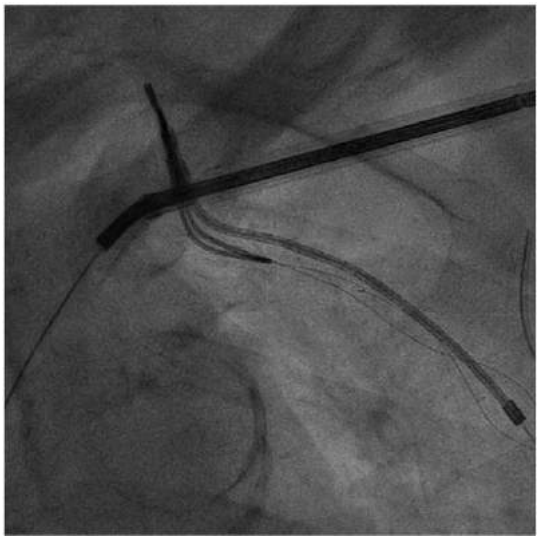

Fig. 2. Fluoroscopic view of the electrode covered by the Evolution sheath.

Fig. 3. Successfully extracted electrode.

be used as a useful method for chronically implanted ICD electrodes in high risk patients.

Conflict of interest

None.

References

1. Esposito M, Kennergren C, Holmstrom N, Nilsson S, Eckerdal J, Thomsen P. Morphologic and immunohistochemical observations of tissues surrounding retrieved transvenous pacemaker leads. *J Biomed Mater Res* 2002;**63**:548–558.
2. Smith MC, Love CJ. Extraction of transvenous pacing and ICD leads. *Pacing Clin Electrophysiol* 2008;**31**:736–752.
3. Güngör H, Duygu H, Yildiz BS, Gül I, Zoghi M, Akin M. Excimer laser assisted implantable cardioverter defibrillator lead extraction: an alternative treatment to the surgery? *Anadolu Kardiyol Derg* 2009;**9**:340–341.
4. Oto A, Aytemir K, Yorgun H, et al. Percutaneous extraction of cardiac pacemaker and implantable cardioverter defibrillator leads with evolution mechanical dilator sheath: a single-centre experience. *Europace* 2011;**13**:543–547.

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Case report

Unexpected guest: Atrial fibrillation due to electrical shock

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ABSTRACT

Cardiac arrhythmias due to electrical injuries are rare among emergency service admittances. A 35 year-old female patient was admitted to emergency service with palpitation after electrical injury as a result of contact with a domestic low-voltage source. Electrocardiography (ECG) showed atrial fibrillation with rapid ventricular response. Transthoracic echocardiography findings were normal. Atrial fibrillation spontaneously converted to normal sinus rhythm after rate limiting treatment with beta-blocker. The patient was discharged without any complication on the third day of hospitalization. Although cardiac arrhythmias rarely occur after electrical injury, cardiac monitoring is recommended for all patients with documented rhythm disorder, loss of consciousness, or abnormal ECG at admission.

Introduction

In parallel with the technological tools which acquire more space in our lives, the electrical injury cases have increased. The actual incidence of electrical injuries is unknown.[1] Soft tissue burns, cardiac arrhythmias, neurological damage and even death may occur in patients who are exposed to electrical injury.[2,3] Cardiac monitoring and medical therapy are recommended for patients with documented rhythm disorder, loss of consciousness, or abnormal ECG at admission.[4] Herein, we present a rare case of electrical injury as a result of contact with a low-voltage domestic source presented with atrial fibrillation.

Case report

A 35-year-old female patient without known cardiac disease was admitted to emergency service with complaint of palpitation after electrical injury that occurred as a result of contact with a low-voltage domestic source. On physical examination, radial pulses were irregular and rapid, blood pressure was 125/80 mm Hg. There were skin-burn injuries on the extensor side of the middle phalanx of the left hand indicating the entrance of electric current and on the extensor side of the right arm showing the exit. The neurologic, respiratory and gastrointestinal system examinations were normal. The cardiac rhythm was monitored and atrial fibrillation with rapid ventricular response (160 beats/min) was seen on an electrocardiogram (Fig. 1). In laboratory analysis; white blood cell count (WBC): 14.200 (4.600–10.200 K/μL), hemoglobin: 11.9 (12.2–18.1 g/dL), creatine-kinase (CK): 386 (40–165 U/L), CK-MB: 12.3 (0.6–6.3 ng/mL) and lactate dehydrogenase (LDH): 309 (125–243 U/L) were detected. Troponin I and other biochemical and hematological parameters were in normal range. Transthoracic echocardiography findings were normal. Intravenously, 5 mg of metoprolol was administered for ventricular rate control. Enoxaparin 8000 IU/0.8 mL was given subcutaneously (sc) at admission for preventing thromboembolic complications. The patient was transferred to the coronary intensive care unit for hemodynamic and rhythm monitorization. In adding to burn dressings, daily sc 2 × 8000 IU/0.8 mL of enoxaparin, and oral 2 × 50 mg of metoprolol were started. On the first day of hospitalization, the rhythm was spontaneously returned to sinus rhythm (Fig. 2). The abnormal laboratory parameters were improved at follow-up and the patient was discharged on the third day of admission without any complication.

Discussion

The exact mechanism of electric shock-induced arrhythmias is not clear. The heart is one of the most susceptible organs to electrical injury. Electrical shock may cause direct myocardial necrosis or cardiac arrhythmias. Asystole and ventricular fibrillation are the most serious arrhythmic complications of electrical injury.[2] Increased cardiac sodium/potassium pump activities and an increase of serum potassium concentration may trigger arrhythmias after electrical injury.[5] The most

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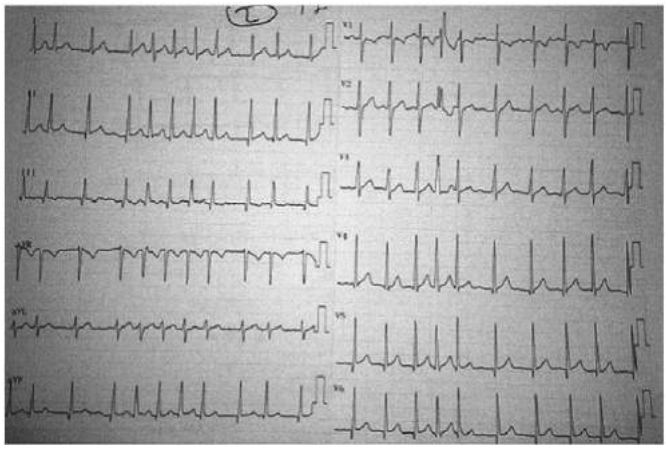

Fig. 1. Initial electrocardiogram showed atrial fibrillation with rapid ventricular response.

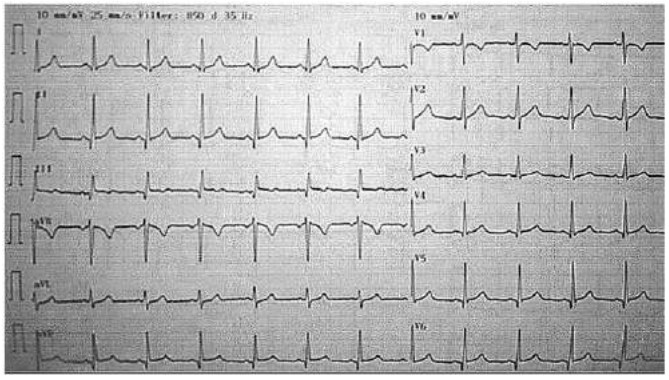

Fig. 2. The rhythm was returned to sinus after medical therapy.

common electrocardiographic changes resulting from electric shock are sinus tachycardia and nonspecific ST-segment deviations and T-wave changes. Atrial fibrillation, atrial and ventricular premature contractions, bundle branch blocks, QT interval prolongation and ventricular fibrillation are less frequently seen. In addition, other cardiac effects such as transient hypertension, myocardial infarction, left ventricular dysfunction and cardiac rupture can be seen.[6,7] In a study by Arrowsmith et al., the frequency of cardiac complications was reported as 3% among 145 persons exposed to electric shock. Atrial fibrillation was detected in only one person who returned to normal sinus rhythm after intravenous digoxin administration.[8] In another study conducted with 182 patients with electrical shock exposure, Butler et al. reported sinus tachycardia in 7 patients, ventricular premature contractions in 5 patients and atrial fibrillation in 2 patients. They reported that cardioversion was required in one of two patients with atrial fibrillation.[9] Uzkeser et al.[10] reported a case of atrial fibrillation after electrical shock exposure and the patient's rhythm had returned to sinus rhythm by medical therapy on the second day. Gözlükaya et al.[11] reported a case with atrial fibrillation developed after electrical shock who returned to sinus rhythm 20 min after intravenous diltiazem administration. Our case was similar with other cases in the literature in terms of diagnosis, treatment and disease course. The patient was treated with intravenous metoprolol at admission and rapid ventricular rate was diminished, then oral metoprolol was continued in the intensive care unit. On the first day, the rhythm was returned to normal sinus rhythm spontaneously. Arrowsmith et al.[8] reported that all patients with dysrhythmias resolved within 48 h of admission either spontaneously or with pharmacologic treatment.

Although the cardiac effects due to electrical shock are rare, it is important to close follow-up for cardiac arrhythmias. Cardiac arrhythmias due to electrical injuries are usually observed during or immediately after the event.[12] Cardiac monitoring is recommended if there is loss of consciousness or documented rhythm disturbance and in cases with abnormal ECG at the time of admission.[4] The monitoring period in studies are usually 24 h.[4,8] Unless there is a loss of consciousness and the 12-lead ECG is normal, it is unlikely that the patient will go on to develop cardiac problems, and it is not necessary to monitor the patient for 24 h.[4,8]

In conclusion, electrical shock occurs in an unexpected time and atrial fibrillation is an uncomfortable cardiac problem that may occur after electrical shock. With a correct treatment and follow-up strategy, atrial fibrillation should be converted to sinus rhythm in patients without structural heart disease. Additionally, it is important to monitor patients who have documented rhythm disorder, loss of consciousness, or abnormal ECG after electrical shock at the time of admission.

Conflict of interest

The authors declare no conflict of interest.

References

1. Fish RM. Electrical injuries. In: JE Tintinalli, GD Kelen, JS Stapczynski, editors. Emergency Medicine: A Comprehensive Study Guide6th ed. ; 2004. p. 1231–1235.
2. SC Carlton. Cardiac problems associated with electrical injury. *Cardiol Clin* 1995;**13**(2):263–266.
3. Fish RM. Electric injury: part II. Specific injuries. *J Emerg Med* 2000;**18**(1):27–34.
4. Arnoldo B, Klein M, Gibran NS. Practice guidelines for the management of electrical injuries. *J Burn Care Res* 2006;**27**(4):439–447.
5. Boggild H, Freund L, Bagger JP. Persistent atrial fibrillation following electrical injury. *Occup Med* 1995;**45**(1):49–50.
6. Celebi A, Gulel O, Cicekcioglu H, Gokaslan S, Kututcularoglu G, Ulusoy V. Myocardial infarction after an electric shock: a rare complication. *Cardiol J* 2009;**16**(4):362–364.
7. Al B, Aldemir M, Guloğlu C, Kara IH, Girgin S. Epidemiological characteristics of electrical injuries of patients applied to the emergency department. *Ulus Travma Acil Cerrahi Derg* 2006;**12**(2):135–142.
8. Arrowsmith J, Usgaocar RP, Dickson WA. Electrical injury and the frequency of cardiac complications. *Burns* 1997;**23**(7–8):576–578.
9. Butler ED, Gant TD. Electrical injuries, with special reference to the upper extremities. *Am J Surg* 1977;**134**(1):95–101.
10. Uzkeser M, Aksakal E, Akoz A, Aslan Ş, Emet M, Cakır Z. Elektrik Carpmasına Bağlı Atriyal Fibrilasyon. *JAEM* 2009;**8**:38–40.
11. Gozlukaya A, Serinken M. Elektrik Carpmasına Bağlı Gelişen Atriyal Fibrilasyon. *JAEM* 2010;**1**:27–29.
12. Akkaş M, Hocagil H, Ay D, Erbil B, Kunt MM, Ozmen MM. Cardiac monitoring in patients with electrocution injury. *Ulus Travma Acil Cerrahi Derg* 2012;**18**(4):301–305.

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Case report

A giant atrial myxoma with fairly atypical features

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ABSTRACT

Atrial myxomas are the most common primary heart tumors. Because of nonspecific symptoms, early diagnosis may be a challenge. Most of these are attached to the inter-atrial septum and are mostly asymptomatic but eventually can cause symptoms depending on the size, mobility, and location of the tumor. We report a case of a large myxoma in the right atrium, which is an uncommon location for this type of tumor.

Case report

A 36-year-old man presented with atypical chest pain and palpitations for the last a month. On examination vital signs were as follows: blood pressure 110/70 mmHg, pulse 110 beats/min, and oxygen saturation 95% on room air, respiratory rate 18 breaths/min. The cardiovascular examinations revealed regular but tachycardic heartbeat with a diastolic murmur of grade 1–2/6 and also jugular venous congestion. The examination of other systems was normal. The ECG detected sinus tachycardia. A chest X-ray showed clear lung fields with cardiomegaly. Complete blood count and basic biochemical parameters were normal. Sedimentation rate was 11 mm/h, CRP was 3 mg/l. Transthoracic echocardiography revealed a mass almost completely fill the right atrium prolapsing through the tricuspid valve into right ventricle (10 × 5 cm diameter) (Fig. 1). The left ventricular size and function were normal in echocardiographic examination. Transesophageal echocardiography confirmed this well-circumscribed mass extending from the right atrium to the right ventricle which caused tricuspid regurgitation and stenosis. Transesophageal images could not be recorded due to technical reasons. The patient was referred to cardiovascular surgery. Right atriotomy was applied (Fig. 2). The tumor, originating from the base of the right atrium, was excised from sufficient depth completely (Fig. 3). Resected area was powered with pericardial patch after extraction process. The result of the pathological examination of tumor was compatible with a myxoma of dimensions 7.5 × 4.5 × 4.5 cm. The patient was discharged one week after the surgery without complaint.

Discussion

This clinical case is quite unusual, considering the massive dimensions of the myxoma, the uncommon localization in the right atrium, the rare clinical presentation as atypical chest pain and palpitation, the sex of the patient and the discrepancy between transthoracic echocardiogram findings and poor clinical presentation.

The clinical manifestations usually depend on the anatomic position and size of the mass. There are mainly 3 types of presentations: embolic, obstructive, and constitutional. Embolic manifestations include visceral infarctions, stroke, and myocardial infarction. In a related study evaluating the risk factors for embolism in cardiac myxoma, location of the myxoma and irregular tumor surface were independently associated with increased risk of embolic complications.[1]

Obstructive manifestations are usually mistaken for valvular stenosis. For instance, mitral valve obstruction due to left atrial myxoma presents with symptoms such as syncope and dyspnea, mimicking mitral valve stenosis. Although right-sided myxomas are rare, the signs and symptoms of RA myxomas are highly variable, to rely on the size, location, and mobility of the tumor.[2] Right atrial myxomas may remain asymptomatic or cause variable signs and symptoms, for example fever, weight loss, arthralgia, Raynaud's phenomenon, anemia, hypergammaglobulinaemia and elevated erythrocyte sedimentation rate.[2] The symptoms were gone after the tumor is removed. Our patient had fever, arthralgia, and anemia. The most prevent symptoms is dyspnea (in 80% of patients). Patients may present with right heart failure secondary to right ventricular outflow tract obstruction, or with syncope secondary to temporary complete obstruction of the tricuspid valve.[3]

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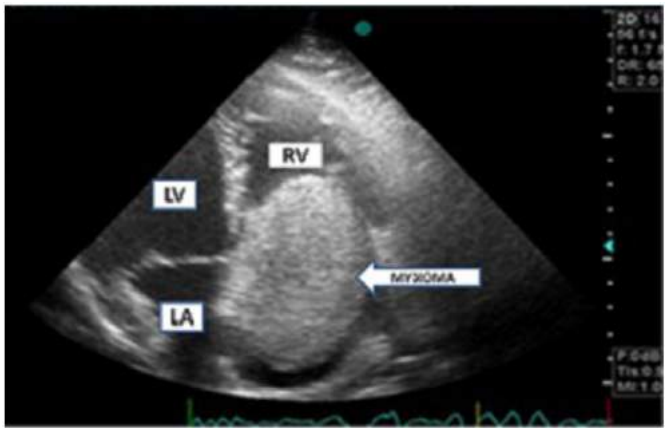

Fig. 1. Transthoracic echocardiography revealed a mass almost completely fill the right atrium prolapsing through the tricuspid valve into right ventricle.

Myxomas are the most common cardiac tumors. Myxomas arise in the left atrium in 75% to 80% of cases but they can also originate from other locations, such as the right atrium (RA) (18% of cases).[4] RA myxomas usually are located from the interatrial septum at the border of the fossa ovalis,[5] but in this case it was originated from the base of the right atrium.

Chest X-rays and electrocardiograms can be non-specific.[6] Transthoracic echocardiography shows an excellent sensitivity in detecting 95% of this unusually situation. On the other hand the sensitivity rises to 100% when transesophageal echocardiography does.[7] Thus echocardiography is the gold standard diagnostic technique. Computed tomography (CT) and magnetic resonance imaging may be useful to show in detail the point of fixation and associated complications. In our patient, an echocardiogram suggested the hypothesis of RA myxoma, which was confirmed by a histopathological exam.

Because of the risk of thromboembolic events, primary treatment is surgical removal of myxoma.[8] but complete resection of tumors is the most important factor. The survival rate after surgery is elevated. The recurrence rate of sporadic tumors is between 1% and 3%.[7] In our case, 6 months and one year after surgery echocardiographic examination showed no evidence of recurrence.

Conclusion

In summary, right atrial myxomas are rare and may have atypical presentation.

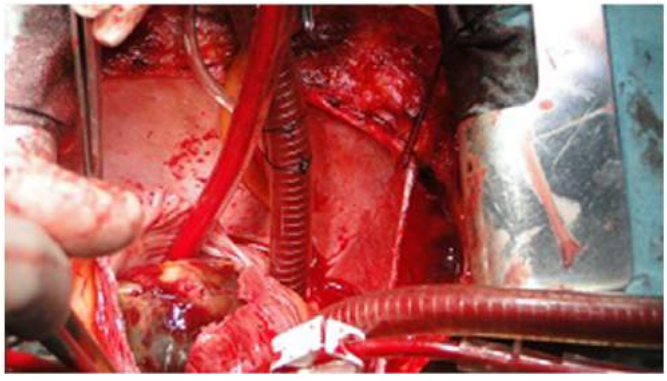

Fig. 2. Macroscopic appearance of the tumor after right atriotomy.

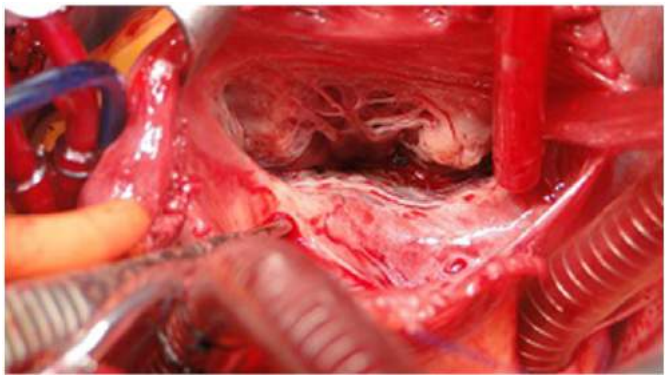

Fig. 3. The view of the right atrium and tricuspid valve after removing the tumor.

Heart tumors should be considered in differential diagnosis of atypical chest pain and palpitation and echocardiography should be performed without delay especially in adult patients with these symptoms. Echocardiography remains the best diagnostic method for locating and assessing the extent of myxomas and for detecting their recurrence, with a sensitivity of up to 100%.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. He DK, Zhang YF, Liang Y, et al. Risk factors for embolism in cardiac myxoma: a retrospective analysis. *Med Sci Monit* 2015;**21**:1146–1154.
2. Reynen K. Cardiac myxoma. *N Engl J Med* 1995;**333**:610–617.
3. Azevedo O, Almeida J, Nolasco T, et al. Massive right atrial myxoma presenting as syncope and exertional dyspnea: case report. *Cardiovasc Ultrasound* 2010;**8**:23-23.
4. Diaz A, Di Salvo C, Lawrence D, et al. Left atrial and right ventricular myxoma: an uncommon presentation of a rare tumor. *Interact Cardiovasc Thorac Surg* 2011;**12**: 622–623.
5. Stolf NA, Benício A, Moreira LF, et al. Right atrium myxoma originating from the inferior vena cava: an unusual location with therapeutic and diagnostic implications. *Rev Bras Cir Cardiovasc* 2000;**15**:255–258.
6. Jang KH, Shin DH, Lee C, et al. Left atrial mass with stalk: thrombus or myxoma? *J Cardiovasc Ultrasound* 2010;**18**:154–156.
7. Oliveira R, Branco L, Galrinho A, et al. Cardiac myxoma: a 13-year experience in echocardiographic diagnosis [in English and Portuguese]. *Rev Port Cardiol* 2010;**29**: 1087–1100.
8. Manfroi W, Vieira SR, Saadi EK, et al. Multiple recurrences of cardiac myxomas with acute tumoral pulmonary embolism [in Portuguese]. *Arq Bras Cardiol* 2001;**77**: 161–163.

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Short communication

Hybrid firefly and Particle Swarm Optimization algorithm for the detection of Bundle Branch Block

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ABSTRACT

Abnormal Cardiac beat identification is a key process in the detection of heart ailments. This work proposes a technique for the detection of Bundle Branch Block (BBB) using hybrid Firefly and Particle Swarm Optimization (FFPSO) technique in combination with Levenberg Marquardt Neural Network (LMNN) classifier. BBB is developed when there is a block along the electrical impulses travel to make heart to beat. ECG feature extraction is a key process in detecting heart ailments. Our present study comes up with a hybrid method combining the two meta-heuristic optimization methods, Firefly algorithm (FFA) and Particle Swarm Optimization (PSO), for feature optimization of ECG (BBB and normal) patterns. One of the major controlling forces is the light intensity attraction of FFA algorithm that models the optimum solution. The light intensity attraction process of the FFA algorithm depends on random directions for search, this may delay in achieving the global optimization solution. The hybrid technique FFPSO, integrates the concepts from FF algorithm and PSO and creates new individuals. In the FFPSO method the local search is performed through the modified light intensity attraction step with PSO operator. The FFPSO features are compared with the classical FF, PSO features. The FFPSO feature values are given as the input to the Levenberg Marquardt Neural Network (LM NN) classifier. It has been observed that the performance of the classifier is improved with the help of the optimized features.

Introduction

Electro-cardiogram is used to access the electrical activity of a human heart. The diagnosis of the heart ailments by the doctors is done by following a standard changes. In this project our aim is to automate the above procedure so that it leads to correct diagnosis. Early diagnosis and treatment is of great importance because immediate treatment can save the life of the patient. BBB is a type of heart block in which disruption to the flow of impulses through the right or left bundle of His, delays activations of the appropriate ventricle that widens QRS complex and makes changes in QRS morphology. The changes in the morphology can be observed through the changes in the ECG. Good performance depends on the accurate detection of ECG features. ECG changes in Left Bundle Branch Block (LBBB) are:

- Increased QRS complex duration (≥0.12 s)
- Increased Q wave amplitude
- Abnormal T wave

ECG changes in Right Bundle Branch Block (RBBB) are:

- Increased QRS complex duration (≥0.12 s)
- RSR' format
- T wave inversion

Detection of BBB using ECG involves three main steps: preprocessing, feature extraction and classification. The first step in preprocessing mainly concentrates in removing the noise from the signal using filters. The next step in the preprocessing is the 'R' peak detection then these 'R' peaks are used to segment the ECG file into beats. The samples that are extracted from each beat contain non-uniform samples. The non-uniform samples in each beat are converted into uniform samples of size 200 by using a technique called resampling. The resampled ECG beat.

In the feature extraction procedure, a fraction of signal around the R peak is extracted as the time-domain features since the R peak of ECG signals is an important index for cardiac diseases. To ensure the important characteristic points of ECG like P, Q, R, S and T are included, a total of 200 sampling points before and after the R peak are collected as one ECG beat sample.

P, Q, R, S and T waves provides information regarding amplitudes and relative time intervals of ECG. These changes in the ECG are called morphological transitions. The morphological changes (P, QRS complex, T, U waves) of ECG are due to the abnormalities in the heart. BBB is one

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such morphological abnormality seen in the heart diseases. In the previous studies morphological features are extracted for clinical observation of heart diseases. The feature extraction using traditional techniques generally yield a large number of features, and many of these might be insignificant. Therefore, the common practice is to extract key features useful in the classification.

This paper presents meta-heuristic FFPSO, is used as a feature extraction method instead of using traditional feature extraction/optimization techniques. A large number of meta-heuristic techniques have been designed to solve feature optimization problem. Some of the methods among all these are Genetic Algorithm (GA),[7] Particle Swarm Optimization (PSO),[6] Bacterial Foraging Optimization(BFO),[4,5,3] Firefly Algorithm (FFA)[13] etc.

Meta-heuristic algorithms are proven to outperform the gradient based algorithms for real world optimization problems. Firefly algorithm[1] is one such newly designed algorithm mimicking flashing mechanism of fireflies. A detailed explanation and formulation of the firefly algorithm is given in Section 4.

Traditional Firefly Algorithm (FFA)[2] has one disadvantage of getting trapped into the local optimum. Sometimes it is unable to come out of that state. The parameters in the firefly algorithm are fixed and do not have any mechanism to remember the previous best situation of each firefly and this makes them move regardless of its previous better solution.

In this paper, a novel hybrid optimization method concurrently combines the FFA with the PSO. Now a days the PSO[10] is a swarm based optimization algorithm and it takes inspiration from a group of birds or a group of fish etc. The proposed hybrid algorithm fulfills local search by using the light intensity operation mechanism of FFA whereas the global search is accomplished by a PSO operator. Using this combination it maintains a balance between 'exploration' and 'exploitation' and enjoying the best of both the algorithms (FFA and PSO).[12] The proposed method, referred to as Firefly Particle Swarm Optimization (FFPSO) has been compared with the normal PSO and FFA. The following comparative measures were used to study the (i) accuracy of the final solution, and (ii) convergence speed. Such comparison shows the superiority of the proposed algorithm. This algorithm outperformed both PSO and FFA over a few ECG benchmarks sets for the classification problem.

The ECG classification flow diagram is shown in the Fig. 1.

Preprocessing

To prove the performance of proposed technique, the usual MIT BIH arrhythmia database[9] is considered. The data used in this algorithm confines to 11 recordings that consists of 5 normal, 3 LBBB and 3 RBBB for a duration of 60 min at 360 Hz sampling rate. Total number of ECG beats used for classification are 19,039. De-noising of ECG data is a preprocessing step that removes noise and makes ECG file useful for subsequent steps in the algorithm. The Sgolay FIR smoothing filter is used for removing the noise in ECG signals. The next step in the preprocessing is the R peak detection, then segmentation of ECG file into beats (P, QRS Complex), by taking R peaks as the reference points.

Feature extraction

In the feature extraction procedure, a fraction of signal around the R peak is extracted as the time-domain features since the R peaks of ECG signal are an important index for cardiac diseases. To ensure the important characteristic points of ECG like P, Q, R, S and T are included, a total of 200 sampling points before and after the R peak are collected as one ECG beat sample. The samples that are extracted from each beat contain nonuniform samples. The nonuniform samples in each beat are

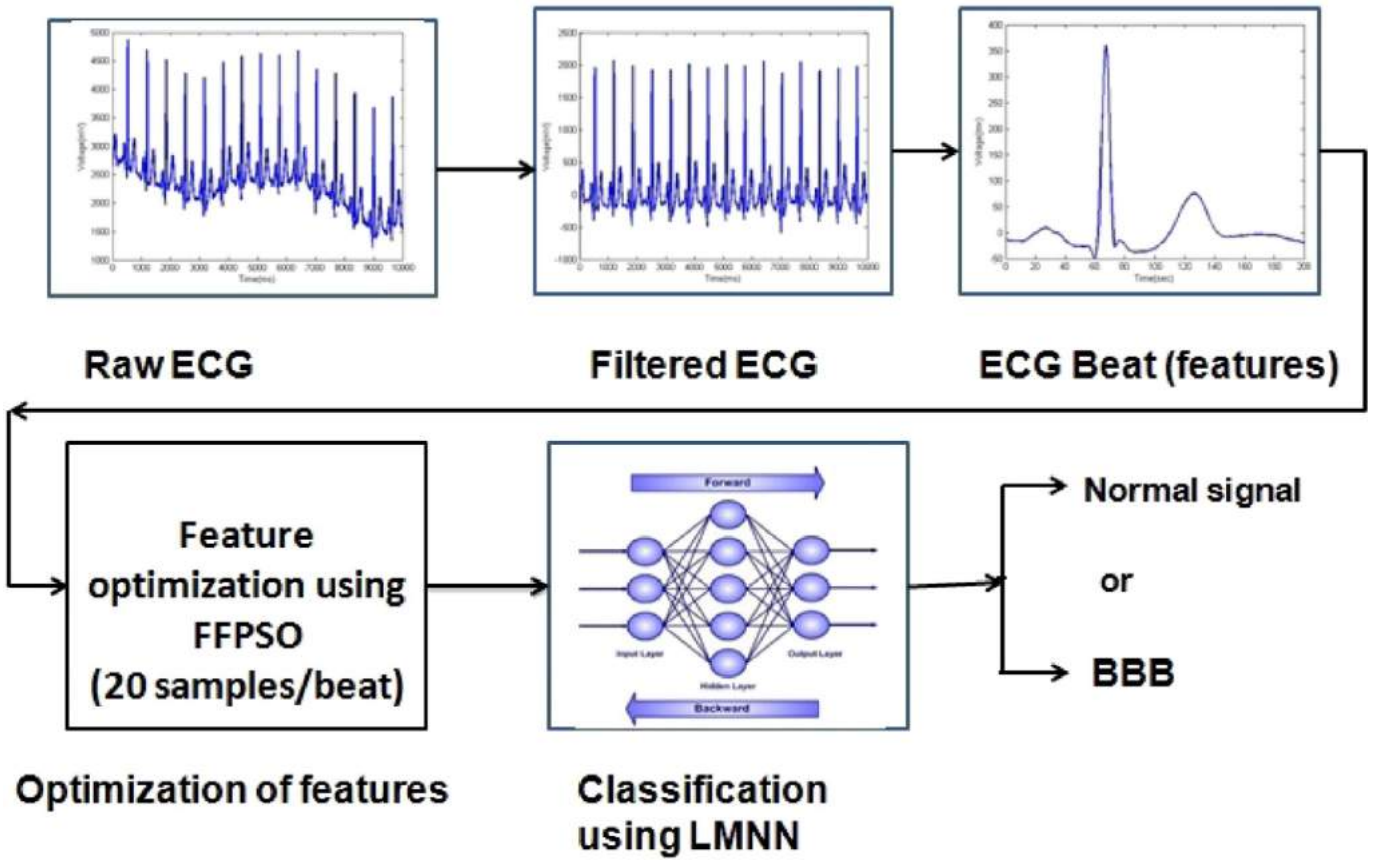

Fig. 1. ECG classification using FFPSO.

converted into uniform samples of size 200 by using a technique called resampling. The resampled ECG beat samples/features.

Feature optimization

Particle Swarm Optimization (PSO)

PSO[11] is a kind of swarm based optimization method developed by Eberhart and Kennedy inspired from the behavior of a flock of birds. Each particle in the group flies in the search domain with a velocity and it tries to attain the best velocity according to its own previous best (pbest) and its companions' best (gbest) flying experience.

The advantage of using PSO over other optimization techniques is its simplicity. And very few parameters need to be adjusted. Due to this, PSO has been widely used in a variety of applications. In an n-dimensional search space, $X_i = (x1, x2, x3,\ldots,xn)$, let the particles be initialized with positions X_i and velocities V_i and the fitness is calculated based on particle positional coordinates as the input values. Then the particles are moved into new positions using the equations below:

$$V_i(i+1) = \omega.V_i(i) + C1.\phi1.(Pbest - X_i(i)) + C2.\phi2.(gbest - X_i(i)) \quad (1)$$

$$X_i(i+1) = X_i(i) + V_i(i+1) \quad (2)$$

Firefly algorithm (FFA)

This algorithm was designed by a mathematician X.S. Yang in the year 2007. FFA was formulated by mimicking the flashing (mating) activity of fireflies. Even though this algorithm is similar to the PSO,[6] Artificial Bee Colony (ABC) Optimization[14] and Ant Colony Optimization (ACO),[15] proved to be much simpler in algorithm implementation.

Fireflies are small insects, which are capable of producing light to attract a prey (mate). They release small rhythmic light flashes. The light intensity attraction 'I' of fireflies decreases with the distance 'r'. Hence, most fireflies are visible only up to several hundreds of meters. To execute this algorithm the fitness function is articulated based on the fluorescence light behavior of fireflies. For simplicity, it is imagined that light intensity attractiveness of firefly is determined by its brightness 'I' which is in turn connected with the fitness function.

Attractiveness and light intensity

At a particular position 'r', the brightness 'I' of a firefly can be chosen as I (r), proportional to the fitness, for a maximization problem. So the I (r) varies according to the well known inverse square law.

$$I(r) = \frac{Is}{r^2} \quad (3)$$

Fireflies attractiveness β is proportional to the I (r) seen by surrounding fireflies can be defined as

$$\beta = \beta_0 e^{-\gamma r^2} \quad (4)$$

where γ is the light absorption coefficient.

Distance

The distance between any 2 fireflies is estimated using the distance formula.

$$r_{i,j} = \sqrt{\sum_{k=1}^{d} (x_{i,k} - x_{j,k})^2} \quad (5)$$

Firefly 'i' is moved towards brighter firefly 'j' and its movement is calculated by

Movement

$$x_i = x_i + \beta_0 e^{-\gamma r_{i,j}^2}(x_i - x_j) + \alpha\epsilon_i \quad (6)$$

The first term in Eq. (6) denotes the current location of a firefly, the second term is used for determining the attractiveness (β) of a firefly (attractive firefly), towards the attractive neighboring fireflies and the third term indicates the random walk of a firefly (random part).

$$xi = xi + \alpha(rand - 1/2) \quad (7)$$

when firefly 'i' lacks the brighter firefly 'j' then it will go for a random walk as in Eq. (7), in search of the best candidate, where the coefficient α is a randomization variable, and 'rand' is a random number consistently spread over the space (0, 1).

The pseudo code for firefly algorithm is given below

Pseudo code: firefly algorithm

1. Generate the initial population randomly.
2. Calculate the fitness of initial population based on light intensity of fireflies.
3. While (t < termination criteria is satisfied)
4. For i = 1:p (p fireflies)
5. For j = 1:p
6. Calculate light intensity (I) using Eq. (3).
7. Distance between two fireflies is calculated using Eq. (5).
8. If (I(i) < I (j))
9. Firefly i is moved towards firefly j using Eq. (6).
10. Determine new solutions.
11. Else
12. Firefly i is moved randomly towards j using Eq. (7).
13. End If
14. End for j.
15. End for i.
16. End while
17. Sort the fireflies according to light intensity values of the new solution.

Proposed approach: hybrid FF and PSO (FFPSO) algorithm

In this section, a part of PSO is used in the FFA to increase convergence and also to enhance its capability for not falling into the local minimum. The FFPSO has exactly the same steps as the FFA with the exception that the position vector of FFA is modified as follows: In the FFPSO, the distance between xi and $pbest_i$, is the Cartesian distance

$$r_{px} = \sqrt{\sum_{k=1}^{d} (pbest_{i,j} - x_{i,j})^2} \quad (8)$$

The distance between xi and $gbest_i$, is the Cartesian distance

$$r_{gx} = \sqrt{\sum_{k=1}^{d} (gbest_{i,j} - x_{i,j})^2} \quad (9)$$

The position vectors xi of the FFPSO is randomly mutated by using Eq. (8)

$$x_i(t+1) = wx_i(t) + c_1 e^{-r_{px}^2}(pbest_i - x_i(t)) + c_2 e^{-r_{gx}^2}(gbest_i - x_i(t)) + \alpha(\gamma - 1/2) \quad (10)$$

Pseudo code: FFPSO

1. Generate the initial population randomly.
2. Initialize pbest and gbest.
3. Calculate the fitness of initial population based on light intensity of fireflies.
4. While (stopping criteria is satisfied)
5. For i = 1:p (p fireflies)
6. For j = 1:p
7. Light intensity I is determined using Eq. (3).
8. Distance between pbest-xi and gbest-xi is calculated using Eqs. (8) and (9).
9. If (I(i) < I(j))
10. Firefly i is moved towards firefly j using Eq. (10)
11. Else
12. Firefly i is moved randomly towards firefly j using Eq. (7).
13. End If
14. Calculate the new solutions and update the light intensity value
15. Update pbest and gbest.
16. End for j
17. End for i
18. End while
19. Sort the fireflies in descending order based on their light intensity

In the suggested approach, the light intensity attraction step of each particle gets mutated by a PSO operator. At this step, each particle is randomly attracted towards the gbest position in the entire population. Local search in different regions is carried by the modified attractiveness step of the FFPSO algorithm. The main objective of FFPSO feature selection stage is to reduce the features of the problem before the supervised neural network classification. Among all the wrapper algorithms used, FFPSO, which solves optimization problems using the methods of flashing behavior of fireflies, has emerged as a promising one.

Classification of ECG with firefly features

The extracted features from FFPSO algorithm (20 features) are classified using different types of classification techniques such as K-Nearest Neighbor (KNN), Support Vector Machine (SVM), LM Neural Network classifiers.

Levenberg-Marquardt Neural Network (LM NN)

In this work for the detection of BBB, back propagation Levenberg-Marquardt Neural Network (LMNN)[19] was used. This NN provides rapid execution of the network to be trained, which is the main advantage in the neural signal processing applications,[8] citeKP. The NN was designed to work well if it was built with 20 input neurons, 10 neurons in the hidden layer and 3 neurons in the output layer. The performance of this algorithm was compared with Scalar Conjugate Gradient (SCG) NN. The LMNN algorithm is a robust and a very simple method for approximating a function. SCG NN method provides conjugate directions of search instead of performing a linear search. The network is trained with 11,039 ECG beats, and tested with 8000 ECG beats. The total number of iterations is set to 1000 and mean square error less than 0.001. The main advantage of this algorithm is that the time required to train the network is less.

Results

ECG features before optimization = [1 2 3 .....200];

The optimized features are = [41, 14, 198, 17, 189, 139, 22, 81, 177, 1, 171, 82, 134, 40, 49, 38, 80, 86, 129, 138];

These reduced features are given as input for the Neural Network so that its convergence speed and final accuracy can be increased. The ECG beats after segmentation are re-sampled to 200 samples/beat. Instead of using morphological feature extraction techniques, in this paper FFPSO is used as the feature extraction technique. Using FFPSO ECG beat features are optimized to 20 features. The FFPSO gives optimized features (best features) for the classification. The performance of FFPSO is compared with classical FFA and PSO techniques. The FFA, PSO and FFPSO features are classified using SVM, KNN, SCG NN and LM NN as in Table 1.

- Count of Normal beats used for classification—9193.
- Count of RBBB beats user for classification—3778.
- Count of LBBB beats user for classification—6068.
- Total number of beats used for classification—19,039.
- Count of correctly classified beats—18,800.
- Total misclassified beats—239.

For measuring accuracy two parameters sensitivity and specificity are calculated using the following equations.

$$Specificity = \frac{True\_Negative}{True\_Negative + False\_Positive} X100 \quad (11)$$

$$Sensitivity = \frac{True\_Positive}{True\_Positive + False\_Negative} X100 \quad (12)$$

$$Accuracy = \frac{TP + TN}{TP + TN + FP + FN} X100 \quad (13)$$

- TP (True_Positive) = count of all the correctly classified normal beats.
- TN (True_Negative) = count of all beats the correctly classified abnormal beats.
- FP (False_Positive) = count of normal beats which are classified as abnormal.
- FN (False_Negative) = count of abnormal beats which are classified as normal.

In the training mode we applied multilayer NN and checked the network performance and decided if any changes were required to the training process or the data set or the network architecture.

Discussion

The proposed FFPSO is compared against other three BBB detection algorithms such as Wavelet Transform (WT), Continuous wavelet Transform (CWT), Wavelet transform and Probabilistic Neural Network (PNN) in terms of related features selected from the original database and classification accuracy obtained from different classifiers using Matlab software.

The work in,[18] explores an experimental study of using WT for extracting relevant features and KNN based classifier for the detection of BBB. The work presented in,[17] uses morphological features for classification using SVM. The work proposed in,[16] uses Arrhythmia dataset

Table 1
Classification with LM NN classifier.

Classifier	Sensi	Speci	Accuracy
FFA + SVM	76.2%	75.47%	72.13%
PSO + SVM	71.0%	73.13%	70.12%
FFPSO + SVM	95.5%	96.9%	96.74%
FFA + SCG NN	88.2%	87.2%	87.9%
PSO + SCG NN	86.1%	85.3%	86.0%
FFPSO + SCG NN	97.42%	92.28%	97.13%
FFA + KNN	53.5%	52.2%	53.22%
PSO + KNN	52.5%	53.2%	65.1%
FFPSO + KNN	92.35%	93.9%	92.17%
FFA + LM NN	93.34.2%	92.2%	93.9%
PSO + LM NN	91.2%	89.2%	80.9%
FFPSO + LM NN	99.97%	98.7%	99.1%

Table 2
Comparative study for detection of BBB.

Studies	Approach	Accuracy
R Ceylan et al. (2011)[17]	Wavelet Transform (WT)	98.1%
Kutlu et al. (2008)[18]	Continuous Wavelet Transform (CWT)	97.3%
Yu et al. (2007)[16]	WT and Probabilistic Neural Network	98.39%
Proposed approach	FFPSO and Neural Network	99.1%

taken from MIT/BIH repository and 20 morphological and wavelet features are extracted then PNN is used for supervised learning and classification. From the experiments, it is concluded that the proposed FFPSO with LMNN classifier outperformed other three algorithms with selection of minimal number of relevant features. This increases the classification accuracy as shown in Table 2. The FFPSO employed to intelligently select the most relevant features that could increase the classification accuracy while ignoring noisy and redundant features.

Conclusion

It is evident from the results that hybrid FFPSO approach out performs the other two optimization methods in terms of accuracy and convergence rates. In the present study, we developed a simple computational model for the detection of ECG BBB using the FFPSO algorithm. The FFPSO algorithm was compared with the FFA and PSO . In our study the following were observed: 1) accuracy 2) convergence speed. The FFPSO method was shown to provide better results than original FFA and PSO for all the tested data.

References

1. Mishra Anurag, et al. Optimized gray-scale image watermarking using DWT – SVD and firefly algorithm. *Expert Syst Appl* 2014;**41**(17):7858–7867.
2. Sahu Rabindra Kumar, Panda Sidhartha, Padhan Saroj, et al. A hybrid firefly algorithm and pattern search technique for automatic generation control of multi area power systems. *Int J Electr Power Energy Syst* 2015;**64**:9–23.
3. Nasir Ahmad NK, Tokhi MO, Ghani N, et al. Novel adaptive bacteria foraging algorithms for global optimization. Applied Computational Intelligence and Soft Computing; 2014.
4. Passino Kevin M. Biomimicry of bacterial foraging for distributed optimization and control. *Control Syst* 2002;**22**(3):52–67.
5. Liu Y, Passino KM, et al. Biomimicry of social foraging bacteria for distributed optimization models, principles, and emergent behaviors. *J Optim Theory Appl* 2002;**115**(3):603–628.
6. Kennedy J, Eberhart R, et al. Particle swarm optimization. Proc. IEEE Int. Conf. Neural Netw; 1995. p. 1942–1948.
7. Back T. Evolutionary algorithms in theory and practice: evolution strategies, evol. programming. Proc. Genetic Algorithms. London, U.K.: Oxford Univ. Press; 1996
8. Sapna S, Tamilarasi A, Kumar MP, et al. Backpropagation learning algorithm based on levenberg marquardt algorithm. CS and IT-CSCP; 2012;393–398.
9. Goldberger AL, Amaral LA, Glass L, Hausdorff JM, Ivanov PC, Mark RG, Stanley HE, et al. Physiobank, physiotoolkit, and physionet components of a new research resource for complex physiologic signals. *Circulation* 2000;**101**(23):e215–e220.
10. Eberhart RC, Kennedy J, et al. A new optimizer using particle swarm theory. Proc. ISMMH S; 1995. p. 39–43.
11. Melgani F, Bazi Y, et al. Classification of electrocardiogram signals with support vector machines and particle swarm optimization. *IEEE Trans Inf Technol Biomed* 2008;**12**(5).
12. Abd-Elazim SM, Ali ES, et al. A hybrid particle swarm optimization and bacterial foraging for optimal power system stabilizers design. *Int J Electr Power Energy Syst* 2013;**46**:334–341.
13. Horng Ming-Huwi, et al. Firefly meta-heuristic algorithm for training the radial basis function network for data classification and disease diagnosis. INTECH Open Access Publisher; 2012.
14. Pal SK, Rai CS, Singh AP, et al. Comparative study of firefly algorithm and particle swarm optimization for noisy non-linear optimization problems. *Int J Intell Syst Appl* 2002;**4**(10):50.
15. Dorigo Marco, Stutzle Thomas. The ant colony optimization metaheuristic: algorithms, applications, and advances. Handbook of metaheuristics. US: Springer; 2003;250–285.
16. Yu Wei, et al. Application of multi-objective genetic algorithm to optimize energy efficiency and thermal comfort in building design. *Energy Build* 2015;**88**:135–143.
17. Ceylan Rahime, ozbay Yksel, et al. Wavelet neural network for classification of bundle branch blocks. *Proc World Congr Eng* 2011;**4**(2).
18. Kutlu Yakup, Kuntalp Damla, Kuntalp Mehmet, et al. Arrhythmia classification using higher order statistics. Signal Processing, Communication and Applications Conference; 2008.
19. Kora Padmavathi, Sri Rama Krishna K. Improved Bat algorithm for the detection of myocardial infarction. *SpringerPlus* 2015;**4**(1):666.

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Case report

Malignant right coronary artery originating from left coronary sinus

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ABSTRACT

Congenital coronary artery anomalies are rare and usually an incidental finding during selective coronary angiography. Most of these variations appear to be of no clinical significance but some do have potentially serious sequelae. Anomalous origin of the right coronary artery from the left sinus of valsalva with subsequent coursing between the aorta and pulmonary trunk called "malignant course" is a relatively uncommon congenital defect. In this report we present a patient with a malignant right coronary artery originating from left coronary sinus

Introduction

Congenital coronary artery anomalies are rare and usually an incidental finding during selective coronary angiography. The incidence of coronary anomalies was reported as 1.3% in a large registry of patients undergoing coronary angiogram.[1] When there is only one coronary artery arising from the aortic trunk by a single coronary ostium the condition is called single coronary artery (SCA). Most of these variations appear to be of no clinical significance but some do have potentially serious sequelae.[2] Anomalous origin of the right coronary artery from the left sinus of valsalva with subsequent coursing between the aorta and pulmonary trunk called "malignant course" is a relatively uncommon congenital defect. The prevalence rate for the RCA branching from the left coronary sinus was reported as 0.43% in patients undergoing computed tomography coronary angiography.[3] Angiography based reports revealed this incidence between 0.03 and 0.17%.[1] It is well established that this anomaly can cause angina pectoris, myocardial infarction, or sudden cardiac death in the absence of atherosclerosis.[4] In this report, we present a patient with a malignant right coronary artery originating from left coronary sinus

Case report

A 63-year-old man with hypertension and chronic obstructive pulmonary disease was admitted to our clinic for evaluation of atypical chest pain and palpitation. On admission, his blood pressure was 120/85 mmHg and the pulse rate was 76 beats/min and a 2/6 degree apical pansystolic murmur was heard. The 12-lead electrocardiography (ECG) showed normal sinus rhythm. Transthoracic echocardiography showed a mildly dilated left atrium with mild-to-moderate mitral regurgitation and preserved ejection fraction. Treadmill exercise testing with the Bruce protocol was ambigous. Ambulatory 24 hour ECG showed frequent ventricular extrasystoles (VES). The selective coronary angiography was performed. Right coronary artery was vizualized as anomalously originating from left coronary sinus valsalva (Fig. 1A). The attempt to engage the right coronary catheter to the right coronary artery was unsuccessful and aortic root angiography showed no coronary ostium originating from the right sinus of valsalva. The coronary system was free of significant atherosclerotic disease. 320-slice ECG-gated multidetector computed tomography (prospective gating) showed a right coronary artery originating from left coronary sinus with an interarterial course called "malignant course" (Fig. 1B–C–D). As the stress myocardial perfusion detected no ischemia, the patient was discharged with the medical treatment including beta blocker threapy for frequent VES and has been followed up at the outpatient clinic uneventfully for two years.

Discussion

When a single coronary ostium provides blood flow for supplying the entire heart, the condition is called SCA. SCA is a rare congenital anomaly of the coronary circulation and is associated with other congenital cardiac malformations such as transposition of the great vessels, coronary arteriovenous fistula, or bicuspid aortic valve.[1] SCA has generally been regarded as a benign anomaly. The prevalence of anomalous origin and course of coronary arteries is about 0.7–1.96%.[5,6] The anomalous origin may have interarterial, retro-aortic, prepulmonic or septal course, the most common being inter-arterial. If RCA course is not

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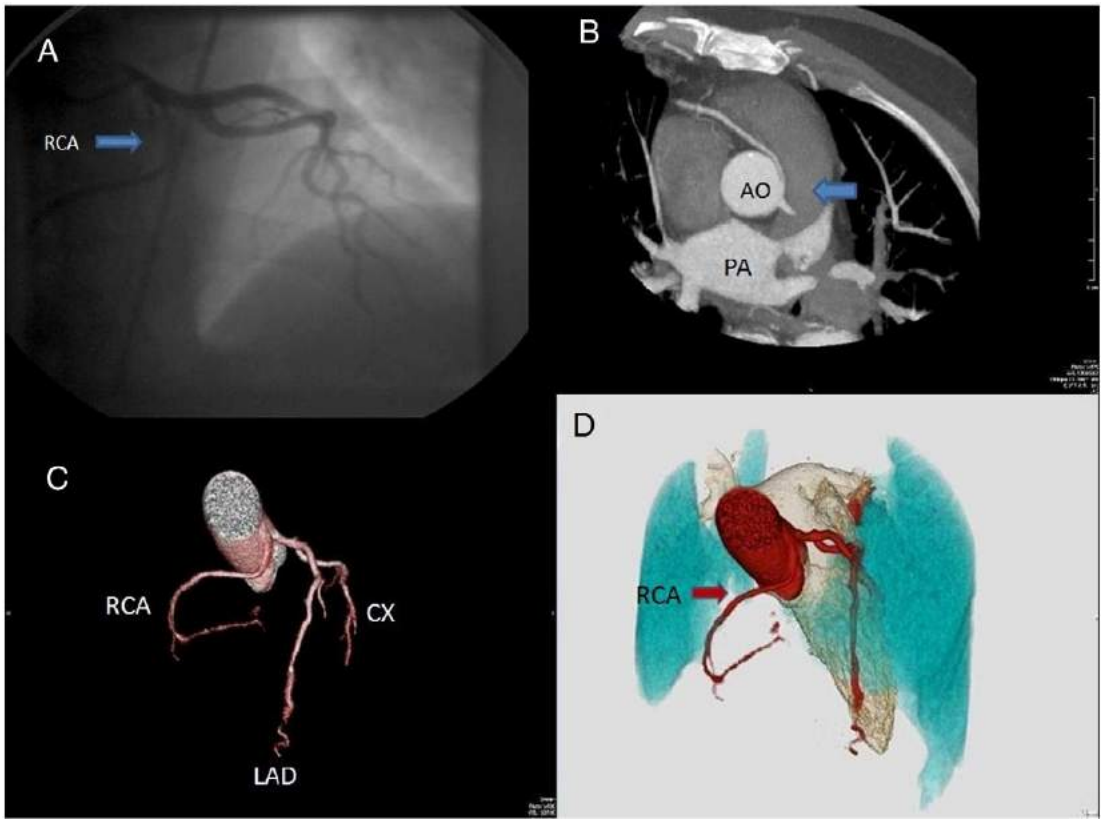

Fig. 1. A: Selective angiogram of the left coronary system in the right anterior oblique projection. The arrow shows right coronary artey (RCA) originating from left coronary sinüs valsalva. **B**: Maximum intensity projection of top of heart by multidetector computed tomography. The arrow is showing both RCA and left coronary artey originating from left coronary sinus. RCA courses between pulmonary artery (PA) and aorta (AO). **C**: 3D-volume rendered image of of multidetector computed tomography showing anomalous right coronary artey (RCA) (pulmonary artery is not demonstrated). **D:** 3D volume rendered image of multidetector computed tomography. The arrow is showing the RCA with an interarterial course. (pulmonary artery is not demonstrated).

between aorta and pulmonary artery, this anomaly is accepted as relatively benign form. A malignant or interarterial course of the right or left coronary artery arising from the opposite coronary sinus has been well described and may have serious sequelae. The interarterial course of a single coronary vessel is subject to compression, which may result in a higher incidence of angina, myocardial infarction, and sudden death. In a majority of previously reported cases, sudden death was triggered by exertion and most of these patients have a positive exercise stress test.[7] Several factors including acute angulation at the origin, compression of the vessel between the aorta and pulmonary artery, slit like ostium and intramural proximal intussusception of the ectopic artery at the aortic-root wall have been proposed for this association.[8] The traditional terminology (interarterial course) implies that the aberrant artery was liable to a scissors-like mechanism, created by the close proximity of the aorta and pulmonary artery. Compared with invasive angiography, multidetector computed tomography allows a more accurate depiction of the origin and course of the anomalous coronary artery. In combination with stress myocardial perfusion, multidetector computed tomography can provide an accurate diagnosis and a complete anatomic and functional assessment of this potentially lethal anomaly to guide patient management.[9,10] The prognosis in single coronary artery is unclear. The incidence of sudden death with this anomaly is estimated at 25–40% and is associated with exercise in most of the reported cases.[11] Revascularization is recommended for documented coronary ischemia in the setting of an anomalous coronary artery coursing between aorta and pulmonary arteries by American College of Cardiology and American Heart Association (ACC/AHA) guidelines for congenital heart diseases.[12] In the present case we decided a conservative strategy, as the patient lived uneventfully for 70 years and there was no documented ischemia with SPECT myocardial perfusion imaging.

Conclusion

Most of these variations appear to be of no clinical significance but some do have potentially serious sequelae. Anomalous origin of the RCA from the left sinus of valsalva with subsequent coursing between the aorta and pulmonary trunk is called “malignant course”. It is well established that this anomaly can cause angina pectoris, myocardial infarction, or sudden cardiac death in the absence of atherosclerosis. Revascularization is recommended only if there is substantial atherosclerosis and documented ischemia.

References

1. Yamanaka O, Hobbs RE. Coronary artery anomalies in 126,595 patients undergoing coronary arteriography. *Catheter Cardiovasc Diagn* 1990;**21**:28–40.
2. Angelini P, Velasco JA, Flamm S. Coronary anomalies: incidence, pathophysiology, and clinical relevance. *Circulation* 2002;**105**:2449–2454.
3. Erol C, Seker M. Coronary artery anomalies: the prevalence of origination, course, and termination anomalies of coronary arteries detected by 64-detector computed tomography coronary angiography. *J Comput Assist Tomogr* 2011;**35**:618–624.
4. Dirksen MS, Bax JJ, Blom NA, et al. Detection of malignant coronary artery anomaly by multi-slice CT coronary angiography. *Eur Radiol* 2002;**12**:S177–S180.
5. Yildiz A, Okcun B, Peker T, Arslan C, Olcay A, Vatan MB. Prevalence of coronary artery anomalies in 12,457 adult patients who underwent coronary angiography. *Clin Cardiol* 2010;**33**:60–64.
6. Safak O, Gursul E, Yesil M, et al. Prevalence of coronary artery anomalies in patients undergoing coronary artery angiography: a reviewof 16768 patients. A retrospective, single-center study. *Minerva Cardioangiol* 2015;**63**:113–120.
7. Basso C, Maron BJ, Corrado D, Thiene G. Clinical profile of congenital coronary artery anomalieswith origin from the wrong aortic sinus leading to sudden death in young competitive athletes. *J Am Coll Cardiol* 2000;**35**:1493–1501.
8. Angelini P, Velasco JA, Ott D, Khoshnevis GR. Anomalous coronary artery arising from the opposite sinus: descriptive features and pathophysiologic mechanisms, as documented by intravascular ultrasonography. *J Invasive Cardiol* 2003;**15**:507–514.

9. Datta J, White CS, Gilkeson RC, et al. Anomalous coronary arteries in adults: depiction at multi-detector row CT angiography. *Radiology* 2005;**235**:812.
10. Cohenpour M, Tourovski A, Zyssman I, et al. Anomalous origin of left main coronary artery: the value of myocardial scintigraphic and spiral computed tomography scans. *Nucl Med Rev Cent East Eur* 2006;**9**:69–71.
11. Frescura C, Basso C, Thiene G. Anomalous origin of coronary arteries and risk of sudden death: a study based on the autopsy population of congenital heart disease. *Hum Pathol* 1998;**29**:689–695.
12. Warnes CA, Williams RG, Bashore TM, et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (writing committee to develop guidelines on the management of adults with congenital heart disease). Developed in collaboration with the American Society of Echocardiography, Heart RhythmSociety, International Society for Adult Congenital Heart Disease, Society for Cardiovascular Angiography and Interventions, and Society ofThoracic Surgeons. *J Am Coll Cardiol* 2008;**52**:e143–e263.

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Case report

ST elevation myocardial infarction caused by coronary slow flow: Case report and brief review of the literature

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ABSTRACT

Coronary Slow Flow Phenomenon (CSFP) is an angiographic phenomenon in which vessel opacification is delayed without any evidence of obstructive epicardial coronary disease. We aim to present, in this paper, extremely slow coronary flow along with its severe clinical manifestation. A 47-year-old male patient was admitted to our emergency department with ST elevation myocardial infarction caused by coronary slow flow. Oral Acetylsalicylic acid, nebivolol and atorvastatin therapy successfully resulted in complete resolution of his symptoms during the 18-month observation.

Introduction

The Coronary Slow Flow Phenomenon (CSFP), an angiographic phenomenon first described by Tambe et al. in 1972, refers to the slow movement of contrast agents in the coronary arteries without significant stenosis or myocardial bridge.[1] The Thrombolysis in Myocardial Infarction (TIMI) frame count, a reproducible index of coronary flow, functions as the quantitative measurement of coronary slow flow.[2] More specifically, it is a numerical representation of cine frames which are necessary for the contrast agents to reach a pre-specified distal coronary artery landmark. The process is continuous with a correction through normalization of the left anterior descending artery (LAD). The corrected TIMI frame count (CTFC) is the division of the absolute TIMI frame count in the LAD by 1.7. The CSFP phenomenon is characterized by CTFC >2 standard deviations from normal published range (21 ± 3).[2] It has been reported that 1–5.5% of patients who undergo coronary angiography experience CSFP.[3] However, the pathophysiology of CSFP has not been comprehensively discerned. In this paper, we present a rare case of STEMI caused by coronary slow flow, which might help us to understand better the CSFP.

Case

A 47-year-old man who complained of chest pain spreading towards his shoulder, the pain having begun nearly an hour prior, was admitted into our emergency department. His blood pressure was 110/75 mm Hg, his heart rate was 52 beats/min, and his cardiopulmonary examination was normal. His electrocardiogram showed ST elevation in leads V1–4 (Fig. 1A). Then, he was immediately taken to the laboratory for coronary angiography. There was no significant stenosis, coronary vasospasm or myocardial bridge in coronary angiography. Further, intracoronary nitro-glycerine fusion was performed to exclude vasospastic angina. However, no change in ST elevation was observed. On the other hand, advanced-degree slow flow was observed in the LAD and right coronary artery (RCA). The TIMI frame-count method was used to measure the degree of slow flow. The CTFCs were observed to be 52 frames for the left anterior descending coronary artery (Fig. 2A) and 35 frames for the RCA (Fig. 2B). After about 30 min, the patient's chest pain had disappeared completely; ST elevation improved (Fig. 1B); and Troponin I was slightly elevated (Troponin I 0.24 μg/L (normal range, 0.010–0.023 μg/L)). In terms of lipid profile, the findings were as follows: total cholesterol: 182 mg/dL, LDL: 109 mg/dL, HDL: 35 mg/dL, and TG: 192 mg/dL. Other biochemical values were normal, his body mass index was in the normal range, and there were no cardiac risk factors other than smoking. Two-dimensional echocardiography showed normal left ventricle function and no wall motion abnormalities. Acetylsalicylic acid 300 mg, nebivolol 5 mg and atorvastatin 20 mg were started. The patient was discharged without chest pain after four days with a prescription of acetylsalicylic acid 100 mg, nebivolol 5 mg and atorvastatin 20 mg. There was no complaint reported during the 18-month period after discharge.

Discussion

Recurrent chest pain is a commonly observed symptom among patients with CSFP.[4] Although it is rare, CSFP may also lead to life-threatening situations such as ST elevation myocardial infarction

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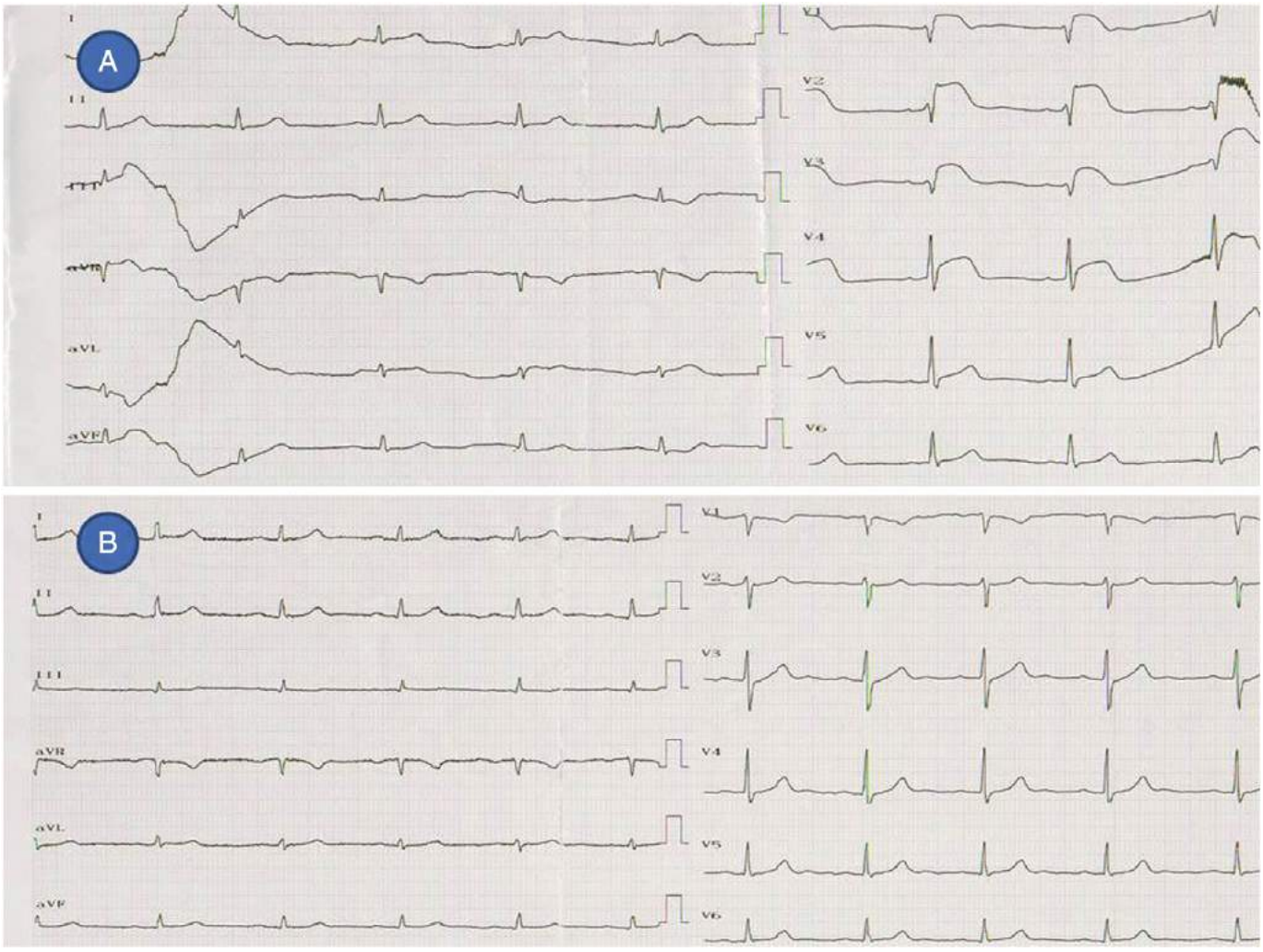

Fig. 1. The electrocardiogram shows ST elevation in leads V1–4 during presence of chest pain (A). ST elevation improved after disappearance of chest pain (B).

(STEMI), ventricular arrhythmias, and sudden cardiac death.[5–10] Recently, a patient presented as having acute coronary syndrome reportedly illustrated an association between CSFP and a new onset of intermittent left bundle branch block (LBBB).[11] Also, Sunbul et al.[12] suggested that slow coronary flow might lead to ST segment elevation in the exercise stress test.

Although different theories have been posited regarding the causes of CSFP, research has not been able to discern comprehensively its pathogenesis. In a variety of researches, small vessel disease, endothelial dysfunction, subclinical atherosclerosis, inflammation, and anatomic properties of coronary arteries have been reported as existing in association with CSFP. Among these, small vessel dysfunction is one of the most typical of the pathogenesis of CSFP.[13] In an effort to confirm this hypothesis, Beltrame et al. suggested that fibromuscular hyperplasia, medial hypertrophy, myointimal proliferation, endothelial edema, and the thickening as well as the degeneration of the coronary micro-vessels correspond with the phenomenon.[14] Therefore, it is perhaps more fitting to suggest that the coronary microcirculation might be a result of the coexistence of structural and functional abnormalities. In a variety of cases, patients with CSFP were reported to have increased levels of plasma homocysteine, increased endothelin-1 release and reduced nitric oxide bioactivity, which indicated impaired endothelial function.[3,15–18] CSFP patients have metabolic syndrome more frequently.[19] As a result of the use of IVUS technique and flow rate measurements, diffuse intimal thickening, widespread calcification along the coronary vessel wall, and non-obstructive atheromatous coronary changes were reported among patients with CSFP.[20] Based on the data presented here, it can be argued that CSFP might reflect diffuse, non-obstructive atherosclerotic disease of epicardial vessels together with microvascular disease. Moreover, elevated plasma concentration of high-sensitivity C-reactive protein and interleukin-6 were documented among CSFP patients.[21] In a similar vein, higher levels of plasma-soluble adhesion molecules such as intercellular adhesion molecule-1, vascular cell adhesion molecule-1 and E-selectin were reported to exist in relation to coronary slow flow.[22] Red cell distribution width and serum uric acid levels, among other inflammatory markers, were studied in association with CSFP patients.[23,24] In endothelial dysfunction, abnormalities in inflammatory parameters can possibly be an indicator; both of these contribute to coronary slow flow. Shao-Ping et al.[25] demonstrated the existence of CSFP in relation to higher tortuosity and more distal branches in coronary arteries. Thus, certain anatomic properties of coronary arteries could be alleged to have an effect on disturbed coronary flow and endothelial damage, therefore resulting in CSFP.

Treatment for CSFP has not yet been clearly defined, but several drugs have proven to be effective at various levels. So far, pharmacological therapy for CSFP has not been executed on a large scale. Certain small studies have presented evidence regarding the drugs. In some studies, dipyridamole, mibefradil and nitroglycerine have been used for the treatment of CSFP.[19,26] Statins present certain benefits for CSFP patients, partially owing to their anti-inflammatory properties.[27] Nebivolol not only can improve endothelial function but also can remedy symptoms considerably; therefore, the drug enhances the

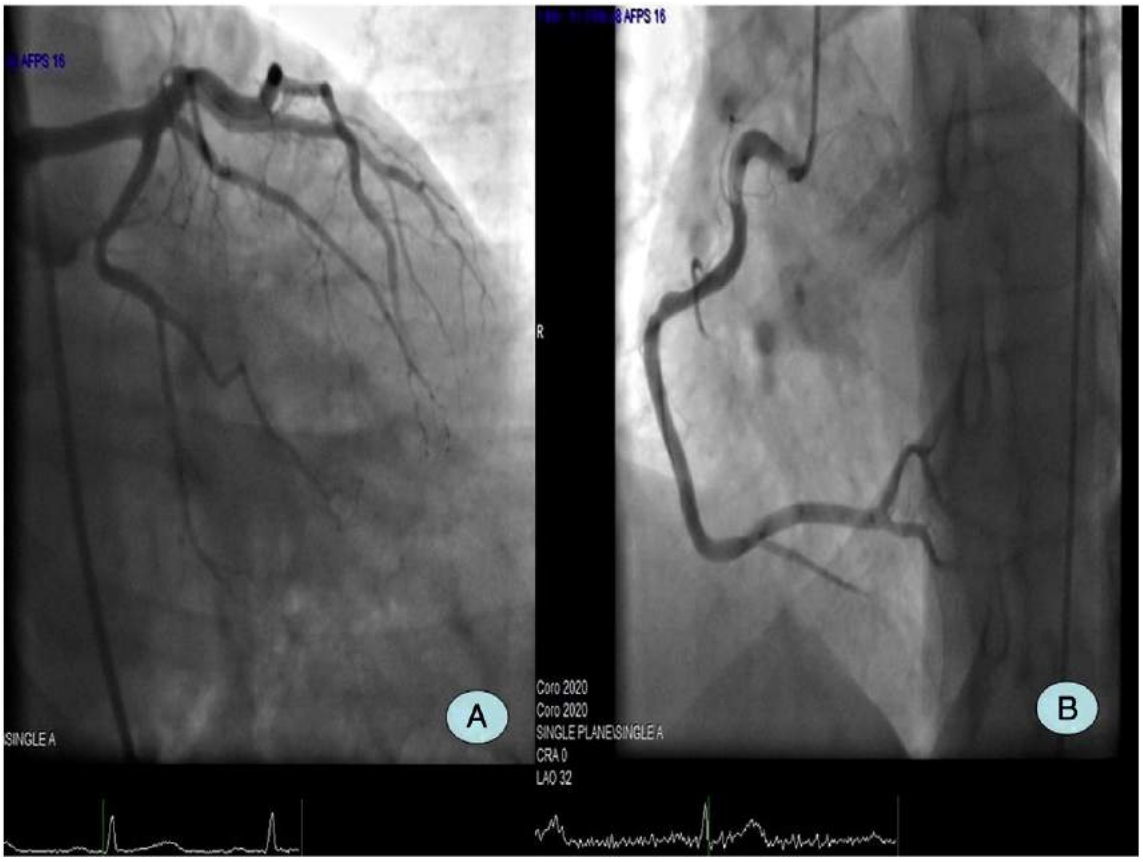

Fig. 2. Coronary angiogram reveals no significant stenosis, together with coronary slow flow in LAD (A) and RCA (B).

quality of life among CSFP patients.[28–30] Moreover, it might serve as a beta-receptor blocker and, due to increased nitric oxide release, result in endothelium-dependent vasodilatation.[28]

Conclusion

Slow flow might sound like a minor condition; however, it can result in such a fatal condition as STEMI. With proper treatment, on the other hand, symptoms are reduced. In conclusion, we recommend that CSFP be considered in the etiology of chest pain with ST elevation.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. Tambe AA, Demany MA, Zimmerman HA, et al. Angina pectoris and slow flow velocity of dye in coronary arteries. A new angiographic finding. *Am Heart J* 1972;**84**:66–71.
2. Gibson CM, Cannon CP, Daley WL, et al. TIMI frame count: a quantitative method of assessing coronary artery flow. *Circulation* 1996;**93**:879–888.
3. Chaudhry MA, Smith M, Hanna EB, Lazzara R. Diverse spectrum of presentation of coronary slow flow phenomenon: a concise review of the literature. *Cardiol Res Pract* 2012;**2012**:383181. http://dx.doi.org/10.1155/2012/383181.
4. Beltrame JF, Limaye SB, Horowitz JD. The coronary slow flow phenomenon — a new coronary microvascular disorder. *Cardiology* 2002;**97**:197–202.
5. Celik T, Iyisoy A, Kursaklioglu H, Yuksel C, Turhan H, Isik E. ST elevation during treadmill exercise test in a young patient with slow coronary flow: a case report and review of literature. *Int J Cardiol* 2006;**112**:e1–e4.
6. Tatli E, Yildirim T, Aktoz M. Does coronary slow flow phenomenon lead to myocardial ischemia? *Int J Cardiol* 2009;**131**:e101–e102.
7. Kapoor A, Goel PK, Gupta S. Slow coronary flow—a cause for angina with ST segment elevation and normal coronary arteries. A case report. *Int J Cardiol* 1998;**67**:257–261.
8. Sen T. Coronary slow flow phenomenon leads to ST elevation myocardial infarction. *Korean Circ J* 2013;**43**(3):196–198.
9. Saya S, Hennebry TA, Lozano P, et al. Coronary slow flow phenomenon and risk for sudden cardiac death due to ventricular arrhythmias: a case report and review of literature. *Clin Cardiol* 2008;**31**:352–355.
10. Amasyali B, Turhan H, Kose S, et al. Aborted sudden cardiac death in a 20-year-old man with slow coronary flow. *Int J Cardiol* 2005;**109**(3):427–429.
11. Acikel S, Bozkaya OA, Akdemir R. The relationship between intermittent left bundle-branch block and slow coronary flow in a patient presenting with acute coronary syndrome. *Blood Coagul Fibrinolysis* 2010;**21**(6):595–597.
12. Sunbul M, Erdogan O, Sari I. Asymptomatic ST segment elevation in the recovery phase of the exercise stress test due to slow coronary flow. *Postep Kardiol Inter* 2014;**10**(35):53–56 [1].
13. Altas Y, Kurtoglu E, Yaylak B, et al. The relationship between eosinophilia and slow coronary flow. *Ther Clin Risk Manag* 2015;**11**:1187–1191.
14. Mosseri M, Yarom R, Gotsman MS, et al. Histologic evidence for small-vessel coronary artery disease in patients with angina pectoris and patent large coronary arteries. *Circulation* 1986;**74**:964–972.
15. Yetkin E, Turhan H, Erbay AR, Aksoy Y, Senen K. Increased thrombolysis in myocardial infarction frame count in patients with myocardial infarction and normal coronary arteriogram: a possible link between slow coronary flow and myocardial infarction. *Atherosclerosis* 2005;**181**:193–199.
16. Mangieri E, Tanzilli G, De Vincentis G, et al. Slow coronary flow and stress myocardial perfusion imaging. Different patterns in acute patients. *J Cardiovasc Med (Hagerstown)* 2006;**7**:322–327.
17. Demirkol MO, Yaymaci B, Mutlu B. Dipyridamole myocardial perfusion single photon emission computed tomography in patients with slow coronary flow. *Coron Artery Dis* 2002;**13**:223–229.
18. Kurtoglu N, Akcay A, Dindar I. Usefulness of oral dipyridamole therapy for angiographic slow coronary artery flow. *Am J Cardiol* 2001;**87**:777–779.
19. Yilmaz H, Demir I, Uyar Z. Clinical and coronary angiographic characteristics of patients with coronary slow flow. *Acta Cardiol* 2008;**63**:579–584.
20. Cin VG, Pekdemir H, Camsar A, et al. Diffuse intimal thickening of coronary arteries in slow coronary flow. *Jpn Heart J* 2003;**44**:907–919.
21. Li JJ, Qin XW, Li ZC, et al. Increased plasma C-reactive protein and interleukin-6 concentrations in patients with slow coronary flow. *Clin Chim Acta* 2007;**385**:43–47.
22. Turhan H, Saydam GS, Erbay AR, et al. Increased plasma soluble adhesion molecules; ICAM-1, VCAM-1, and E-selectin levels in patients with slow coronary flow. *Int J Cardiol* 2006;**108**:224–230.
23. Kalay N, Aytekin M, Kaya MG, et al. The relationship between inflammation and slow coronary flow: increased red cell distribution width and serum uric acid levels. *Turk Kardiyol Dern Ars* 2011;**39**:463–468.
24. Yildiz A, Yilmaz R, Demirbag R, et al. Association of serum uric acid level and coronary blood flow. *Coron Artery Dis* 2007;**18**:607–613.

25. Niea Shao-Ping, Wanga Xiao, Genga Li-Li, et al. Anatomic properties of coronary arteries are correlated to the corrected thrombolysis in myocardial infarction frame count in the coronary slow flow phenomenon. *Coron Artery Dis* 2012 May;**23**(3): 174–180.
26. Beltrame JF, Turner SP, Leslie SL, et al. The angiographic and clinical benefits of mibefradil in the coronary slow flow phenomenon. *J Am Coll Cardiol* 2004;**44**:57–62.
27. Li JJ, Zheng X, Li J. Statins may be beneficial for patients with slow coronary flow syndrome due to its antiinflammatory property. *Med Hypotheses* 2007;**69**:333–337.
28. Gunes Y, Tuncer M, Guntekin U, et al. Regional functions of the left ventricle in patients with coronary slow flow and the effects of nebivolol. *Ther Adv Cardiovasc Dis* 2009;**3**:441–446.
29. Fragasso G. Nebivolol in patients with coronary slow flow: the right drug for the right case? *Anadolu Kardiyol Derg* 2009;**9**:296–297.
30. Albayrak S, Ordu S, Yuksel H, et al. Efficacy of nebivolol on flow-mediated dilation in patients with slow coronary flow. *Int Heart J* 2009;**50**:545–553.

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