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Review

Long-term clinical results of saphenous vein bypass graft lesions treated with bare-metal stents and drug eluting stents

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ABSTRACT

Objective: To evaluate the long-term clinical results of bare stents (BMS) and drug eluting stents (DES) for the treatment of saphenous vein graft (SVG) lesions, to examine the efficacy and safety of both and to determine the parameters that have predictive value for long term clinical results.
Methods: Between 2009 and 2011, the long-term results were examined and compared respectively in 107 patients with SVG lesions on whom revascularization was applied using BMS or DES.
Results: The long-term results of BMS (n: 56) and DES groups (n: 51) were compared (average follow-up time for both groups: 22.1 ± 10.7 months). At one-year follow-up, the BMS group had higher target vessel revascularization (TVR) (33.9% vs 11.8%, p = .01) and major adverse cardiac events (MACE) (35.7% vs 15.7%, p: .02) compared to the DES group. There were no significant differences in myocardial infarction (MI) and mortality rates between the two groups. At a median follow-up of 2 years, there were no significant differences in composite MACE, TVR, MI and mortality rates between the two groups. Event free survival at 1 and 2 years was 84.3%, 66.7% vs 64.3%, 50% for DES and BMS group, respectively.
Conclusion: At one year follow-up, patients receiving DES had significantly better clinical outcomes than their BMS counterparts. However, long term outcomes among the two groups were similar.

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Introduction

Since the first coronary artery bypass operation by Favaloro in 1968, the improvements in revascularization have been continuing to accelerate.[1] However, the fragile structure, degeneration and occlusion that were seen in SVG have emerged as long-term problems. The development of stenosis or occlusion in SVG in 15% during the first year and 50% in 10 years has revealed that new techniques must be used.[2] The initial implementation of balloon angioplasty has been superseded by direct stent implantation due to low success, high restenosis rates and increases in MACE in the former. In 1998, Figulla et al. obtained better results in clinical applications via introducing direct stent technique without angioplasty.[3] Advances in stent technology paved the way for

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the use of DES as well as BMS in SVG lesions. The studies revealed conflicting results on BMS and DES application results in saphenous vein grafts. In the short term, SVG revascularization with BMS resulted in high rates of restenosis.[4,5] Although short term restenosis rates were found significantly lower with DES, the studies reported that this benefit disappeared in the long term and the mortality rate increased.[6–9] Although there is consensus on short term results, more comprehensive research is needed on long term results. In this study, we compare the long term results of BMS and DES implantations in saphenous vein grafts, and determine the parameters that have predictive value for MACE.

Methods

Between 2009 and 2011, 107 patients who received percutaneous coronary intervention (PCI) due to SVG lesions were retrospectively analyzed. The investigations were performed by two experienced cardiologists. Patient data was obtained from medical records, telephone contact and outpatient examinations. After a detailed history had been taken, their physical examinations were performed. All patients, who underwent at least one exercise stress test and myocardial perfusion scintigraphy (99mTc-MIBI) for the investigation of ischemia were included in the study. Patients who had signs of ischemia received coronary angiography as the standard approach. The patients were administered 325 mg/day of aspirin, 600 mg clopidogrel, if the patients are not on it, and 100 units/kg of heparin administered before the procedure to reach the target clotting time (ACT) of 250–300 s. Biochemical analysis of blood samples is held before the procedure. 56 applied BMS and 51 patients applied DES. The dates when the first and the last applications done to the patients are recorded and their total monitoring times were calculated. All patients received clopidogrel and aspirin for 1 year and only aspirin after that.

Definitions

MACE was defined as a sum of cardiac death, myocardial infarction, target vessel revascularization (TVR). Target lesion revascularization (TLR); revascularization procedure for lesions that have more than 50% stenosis within the stent, 5 mm proximal or distal to the stent. Stent thromboses were evaluated according to the criteria defined by the Academic Research Consortium.[10] The diagnosis of myocardial infarction required 2 of the following: 1) prolonged (>30 min) chest pain; 2) a rise in creatine kinase levels more than twice the local upper normal value (with abnormal MB fraction); and 3) development of persistent ischemic electrocardiographic changes (with or without new pathological Q waves). Deaths of unknown etiology are considered to be cardiac deaths. Procedural success was considered to be <20% of residual stenosis and TIMI (Thrombolysis In Myocardial Infarction) grade III flow.[11]

Table 1
Baseline clinical characteristics.

Parameters	BMS group (n: 56)	DES group (n: 51)	p
Age (years)	63.1 ± 6.8	64.2 ± 8.1	0.747
Male, n (%)	44 (78.6)	36 (70.6)	0.379
Female, n (%)	12 (21.4)	15 (29.4)	
Hypertension, n (%)	38 (67.8)	31 (60.7)	0.874
Diabetes mellitus, n (%)	18 (32.1)	19 (37.2)	0.252
Smoking, n (%)	29 (63.0)	5 (55.6)	0.719
SVG age (years)	8.0 ± 6.3	7.5 ± 3.6	0.879
SVG vessels, n	3.4 ± 0.7	3.3 ± 1.3	0.478
Beta blocker, n (%)	42 (75)	40 (78.4)	0.885
Statin, n (%)	41 (73.2)	40 (78.4)	0.775
Hematologic parameters			
MPV (μm^3)	8.7 ± 9	8.9 ± 1.5	0.737
PDW (%)	16.3 ± 11.0	13.4 ± 1.2	0.06
NLR	2.7 ± 1.2	2.2 ± 0.6	0.323
RDW (%)	14.1 ± 1.8	12.9 ± 1.8	0.232
Platelet count (10^9/l)	237.3 ± 56.3	241.0 ± 65.8	0.637

SVG: saphenous vein graft, MPV: main platelet volume, PDV: platelet distribution volume, NLR: neutrophil lymphocyte ratio, RDW: red cell distribution wide[9]; BMS, bare metal stent; DES, drug eluting stent.

Table 2
Lesion and procedural characteristics.

Parameters	BMS group (n: 56)	DES group (n: 51)	p
Number of stents, n (%)	1.8 ± 0.9	1.7 ± 0.9	0.688
Diameter stenosis, %	86.0 ± 11	95.0 ± 0.3	0.548
Stent diameter, mm	3.2 ± 0.6	3.1 ± 0.5	0.791
Stent length, mm	17.5 ± 6.0	25.8 ± 11.9	**0.030**
Maximum balloon pressure (atm.)	13.3 ± 1.6	14.0 ± 4.2	0.718
No reflow, n (%)	4 (7.1)	3 (5.8)	0.771
Angiographic success, n (%)	54 (96.4)	50 (98)	0.876

BMS, bare metal stent; DES, drug eluting stent.
Bold values indicate significance at $p < 0.05$.

Statistics

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 used to identify the independent predictor of MACE. Event-free survival curves were generated by the Kaplan–Meier method and differences in survival were compared using log-rank test. All variables showing significance values less than 0.05. Two-tailed p values less than 0.05 were considered significant and the confidence interval was 95%. All statistical studies were carried out using the SPSS program (version 22.0; SPSS Inc., Chicago, Illinois, USA).

Results

Clinical and demographic characteristic of patients are shown in Table 1. In total of 107 patients (27 women, 80 men, mean age: 62.3 ± 7.3 years) were included in the study. There were similarities between these two groups in terms of demographic and clinical characteristics and medical treatment. There were no statistical differences between the groups in terms of age, gender, hypertension, diabetes, and smoking. Average age of SVG was 7.73 ± 4.6 years. There was no significant difference between the two groups in terms of bypass graft age and number of grafts.

Table 2 shows lesion and procedure characteristics. The number of stents implanted per patient was 1.65 ± 8.9, with an average diameter of 3.04 ± 0.5 mm. Stent length in the DES group was significantly longer than that in the BMS group (25. 8 ± 11.9 mm vs. 17.5 ± 6.0 mm, $p = .03$). Maximum inflation pressure (pressure: 13.8 ± 2.0 atm.)

Table 3
Clinical outcomes at long term follow-up.

Parameters	BMS group (n: 56)	DES group (n: 51)	p
One year outcomes			
Composite MACE, n (%)	20 (35.7)	8 (15.7)	0.02
TVR, n (%)	19 (33.9)	6 (11.8)	0.01
Myocardial infarction, n (%)	5 (9.1)	4 (8)	1.00
Mortality, n (%)	2 (3.6)	1 (2)	1.00
Total follow up time outcomes			
Composite MACE, n (%)	30 (53.6)	18 (35.3)	0.08
TVR, n (%)	22 (39.3)	14 (27.5)	0.223
Myocardial infarction, n (%)	9 (16.1)	7 (813.7)	0.791
Mortality, n (%)	3 (5.4)	2 (3.9)	1.00

MACE: major adverse cardiac event, TVR: target vessel revascularization; BMS, bare metal stent; DES, drug eluting stent.

Table 4
Multivariate predictors of composite MACE.

	OR	95% CI	p
Hypertension	0.072	0.023–0.225	0.00
Diabetes mellitus	0.346	0.151–790	0.01
SVG age	0.983	0.852–1.13	0.809
Stent length (mm)	1.04	0.993–1.14	0.08
Stent çapı (mm)	1.45	0.666–0.317	0.34
Treatment with BM or DES	2.90	1.15–7.28	0.02

CI, confidence interval; OR, odds ratio; BMS, bare metal stent; DES, drug eluting stent; SVG, saphenous vein graft.

was applied. Stent implantation was successfully placed in 104 (97.1%) cases. The intervention was unsuccessful in 3 (2.8%) cases.

Table 3 shows clinical outcomes at one year and long term follow-up. At one year follow-up mortality and risk of MI did not differ between DES and BMS group (9.1% vs. 8%, p = 1). However, TVR and MACE rates were significantly higher in the BMS group. Rates of TVR and MACE were 33.9% vs. 11.8% (p = .01) and 35.7% vs. 15.7%, (p = .02) for the BMS and DES groups, respectively. Rates of two year (follow-up time: 22.1 ± 10.7 months) MI, mortality, TVR and composite MACE were not different between DES and BMS patients.

One and two year TVR rates in DES group were 11.8% and 27.5% respectively.

Table 4 shows multivariate predictors of Composite MACE. Hypertension (OR: 0.072; 95% CI: 0.023–0.225; p = .000), diabetes mellitus (OR: 0.346; 95% CI: 0.151–0.790; p = .01) and type of stent (DES vs. BMS) (OR: 2.90; 95% CI: 1.15–7.28; p = .02) were independent predictors of composite MACE. Kaplan Meier survival curves for event free survival at 1 and 2 years was 84.3%, 66,7% vs 64.3%, 50% for DES and BMS group, respectively (Fig. 1).

Discussion

In saphenous vein grafts, early benefit of DES seen in the first year was not sustained with longer-term follow-up. We found that, DES, compared with BMS, reduced TVR in first year, but this advantage decreased over time such that at 2 years. At two-year follow-up, there was a 2.33 fold increase in TVR (11.8% vs. 27.5%) in DES treated patients. These results seem to be consistent with the results of research examining the late DES restenosis.

It is estimated that, at least 50% of SVG lesions will develop stenosis or occlusion within 10 years of implantation.[2] Due to the high morbidity and mortality of reoperations, percutaneous intervention and revascularization are recommended in such cases. SVG lesions typically have a low fibrotic component and a fragile structure rich in necrotic debris, cholesterol and foam cell. For this reason, stent therapy is emerging as a more affordable option rather than balloon angioplasty.[3]

The process started with BMS initially, and then evolved with DES. In previous studies made with BMS and DES, conflicting results were found. While no differences between BMS and DES were found in some of the studies, in others DES were found superior.[6,12] In the first 6 months, better results were obtained with DES, however this superiority disappeared after 2 years, and in some studies the results even turned in favor of BMS.[7,8]

In the SOS (Stenting Saphenous Vein Graft) study by Brilakis et al., the one-year rates of TLR and TVL in the BMS and DES groups, were found to be 28% and 5%, 31% and 15% respectively in favor of DES. Mortality was found to be similar in both groups.[6] In the RRİSC (Reduction of Restenosis with Cypher Sirolimus eluting stent in Saphenous Vein Grafts) study, the rates of TLR and TVR in 6 months were 32.6% and 13.6% and 27% and 5.3% in BMS and DES groups respectively in favor of DES. In the late period (32 months later), mortality was found to be higher in the DES group.[7,8] In the STENT (Strategic Transcatheter Evaluation of New Therapies) study, 343 patients were treated with BMS, and 785 with DES, and the results showed that mortality was lower in the DES group in the second year.[9]

According to the results of meta analysis, in those to whom DES was applied, TVR and TLR were found to be lower. Although the short term consequences were in favor of DES, late "catch-up phenomenon" occurring in the long term may result with stent thrombosis and myocardial infarction.[13–16] The reason that the benefit provided in the early period gradually disappears is related to delayed endothelialization, restenosis, and stent thrombosis.[17,18] The frequency of BMS restenosis has been reported as 20–40%, and is accompanied by early elastic recoil and late vascular remodeling and neointimal hyperplasia.[5,19] In those to whom

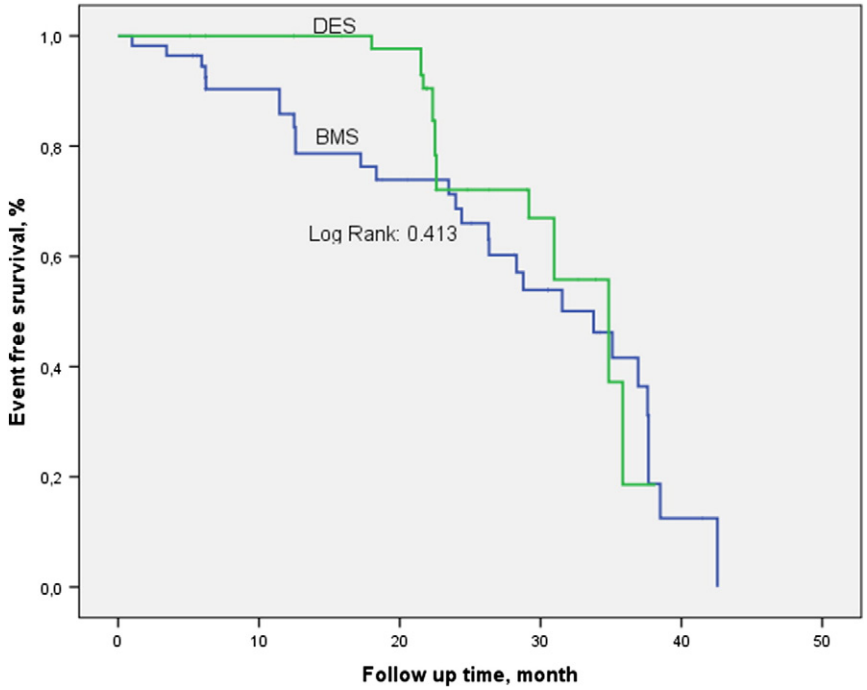

Fig. 1. Kaplan Meier survival curves for freedom from major adverse cardiac events (MACE) Log Rank: 0.413. DES, drug eluting stent; BMS, bare metal stent.

BMS is applied, maximal neointimal thickening occurs in the first 6 months.[20] It is felt that DES shows its effect by preventing neointimal thickening in the early stages.

Restenosis is an independent predictor of MACE and long term mortality after PCI. Other predictors of MACE are age of SVG, stent type, diabetes mellitus, and reference vessel diameter <3.5 mm.[9,21–23] Our study found that diabetes mellitus, stent type and hypertension were independent predictors of composite MACE. The rate of late restenosis related TVR after one year tended to be higher in DES patients, resulting in a comparable rate of composite MACE. These findings are consistent with other studies.

Study limitations

This study has several limitations. The retrospective nature of the study, being a single center study, the small number of patients, and lack of routine angiographic follow-up are the main limitations of the study. Also loss to follow up due to time lapse is another limitation of the study.

Conclusion

Treatment of SVG disease remains a challenge. Although DES were used in longer lesions, the rates of TVR and MACE at the 1-year evaluation were significantly lower in the DES group compared to the BMS group. However, the initial benefits of DES seen in the first year were not sustained at 2-year follow-up because of recurrent revascularizations. At-year follow-up, there were no significant differences in composite MACE, MI, mortality and TVR rates. Hypertension, diabetes mellitus, and type of stent (DES vs. BMS) were identified as independent predictors of composite MACE.

Conflict of interest

The authors have no conflict of interests to declare.

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Review

NOACs and routine coagulation assays. How to interpret?

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ABSTRACT

Since the non-vitamin K antagonist oral anticoagulants (NOACs) have been introduced to clinical practice, there is a conflict on coagulation tests, which have been used to monitor the anticoagulation effect of vitamin K antagonists (VKAs) and unfractionated heparin (UFH). NOACs have alternative modes of action. They react differently than VKAs and UFH and therefore conventional coagulation tests are not suitable to monitor their anticoagulant effect. The interactions between NOACs and routine coagulation tests are discussed in this review.

Contents

Introduction

Many anticoagulant agents are available for prevention and treatment of thromboembolic disorders such as the warfarin derivatives (vitamin K antagonists—VKAs), unfractionated heparin (UFH) and low molecular weight heparins (LMWHs).[1,2] In the last decade a new class of anticoagulant drugs has been developed for clinical use. The non-vitamin K antagonist oral anticoagulants (NOACs) are orally active and inhibit coagulation serine proteases selectively and specifically.[3] Unlike VKA and UFH,[4,5] the NOACs such as dabigatran (direct thrombin inhibitor), rivaroxaban, apixaban and edoxaban (all direct Factor Xa inhibitors) do not require routine coagulation monitoring because they have a rapid onset of action, their therapeutic window is wide and their pharmacokinetics and pharmacodynamics are predictable.[6,7] However in some clinical situations the need for assessment of the anticoagulant effect of these new agents might arise, which requires an understanding of the different mechanism of action of each agent and their implications when interpreting routine coagulation tests are necessary.[1] The most commonly used clot-based tests are prothrombin time (PT)/international normalized ratio (INR) and activated partial thromboplastin time (aPTT).[1] PT is the time in seconds for plasma to coagulate after addition of calcium and an activator of the extrinsic pathway of the coagulation cascade as thromboplastin.[8] PT is mostly used to monitor VKAs which inhibits factors II, VII and X.[9] Because of varying sensitivities of the available thromboplastin agents, PT is converted to INR through a mathematical formula that includes the International Sensitivity Index (ISI) of the thromboplastin reagent used. ISI is obtained from the manufacturer.[10] After the calculation, INR can be interpreted without regard to the thromboplastin reagent.[8] While aPTT is similar to PT, aPTT reflects the presence and activity of factors II, V, VIII, XII and fibrinogen.[8] Therefore, the test is used to measure the overall function of the intrinsic coagulation pathway.[1] However these tests do not react in the same way for NOACs and may be misinterpreted by non-expert clinicians.[1,3]

Dabigatran

The PT is relatively insensitive to dabigatran and the PT reagent sensitivity is extremely variable.[11,12] The INR/ISI calculation which is used for warfarin derivatives is not suitable for dabigatran. The contradiction between PT results is increased by point of care-derived INR results.[13,14]

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The aPTT is more sensitive to the anticoagulant effect of dabigatran and shows a curvilinear dose response with an increase at low concentrations and linearity at high concentration.[12,15] The aPTT results are influenced by coagulometers and reagents and need to be evaluated carefully. In the nationwide Belgian survey, Actin FSL is found the least sensitive among aPTT reagents. The other reagents (Actin FS, APTT-SP, STA-CK Prest, STA-Cephascreen, STA-PTT A, SynthASil and TriniCLOT aPTT HS) used in the survey show slightly higher sensitivities.[21] A normal aPTT can presumably exclude a therapeutic intensity of dabigatran but cannot exclude some degree of anticoagulation caused by dabigatran.[3] If the aPTT level at trough exceeds two times the upper limit of normal, this can be considered as a higher risk of bleeding.[16]

Rivaroxaban

Rivaroxaban plasma levels in healthy subjects correlate closely with the inhibition of Factor Xa activity and prolongation of PT and aPTT.[17,18] The PT is more sensitive for rivaroxaban. There is a linear concentration–response correlation but because of the high variability between PT reagents, up to three-fold difference between them can be observed.[3,19] The INR calculation increases the contradiction between PT results (nearly four-fold), because ISI are calculated for VKAs, and therefore INR results should not be used.[1,3,19] A normal PT level cannot exclude some degree of anticoagulation effect but a normal PT level achieved with most reagents excludes a therapeutic intensity of rivaroxaban.[3] With the thromboplastin reagents, neoplastin and neoplastin plus, the PT is influenced in a dose-dependent manner with a close correlation to plasma concentrations.[16] In the Belgian survey, where six different PT reagents were used, the reagents Innovin and Thrombel S were found least sensitive to rivaroxaban and Neoplastin R was the most sensitive PT reagent to rivaroxaban.[21] The sensitivity difference was not connected with the source of tissue factor and it may be related to the composition of the reagents.[21]

Apixaban

Apixaban prolongs PT in human plasma in vitro.[20] As with rivaroxaban, there is variability in the PT sensitivity between thromboplastin reagents and the INR calculation increases variability.[3,19] To measure the inhibition of Factor Xa activity gives a better sign of apixaban plasma concentration than a PT test.[19]

Apixaban has been shown to prolong the aPTT in a concentration dependent manner in vitro but the aPTT test is not sensitive to apixaban.[1,20]

Discussion

In clinical practice NOACs are prescribed at fixed doses and do not require routine coagulation monitoring, but their presence can affect routine coagulation tests especially in high-doses or if the blood sample is taken at the peak plasma concentration level and these may lead to interpret the test results improperly.[21] NOACs prolong the PT and aPTT in a concentration- and reagent-dependent manner. There is a significant difference in sensitivity of reagents. PT reagents are more influenced by rivaroxaban and apixaban and aPTT reagents are more influenced by dabigatran.[21] In monitoring VKA therapy, PT results are expressed as INR levels commonly in daily clinical practice. However for NOACs, converting PT to INR does not decrease but even increases the variability between reagents and cannot be applied because the conventional INR system was developed to monitor VKAs and correct the varying sensitivities of reagents for VKAs, not for NOACs.[21]

Conclusion

It is important for clinicians to appreciate the variable effects of individual NOACs on PT and aPTT reagents utilized to avoid erroneous interpretation of results.

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Short communication

A case of Kounis syndrome with anaphylactic shock secondary to penicillin G injection in a 32-year old woman

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ABSTRACT

Our case report describes a 32-year old woman who had myocardial infarction with anaphylactic shock secondary to penicillin injection. She had hypotension and chest pain after 1.2000.000 U penicillin G injection for criptic tonsillitis. Her ECG showed ST elevation on D1 and aVL derivations and ST depression and T wave inversion on D2,D3,aVF, V3–6 derivations. Her ECG abnormalities regressed after the chest pain resolved. Her serum troponin level was elevated to 5.2 ng/ml. She had no pathology on echocardiographic examination. She was given antiplatelet and anti-thrombotic treatment, monitorized and followed in intensive coronary care unit. No cardiac complications were observed in her follow-up. Her coronary angiography was completely normal. The hyperventilation test to induce coronary spasm was negative during the coronary angiography. The myocardial injury seen in this case may be due to hypotension, allergic reaction itself, the potential vasospasm at the time of the anaphylactic shock or to intravenous adrenalin administered. Such cases of Kounis syndrome with anaphylactic shock are rarely observed. Emergency physicians should be aware of Kounis syndrome when there is a young patient without any risk factors having chest pain after an allergic insult.

Introduction

Kounis syndrome is the coexistence of acute coronary syndromes with hypersensitivity reactions. There are many etiologies that have been reported including drugs, medical conditions, environmental exposure and stent implantation. There are 3 types of Kounis syndrome. Type 1 Kounis syndrome is the one seen in patients with normal coronary arteries. Type 2 is seen in patients with pre-existing atheromatous disease. Type 3 is stent thrombosis due to hypersensitivity reactions to the components of the stent. The exact mechanism of this syndrome is not clearly identified. There are some possible mechanisms. The most accepted mechanism is mast cell degranulation after the allergic insult. Chemical mediators released from mast cells are thought to induce coronary artery spasm or to promote platelet aggregation.[1–4]

Case report

We present a 32-year old woman who had no history of cardiovascular disease or any risk factor. She had migraine for 10 years. She was admitted to emergency department because of squeezing chest pain after intramuscular injection of 1.200.000 U penicillin G for criptic tonsillitis. Her arterial blood pressure was 70/50 mm Hg. Her ECG showed prominent ST depression and T wave inversion on leads DII, DIII, AVF, V3–6, and 1 mm ST elevation on leads D1 and aVL (Fig. 1A). She was given 0.5 mg intravenous adrenalin. She chewed 300 mg of non-enteric acetylsalicylic acid. Her chest pain resolved in 5 min. And a second ECG afterwards showed only T wave inversion on leads D1 and aVL (Fig. 1B). Her blood biochemistry revealed an elevated serum troponin level of 5.2 ng/ml (reference range: 0–0.3). Echocardiography was normal. We admitted the patient to the intensive coronary care unit with a diagnosis of Kounis syndrome. She was started enoxaparine 6000 IU subcutaneously twice a day, clopidogrel 75 mg once a day orally after 300 mg loading dose and atorvastatin 80 mg once a day orally. She had already taken acetylsalicylic acid. The next day she had coronary angiography. The coronary angiography showed completely normal coronary arteries (Fig. 2). A hyperventilation test was also performed during the procedure to provoke coronary spasm. But it was negative. The patient was discharged from the hospital with a prescription of acetylsalicylic acid 100 mg per day.

Discussion

Our case is a sample of Type 1 Kounis syndrome. There are not many cases of Kounis syndrome in the literature presenting after penicillin administration and accompanied by anaphylactic shock. One of these

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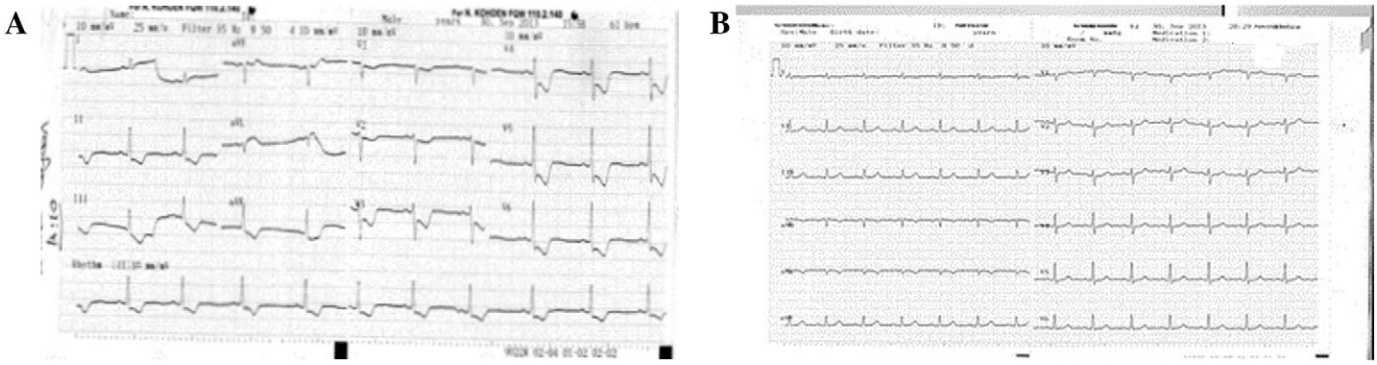

Fig. 1. The ECG of the patient when she had chest pain (A) and when the chest pain resolved (B).

cases was reported by Gikas et al., who presented a Kounis syndrome with anaphylactic shock in a young man induced by oral amoxycillin intake.[5] Another case was a sample of Kounis syndrome with a previous history of significant coronary artery disease.[6] The third case was presented by Viana-Tejedor et al. and was diagnosed to have type 2 Kounis syndrome.[7] Tok et al. reported a case similar to ours.[8] In this case a 52-year old man developed ST elevation on leads D1 and aVL after intramuscular penicillin injection due to criptic tonsillitis. But in this case the patient did not experience anaphylactic shock. A more recent case is reported by Bilgin et al. secondary to clarithromycin use in a 36 year-old woman.[9] Kounis syndrome may also be seen in very young adolescent patients. Ilhan et al. presented a case in a 16 year-old child.[10] Our patient differs from these cases. Because our patient did not have an allergy or cardiovascular disease history before. She experienced a very serious anaphylactic shock after penicillin injection which resulted in acute myocardial infarction.

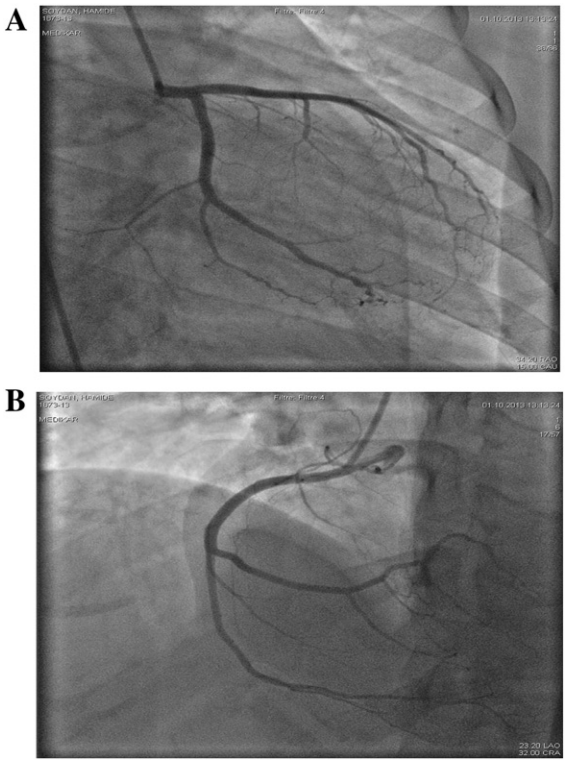

Fig. 2. A. The left coronary angiogram of the patient. Left anterior descending and circumflex arteries showed no pathology. B. The right coronary angiogram of the patient. Right coronary artery of the patient showed no atherosclerosis.

There can be some possible mechanisms to explain the myocardial infarction in our case. The anaphylactic shock itself due to hypotension may be the cause. Because after saline infusion, when the blood pressure was normalized the chest pain regressed and ECG findings resolved. Although we could not demonstrate coronary spasm with hyperventilation method during the coronary angiography, at the time of anaphylactic shock acute coronary spasm might have contributed to the process. The allergic process itself may also be another explanation.

In conclusion, we should keep in mind the possibility of Kounis syndrome when there is a young patient without any risk factors having chest pain after an allergic insult.

Note: The patient and her husband gave informed consent for this case report. And this article fulfilled the principles of the ethical guidelines of the 1975 Declaration of Helsinki.

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Short communication

Acute myocardial infarction with single coronary artery

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Introduction

Single coronary artery is very rare among the different variations of anomalous coronary patterns. In acute myocardial infarction settings, primary angioplasty of these arteries can be challenging and is associated with the risk of complications. We report a 78-year-old female patient who presented acute high lateral wall infarction with aberrant circumflex artery arising from the right coronary artery treated by stent implantation.

Case

A 78-year-old female patient was admitted into our emergency department with a compliant feeling of pressure in her chest for approximately two hours. Her medical history revealed dyspnea and exertional angina for a couple of months with increasing severity.

On physical examination, blood pressure was 80/50 mm Hg and heart rate was 68 bpm. There was apical 2/6 mid-systolic murmur. Bilateral lung sounds and other findings were normal. On electrocardiogram sinus rhythm with 2–4 mm of ST elevation in lead I and aVL, 2–5 mm of ST depression on leads II, III, aVF and V1–V6 were evident (Fig. 1). The diagnosis was high lateral myocardial infarction. The results of biochemical laboratory tests are summarized in Table 1.

Tigacrelor 180 mg, acetylsalicylic acid 300 mg and heparin 100 IU/Kg IV bolus were administered and the patient was urgently referred to the cardiac catheterization laboratory. A 6 French Left Judkins diagnostic catheter failed to engage the left main coronary ostium at the level of left sinus of Valsalva. Contrast injection with right Amplatz catheter showed a single coronary originated from the right coronary cusp. The left anterior descending (LAD) and circumflex (Cx) artery originated from the right coronary ostium separately. LAD was rudimentary and subtotal discrete lesion was present on the proximal segment of circumflex artery (Fig. 2). After crossing the lesion with 0.014-inch guide wire, a 2.5 × 10 mm balloon was inflated at 8 atm. Then, a 3.0 × 16 mm Taxus-Liberte Drug Eluting Stent (DES) was implanted at 12 atm. Immediately after stent implantation, localized small linear dissections were observed on the proximal edge of the stented segment. A Taxus Liberte 3.0 × 15 mm DES were implanted on the dissected segment and successfully sealed (Fig. 3). Following the procedure, the patient was transferred to Coronary care unit (CCU). Angina and hemodynamic symptoms were resolved. Blood test results were in the normal range except troponin, which was 0.043 ng/mL on admission (Fig. 1). Echocardiogram revealed posterolateral hypokinesis with left ejection fraction of 45%. Follow-up cardiac enzymes showed early peaking values (Table 2). The patient was discharged on day 4.

Discussion

Single coronary artery is a term that is used to describe left and right coronary arteries originated from one coronary ostium. It was first angiographically described in 1967 in two patients.[1,2] Though conus artery can originate from separate ostium, it is ignored, and the term "single coronary artery" has been used. Prevalence is 0.02–0.04% and is nearly equal for the right and left coronary arteries.[1–4] Based on the Lipton classification, single coronary artery types can be grouped into three classes. Class I contains coronaries with normal course. For example, the right coronary (R-I) is at the normal location and continues as circumflex artery after the posterolateral branch and left anterior descending after the posterior descending branch. At the left-sided single coronary (L-II), left anterior descending is normal and the circumflex artery terminally continues as the right coronary. In Class II, the single left or right coronary artery gives rise to a truncus that has a transverse course on the base of the heart to yield contralateral coronary arteries. This can be subclassified as A, B, or P according to the course of truncus.

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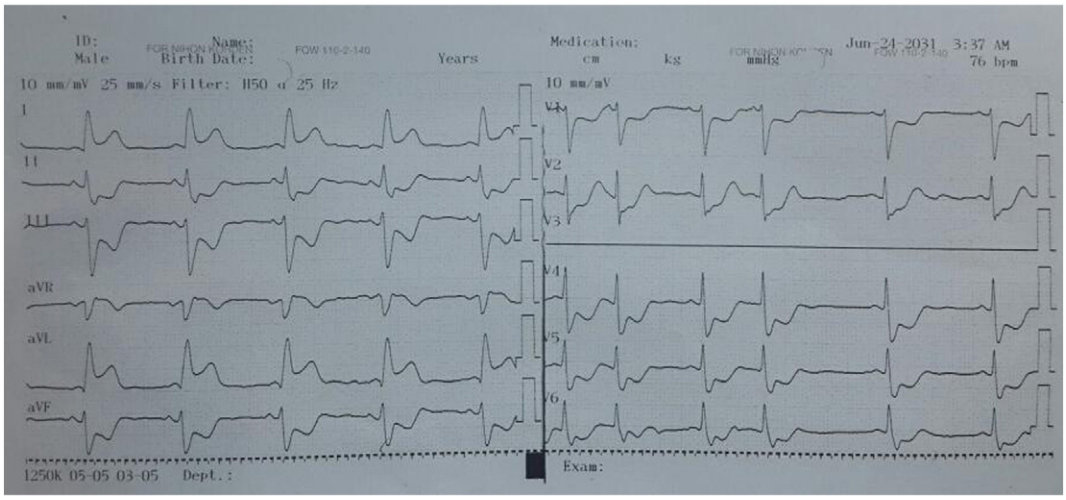

Fig. 1. ECG on admission.

For example, if the truncus originating from the right coronary truncus (R-II) has a course on the anterior of the pulmonary artery or the anterior of the right ventricle conus, it subclassifies as R-II A. If it has a course between the main pulmonary artery and the aorta, it subclassifies as R-II-B. Finally, if it continues posterior of the aortic root, it subclassifies as R-II-P. A similar subclassification scheme is available for the left single coronary artery (L-II-A, L-II-B, and L-II-P). In Class III, the single coronary artery originates from the right, LAD, and Cx separately originates from the right coronary contrary to the common truncus, similar to Class II. Cx takes a retroaortic course to the left atrioventricular sulcus. LAD courses between the aorta and the pulmonary artery and is directed to the left interventricular sulcus. This variation is known as Type R-III. Our case was consistent with R-III. Another classification of single coronary arteries based on origin and course has been described.[1]

Exercise-induced acute myocardial infarction and sudden deaths have been described in cases in which the right coronary or left main coronary artery lay between the main pulmonary artery and the aorta.[5] The cause is attributed to jamming of the coronary artery between two major arteries.[5,6] In addition to this phenomenon, the slit-like ostium of the coronary artery could be a reason for sudden death from ischemic causes.[5] Coronary anomaly can be present with subaortic or subpulmonary stenosis, ventricular septal defect, coronary artery fistula or transposition of the great arteries and tetralogy of Fallot.[7–14] Horan PG et al.[15] have also described a familial single coronary artery case.

Table 1
The results of biochemical laboratory tests.

Parameter	Result	Unit	Reference
Glucose	88	mg/dL	74 – 106
Urea	36	mg/dL	17 – 49
Creatinine	0.8	Mg/dL	0.5 – 0.9
AST	19	U/L	0–32
ALT	11	U/L	17–49
LDH	209	U/L	135–214
Sodium	141	mmol/L	136–145
Potassium	4.11	mmol/L	3.5–5.1
Chloride	100.7	mmol/L	98–107
CK	94	U/L	20–170
CK-MB	26	U/L	<25
Troponin T	0.043	ng/mL	<0.014
WBC	9.452	10 ^ 3/mm^3	3.9–11.7
RBC	5.21	10 ^ 3/mm^3	3.85–5.16
Hb	15.1	gr/dL	12–15
Hct	46.3	RU	34.8–45
PLT	220	10 ^ 3/mm^3	130–400

Blood tests on admission.

Angioplasty of a case with single coronary artery includes risks due to osteal obstruction of catheters with large diameters and can result in chest pain, dyspnea, dizziness, hypotension and hemodynamic compromise. The medical team should be ready for any potential complication and they should be aware that small obstructions can cause ischemia.

In our case, LAD and Cx were originated from the RCA and LAD was rudimentary. Some cases in the literature have described patients with single coronary with rudimentary LAD who had undergone PCI.[16–18] PCI of R-III type single coronary artery patients who had a culprit lesion of circumflex artery have also been reported.[17,19] Some single coronary artery patients who have acute coronary syndrome had successfully received angioplasty via the radial approach.[20–22] Additionally, transcatheter aortic valve implantation with Sapien XT and CoreValve have been performed on two patients with SCA.[23]

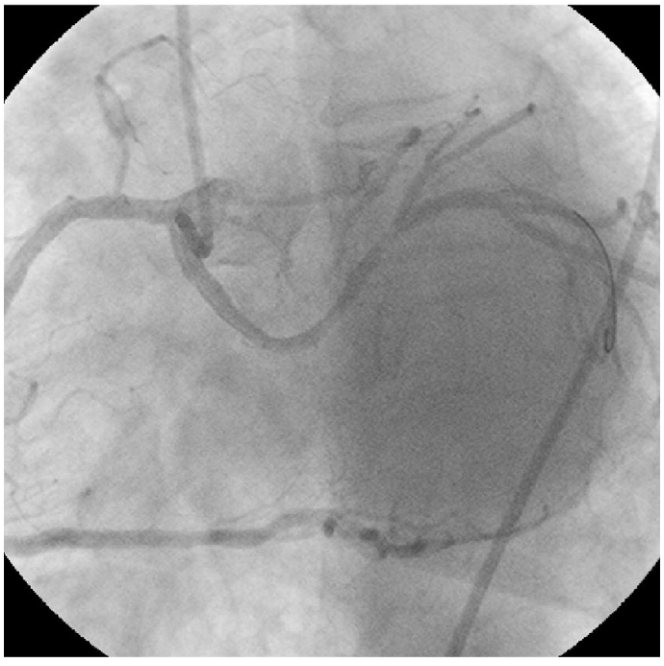

Fig. 2. Culprit lesion was on proximal Cx.

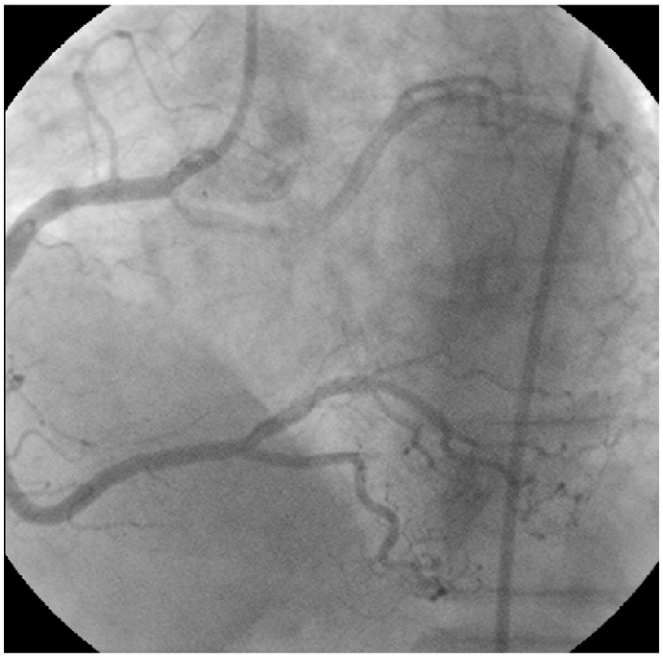

Fig. 3. Angiographic result were optimal after PCI of aberrant Cx.

Table 2
Follow-up cardiac enzyme values showed early peaking values.

Parameter	On admission	6th hour	12th hour
CK (U/L)	94	1871	1832
CK-MB (U/L)	26	319	275
Troponin (ng/mL)	0.043	9.13	5.43

Conclusion

The case described above is a rare instance of a patient with SCA (Lipton R-III group) with STEMI undergoing PCI to the Cx. Percutaneous coronary intervention of a single coronary artery can be difficult and susceptible to complications. Because of vessel compromise during angioplasty, this will have an impact on all three territories.[17] Coronary ischemia could cause hemodynamic compromise. Operator should be aware of the potential risk of complications and the limitations of the procedure.

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Short communication

Triple leaflet perforation due to endocarditis in aortic valve complicated by pneumonia and exacerbation of chronic obstructive pulmonary disease

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ABSTRACT

Valve perforation complicating infective endocarditis has been for decades a bad sign leading to severe valve destruction, intractable heart failure and even death if surgical therapy is not administered in time. Here we present a 57 years old male patient inadvertently diagnosed with pneumonia and chronic obstructive pulmonary disease exacerbation in another hospital. After 20 days of broad spectrum antibiotics and bronchodilator therapy no improvement was achieved. During examination a severe aortic regurgitation was recognized. Immediately after, patient was transferred to our hospital for aortic valve surgery evaluation. Transthorasic echocardiography (TTE) showed a severe aortic regurgitation and vegetation like echogenicity over the noncoronary leaflet. An aortic valve replacement surgical therapy was decided. During the aortic valve excision underneath the vegetations, multiple small perforations in all the three leaflets were noticed. The destructed valve was excised and a mechanical aortic prosthesis (St Jude No: 23, MN, USA) was successfully replaced. After 14 days of treatment patient was healthily discharged.

Introduction

Valve perforation complicating infective endocarditis has been for decades a bad sign leading to severe valve destruction, intractable heart failure and even death if surgical therapy is not administered in time. Here we present a 57 years old male patient inadvertently diagnosed with pneumonia and chronic obstructive pulmonary disease exacerbation in another hospital. After 20 days of broad spectrum antibiotics and bronchodilator therapy no improvement was achieved. During examination a severe aortic regurgitation was recognized. Immediately after, patient was transferred to our hospital for aortic valve surgery evaluation. Transthorasic echocardiography (TTE) showed a severe aortic regurgitation and vegetation like echogenicity over the noncoronary leaflet. An aortic valve replacement surgical therapy was decided. During the aortic valve excision underneath the vegetations, multiple small perforations in all the three leaflets were noticed. The destructed valve was excised and a mechanical aortic prosthesis (St Jude No: 23, MN, USA) was successfully replaced. After 14 days of treatment patient was healthily discharged.

Case report

A 57 year old male patient presented at our hospital with palourness and shortness of breath. He was referred form another center where he had been treated for about 20 days. He had had a sudden fever (38,5 °C) and shortness of breath on admission. He was diagnosed with exacerbation of chronic obstructive pulmonary disease (COPD) and pneumonia. Although an empirical broad spectrum of antibiotics (Ampicillin/Sulbactam 4 gr/day IV + Klarithromycin 500 mg/day IV) and bronchodilator treatment was administered he had not seen any relief of symptoms. On admission he looked pale, dyspneic and not willing to lye supine. He had no medical history except for a long intense smoking habit. His temperature was 37,5 °C. His electrocardiography showed a sinus tachycardia with a heart rate of 110/min. His blood pressure was measured as 110/50 mm Hg. On lung auscultation broad fine rales and rhonchus were noticed. His heart auscultation showed a 3/6 diastolic murmur on the right side of the sternum at the 2-nd intercostal space. TTE showed a severe aortic regurgitation and vegetation like echogenicity over the noncoronary leaflet. A mild tricuspid regurgitation and a systolic pulmonary artery pressure of 60 mm Hg were found. The ejection fraction was estimated as 50%. An unrecognized infective endocarditis (İE) was found to be the cause of decompansated heart failure. Loop diuretic and bronchodilator regimen with proper oxygen supply were immediately administered. No growth from

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blood and urine cultures were found as a long-term antibiotic treatment was previously applied. According to the 2009 European Society of Cardiology Infective Endocarditis Guideline we had a class 1 B indication for urgent surgery of severe aortic regurgitation with pulmonary hypertension and persisting heart failure. As the patient stabilized a preoperative coronary angiography was performed. Noncritic coronary lesions were found. Thereafter an open heart surgery treatment for aortic valve endocarditis was decided. During native aortic valve excision multiple small vegetations were cleaned off (Fig. 1). Underneath them multiple perforations involving all of the three leaflets of the aortic valve were noticed (Fig. 2). The destructed valve was excised and a mechanical aortic prosthesis (St Jude No: 23, MN, USA) was successfully replaced. After 14 days of treatment with bronchodilator and broad spectrum antibiotics (Ampicillin/Sulbactam 12 gr/day IV + Gentamycin 240 mg/day IV) patient was discharged in a healthy condition.

Discussion

This case illustrates what appears to be an important complication of infective endocarditis, namely, perforation or destruction of the aortic valve leaflets. As we look at the literature valve perforation is found to be a common complication of left side native valve infective endocarditis (LNVIE) firstly described in autopsy studies [1–3]. In a prospective echocardiographic study the frequency of LNVIE valve perforation was found 34% [5]. Several studies report that valve perforation in İE is usually associated with valve destruction, valve regurgitation, progressive heart failure and a high rate in hospital mortality [3–5]. K. Bachour et al. showed that in LNVIE mitral valve perforation was more common than aortic valve perforation [6]. However, perforation of the aortic leaflets, rather than the mitral cusps was found to correlate to a worse prognosis requiring early surgical treatment [5]. Interestingly we noticed multiple perforations in different sizes involving all of the three leaflets of the aortic valve. Triple aortic valve perforation being a rare finding in infective aortic valve endocarditis, was a remarkable sign of an advanced and intractable infection that already had destructed the aortic valve apparatus leading to severe acute aortic regurgitation and heart failure. As in our patient severe acute aortic regurgitation is seen to be more life threatening than severe acute mitral regurgitation probably as a result of the left ventricle being less compliant than the left atrium and the left ventricle end diastolic pressure being higher in acute aortic regurgitation requiring early surgical treatment [7,8]. In addition, late diagnoses and empirical ineffective, perhaps insufficient dosages of antibiotics due to pneumonia could have attributed to progression of the aortic valve destruction, thus leading to multiple valve perforations, aortic regurgitation and acute heart failure. This finding underscores the rare course of infection leading to severe aortic regurgitation by multiple perforations in all the three leaflets rather than a perivalvular abscess formation, rupture or valve destruction suggesting the need for prompt surgical evaluation before clinical deterioration.

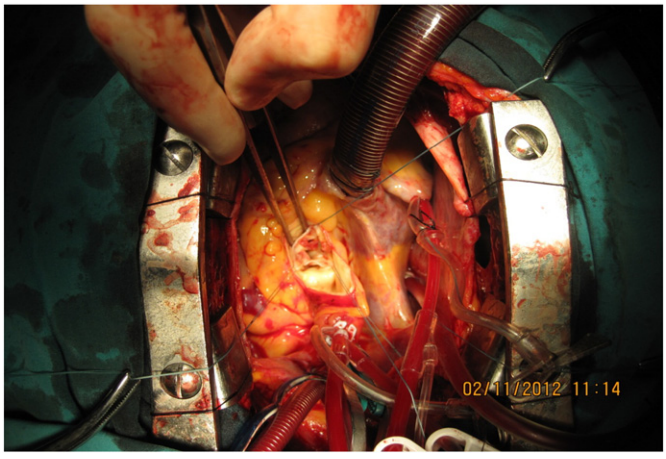

Fig. 1. Aortic valve endocarditis with multiple small vegetations.

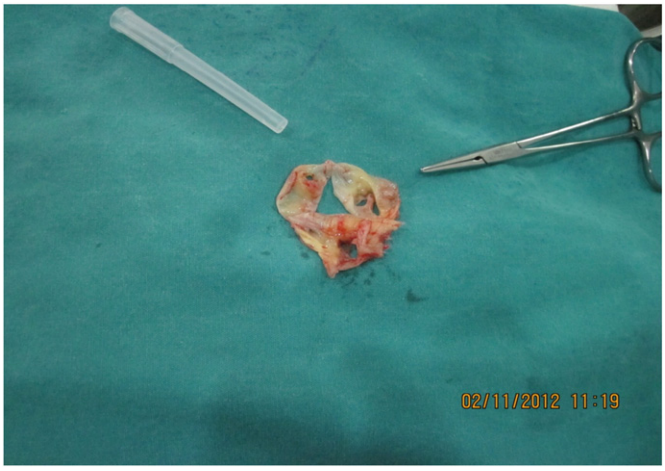

Fig. 2. The excised native aortic valve with multiple perforations in all the three cusps (Surgical clamp indicates the right coronary cusp; plastic clamp indicates the left coronary cusp).

Conclusion

Infective endocarditis is a life threatening disease that can present in different clinical scenarios. Our findings show a rare course of infection over the aortic valve leading to valvular destruction and progressive heart failure and a worse outcome. Aortic valve perforation must be regarded as a bad prognostic sign that emergent surgical therapy be considered as soon as possible before clinical deterioration ensues.

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Short communication

Bovine aortic arch and idiopathic pulmonary artery aneurysm associated with bronchial compression

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ABSTRACT

The left common carotid artery originating from the brachiocephalic trunk is termed the bovine aortic arch. Although it is the third most-common normal variant found in 9% humans, the origin of this term remains unclear. Until now, It has not been reported in the literature bovine aortic arch togetherness with pulmonary aneurysm and bronchial compression. Herein, we present a case with bovine aorta arch and pulmonary artery aneurysm associated with bronchial compression, which is incidentally detected by X-ray film. A 56-year-old Caucasian female admitted to the cardiology clinic with complaint of chest pain. Physical examination was unremarkable. Blood biochemistry values and cardiac markers were in normal range. Chest radiography revealed a widened mediastinum and prominent pulmonary conus with no active pulmonary disease. A subsequent transthoracic echocardiography revealed left ventricular hypertrophy, left atrial enlargement (diameter: 41 mm), mild mitral and tricuspid valve insufficiency, dilatation of main pulmonary artery (parasternal short-axis view diameter: 33 mm), normal pulmonary artery pressure and normal left ventricular systolic function. Computed tomography revealed bovine aortic arch associated with pulmonary artery aneurysm (diameter: 53 mm). And left main bronch of trachea was critically squeezed by aortic arch. Aortic and pulmonary vascular anomalies should be considered in patients with chest pain. And, identification with imaging modalities is important for prevention of chronic and irreversible complications.

Introduction

The left common carotid artery which originates from the brachiocephalic trunk (BT) is called as the bovine aortic arch (BAA).[1] Although it is the third most-common normal variant found 9% in human, the origin of this term remains unclear. Idiopathic pulmonary artery aneurysm (IPAA) is a rare condition, mostly arising from main pulmonary artery.[2] Even though specific prevalence of PAA is unknown, it has been reported in 1 out of every 14,000 autopsies.[3] Until now, no association of BAA with IPAA and bronchial compression has been reported in the literature. In this paper, we present a case with BAA and IPAA associated with bronchial compression, which is incidentally detected by a chest radiograph.

Case report

A 56-year-old Caucasian female admitted to the cardiology clinic with complaint of chest pain, which was located retrosternal, not induced with exercise. She had history of hypertension and diabetes mellitus. Physical examination was unremarkable. Her blood pressure on admission was 140/95 mm Hg. All routine biochemical tests were normal. An electrocardiogram (ECG) revealed sinus rhythm with normal axis. A chest radiograph done upon admission demonstrated a widened mediastinum and prominent pulmonary conus (Fig. 1A). The treadmill exercise stress test, which was done to determine myocardial ischemia, was normal. A subsequent transthoracic echocardiography (TTE) revealed left ventricular hypertrophy, left atrial dilatation, mild mitral and tricuspid valve insufficiency, dilatation of main pulmonary artery (parasternal short-axis view diameter: 33 mm), no significant trans-pulmonary valve pressure gradient and normal left ventricular systolic function (Fig. 1B). There were no echocardiographic features of right cardiac failure. A computed tomographic scan of thorax revealed bovine aortic arch associated with a massively dilated main pulmonary artery (MPA) as well as dilated right (RPA) and left pulmonary (LPA) arteries. The main pulmonary artery, RPA and LPA were dilated to 53, 33 and 45 mm in diameter, respectively. Additionally, the left main bronchus was compressed by LPA (Figs. 1C, 2A, 2B). Then, the patient was consulted by cardiovascular surgeon. Since our patient did not have an aneurysm 60 mm or greater in diameter, transpulmonary valve pressure gradient, and typical symptoms, it was

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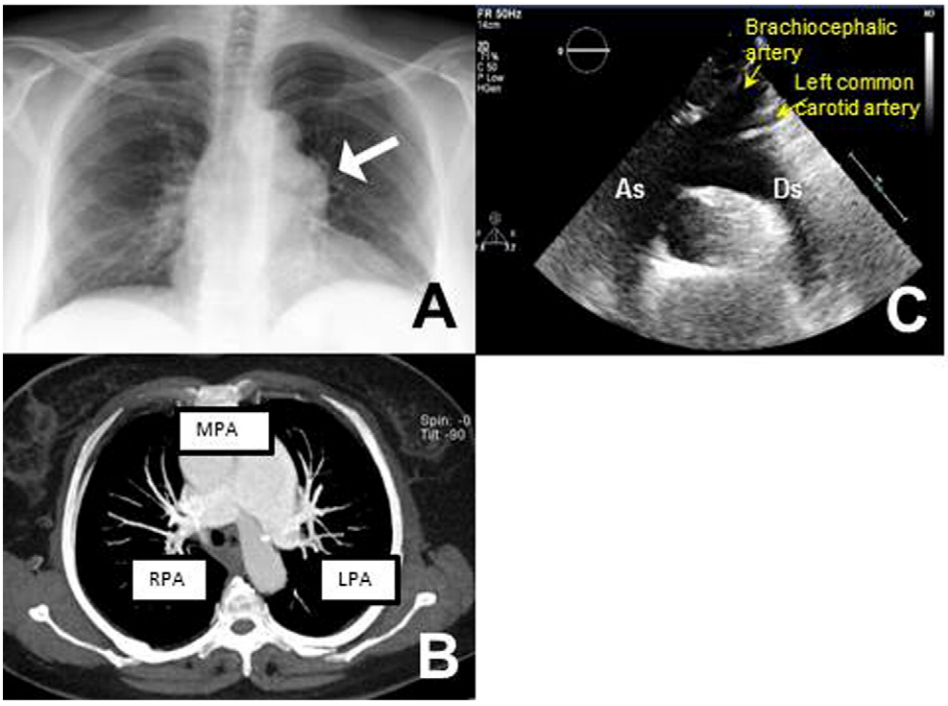

Fig. 1. (A) Posteroanterior chest radiography view demonstrated massive enlargement of the main pulmonary artery (arrow). (B) Axial contrast-enhanced computed tomography imaging revealed dilation of the main pulmonary artery and its branches. (C) Suprasternal view of echocardiography demonstrates bovine aortic arch. As = ascending aorta, Ds = descending aorta. MPA: main pulmonary artery, RPA: right pulmonary artery, LPA: left pulmonary artery.

decided to follow up at the clinic to re-assess her clinical status as well as the trans-pulmonary valve pressure gradient as an index of stability.

Discussion

Pulmonary artery aneurysm (PAA) is a rare condition, mostly arising from main pulmonary artery.[4] The cause of PAA may be idiopathic; however, other causes include congenital shunt disease, infection (tuberculosis, syphilis, osteomyelitis, pneumonia), systemic vasculitides (Hughes–Stovin's disease, Behcet's disease), collagen vascular diseases, connective tissue disorders, (Marfan's syndrome, Ehlers–Danlos syndrome), trauma (direct or blunt chest injury), mucoid vasculopathic changes, valvular pulmonary stenosis, and pulmonary hypertension.[5–7] Greene et al. described idiopathic PAA as one that satisfies the following criteria: a) enlargement of the pulmonary artery with or without

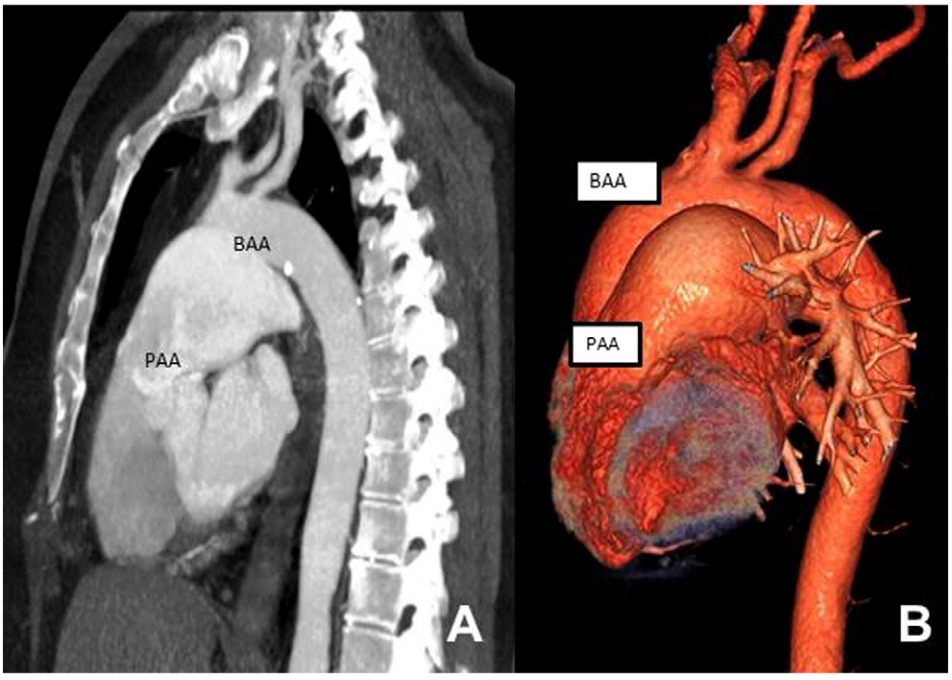

Fig. 2. (A) Lateral image of computed tomography demonstrates severe dilation of the pulmonary artery trunk (diameter: 53 mm) and bovine aortic arch. (B) The three-dimensional computed tomography image demonstrates the anatomy of the bovine aortic arch and pulmonary artery aneurysm. BAA: bovine aortic arch, PAA: pulmonary artery aneurysm.

involvement of the adjacent arterial trunk, b) absence of abnormal extracardiac or intracardiac shunts; c) absence of chronic pulmonary or cardiac disease, and d) more than minimal atheromatosis or pulmonary vascular tree arteriosclerosis or absence of arterial disease.[6] For the patient reported here, the degree of dilatation was marked and both right and left pulmonary arteries were involved. Since other causes of PAA were unavailable in her family and medical history, it was considered as idiopathic PAA. Diagnosis of idiopathic PAA is generally established with echocardiography to confirm a dilated main pulmonary artery and its branches, along with the presence or absence of valvular regurgitation.[4] Pathologically, the artery shows fragmentation of the media with degeneration and with less smooth muscle cells, which lead to progressive dilatation of the artery. These aneurysms are generally considered to be benign and less lethal, and there are no clear guidelines for the management of these aneurysms. Treatment ranges from simple follow-up with periodic echocardiographic assessments to surgical intervention. Surgical intervention has been recommended for those with an aneurysm that has a diameter of 60 mm or greater.[7] It was showed that long-term follow-up for several decades is possible in different studies.[4,6,7] One congenital variation of human aortic arch (AA) branching pattern in which the left common carotid artery (LCCA) originates from the BT is called as BAA. Although both BAA and idiopathic PAA have been reported separately in literature, no case has been reported as having both BAA and PAA.

Conclusion

We report the case of a patient with BAA associated with idiopathic PAA and bronchial compression, which was diagnosed by a simple chest radiograph. Since idiopathic PAA is a possible cause of rupture or dissection of pulmonary artery, and cardiac sudden death, it is considered to be in asymptomatic patients as well and needs a multidisciplinary approach for diagnosis and treatment.

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Short communication

Single coronary artery accompanying myocardial bridging on LAD and retroaortic course of LCX☆,☆☆,☆☆☆,★,★★

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ABSTRACT

Introduction: Coronary artery anomalies are a group of highly variable disorders. Patients with multiple coronary anomalies are very uncommon. Clinical manifestations of coronary artery anomalies can vary from asymptomatic to sudden cardiac death.
Case presentation: A symptomatic 43-year-old female patient with single coronary artery, symptomatic myocardial bridging on left anterior descending artery and retroaortic course of left circumflex artery is presented.
Management and outcome: A reversible perfusion defect was detected only in the anterior wall of the left ventricle. After treatment with beta blocker therapy and lifestyle changes, the patient is asymptomatic at 3 months of follow up.
Discussion: In most cases, it is difficult to determine the coronary anomalies by catheter coronary angiography alone. Similar to our case, non invasive methods such as multislice computed tomography coronary angiography may be useful to evaluate the coronary anatomy and define the coronary artery anomalies.

Introduction

Coronary artery anomalies consist a rare group of congenital heart diseases whose presentations and pathophysiological mechanisms are highly variable.[1] Incidence of coronary artery anomalies is approximately 1% for patients that undergone cardiac catheterisation.[2] A combination of multiple anomalies is very uncommon. We described a 43-year-old female patient with three coronary artery anomalies that are single coronary artery (SCA), myocardial bridging (MB) on the left anterior descending artery (LAD) and retroaortic course of the left circumflex artery (LCX).

Case report

A 43-year-old female patient was applied to cardiology polyclinic due to typical anginal complaint with effort. Rest electrocardiography was normal. Echocardiography and the physical examination were unremarkable. The patient has no risk factors except smoking and hypertension. We decided to perform catheter coronary angiography because of the occurrence of chest pain at exercise stress test. In her angiogram, we found that the LAD, LCX and right coronary artery (RCA) arose from a single coronary artery which originates from the right sinus of valsalva (Fig. 1A). Also, the MB was seen on the proximal segment of the LAD. Then, multislice computed tomography (MSCT) coronary angiography was performed to evaluate the patient's coronary anotomy in detail. As a result of MSCT coronary angiography, we saw that there was no coronary artery ostium at the left sinus of valsalva and the whole coronary system arose from a single trunk that originated from the right sinus of valsalva. This short common trunk trifurcated to three main branches; RCA, LAD and LCX (Fig. 1B). The proximal segment of the LAD proceeds intramurally through the myocardium and causes to MB (Fig. 2). A reversible perfusion defect was detected at the anterior wall of the left ventricle at myocardial perfusion scintigraphy. The course of LCX is retroaortic between the aorta and left atrium, and then reaches to posterolateral wall

☆ Prior publication: This article is an original case and it has not been published or submitted for publication elsewhere, in whole or in part, before submission to journal.
☆☆ Conflict of interest: We declared that we have no commercial, financial, and other relationships in any way related to the subject of this article all that might create any potential conflict of interest.
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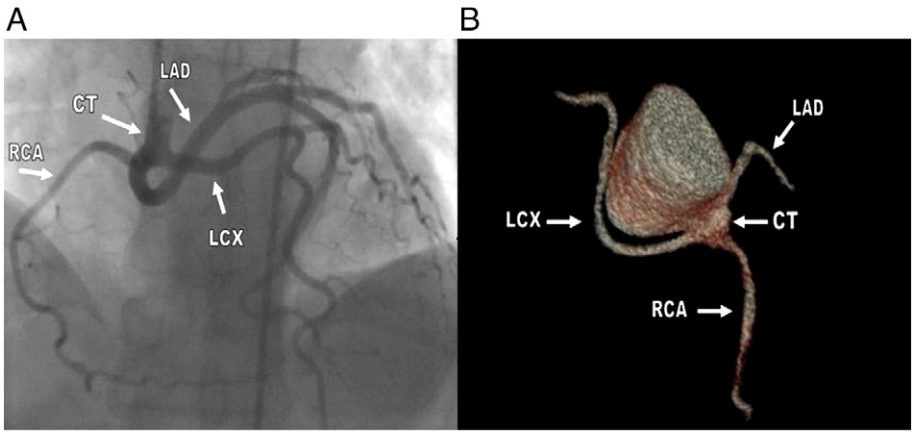

Fig. 1. A: Catheter coronary angiography image shows trifurcation of common trunk (CT: common trunk) B: 3D image of MSCT coronary angiography demonstrates trifurcation of common trunk (MSCT: Multislice computed tomography, CT: common trunk).

of the left ventricle (Fig. 3). The course of LAD was anterior to the pulmonary artery and there was no critical atherosclerotic stenosis in any coronary arteries.

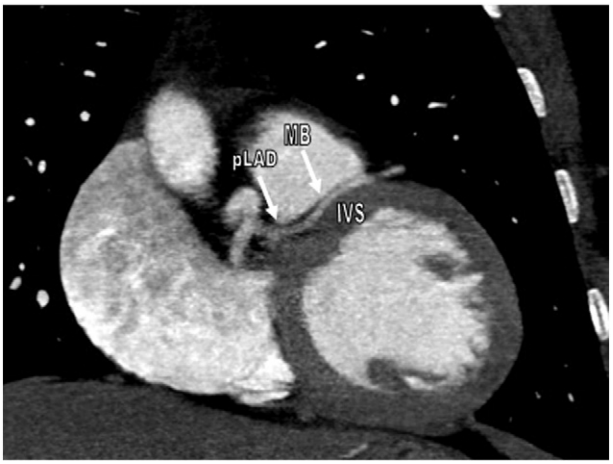

Fig. 2. Multiplanar image of MSCT coronary angiography shows myocardial bridging into the interventricular septum (MSCT: Multislice computed tomography, pLAD: proximal LAD, MB: myocardial bridging, IVS: interventricular septum).

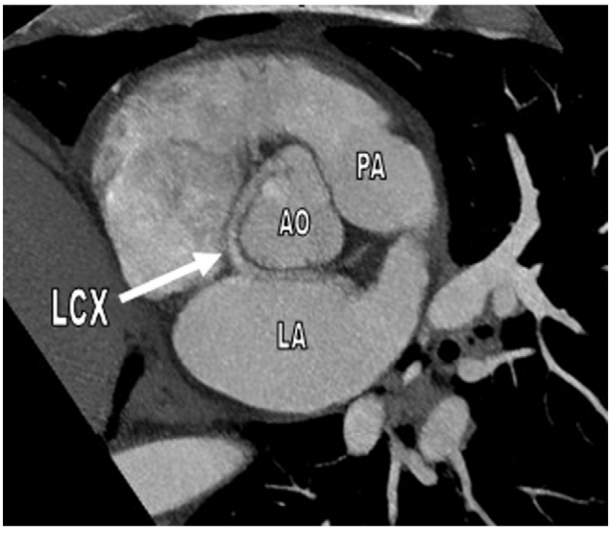

Fig. 3. Axial image of MSCT coronary angiography shows retroaortic course of LCX (MSCT: Multislice computed tomography).

Discussion

Coronary artery anomalies are frequently seen in combination with major congenital cardiac defects. The presence of multiple anomalies can be associated with potentially lethal conditions. SCA is an uncommon anomaly that only one coronary artery arises from the single coronary ostium and its clinical significance is unknown.[3] It's generally considered to be benign. However, some patients may have myocardial ischemia directly caused by the abnormal anatomy.[4] Right SCA is more uncommon.[5] In our case, SCA originates from the right sinus of valsalva. MB is a band of myocardium that lies on top of a coronary artery and it is characterized by systolic compression of this segment. MB is mostly localized on the LAD and it is clinically silent in most cases, but sometimes it can be associated with myocardial ischemia.[6,7] Both SCA and MB could be the cause of the patient's chest pain in our case. After performing myocardial perfusion scintigraphy, a reversible ischemic perfusion defect was detected only in the anterior wall of the left ventricle. We offered to the patient to avoid competitive sports and excessive exercise. These suggestions are combined with beta blocker treatment. When the drug dose is titrated to metoprolol 100 mg daily, the patient's chest pain was resolved. After three months of follow up, the patient is asymptomatic and no complication was observed. These findings also suggest that MB was the cause of chest pain for this patient. Both SCA and retroaortic course of the LCX may be only anatomic variations. Mostly, it is difficult to determine the coronary anomalies by catheter coronary angiography alone. MSCT coronary angiography is a non invasive, also a 3D imaging technique may be useful to evaluate the coronary anatomy and define the coronary artery anomalies.[8] We demonstrated an unusual case of a patient with coronary artery anomaly; single coronary artery, symptomatic myocardial bridging on the LAD and retroaortic course of the LCX was detected by catheter and a 64-multidetector MSCT coronary angiography.

Conflict of interest

Nothing to declare.

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Short communication

Presentation of adult Bland–White–Garland syndrome in a 32-year old female☆

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ABSTRACT

Anomalous left coronary artery originating from the pulmonary artery (ALCAPA or Bland–White–Garland Syndrome) is a congenital coronary artery anomaly characterized by risk of death due to heart failure and sudden cardiac death. The probability of survival to adulthood is very poor in untreated patients. We present the case of an untreated 32-year old female patient with this congenital anomaly who has been asymptomatic till now even though she has had 3 uncomplicated pregnancies.

Introduction

ALCAPA or Bland–White–Garland (BWG) Syndrome is very rare congenital anomaly affecting 1 in 300,000 live births, and accounts for 0.5% of cases of congenital heart disease.[1] In this rare congenital condition, the left coronary artery originates from the pulmonary artery instead of the left sinus of Valsalva and perfusion of left ventricle is, therefore, dependent on the development of collaterals from the right coronary artery which originates from the right sinus of Valsalva. Approximately 80–90% of the afflicted infants die within 4 months of age due to heart failure and/or sudden cardiac death,[2] and immediate surgical correction is recommended because of high risk of death. Nonetheless, survival to adulthood without any surgical correction is unusual and very few such cases have been reported to date. In this report, we present the case of a 32 year-old female with Bland–White–Garland Syndrome who has been asymptomatic until now, even though she has had 3 uncomplicated successful pregnancies.

Case

A 32 year-old female was admitted to our emergency department because of continuous chest pain for the last one hour. Her medical history was unremarkable and she was not taking any medication. She previously had three uncomplicated pregnancies and deliveries (11 year-old, 7-year-old and 5-year-old healthy children) and did not experience any problem during the pregnancies. Nonetheless, she complained of increasing shortness of breath and atypical chest pain with exertion for the last two months. Physical examination was unremarkable. A 12-lead electrocardiograph (ECG), administered upon admission, showed a left anterior fascicular block and ST-segment depression in leads V3–V6. ECG changes are compatible with ischemia. There are no specify changes of ST/T waves in this condition Transthoracic echocardiography demonstrated absence of any valvular heart disease with intact left systolic function. There wasn't left ventricular hypertrophy. There were no abnormalities in complete blood count and standard biochemical tests on admission, and repeat troponin tests were negative. We next performed an elective coronary angiography. During coronary angiography, we were unable to visualize the left coronary artery (LCA) in the aorta (Fig. 1A). It was also noticed that the right coronary artery (RCA) was markedly dilated and tortuous, with retrograde supply to the LCA through numerous prominent collaterals. The contrast agent drained from the left main coronary artery into the pulmonary artery, thus permitting a diagnosis of anomalous LCA originating from the pulmonary artery (ALCAPA or BWG Syndrome) (Fig. 1B). Subsequently, in order to delineate coronary anatomy, multi-slice computed tomography (MSCT) coronary angiography was performed which showed that the RCA arose normally from the right sinus of Valsalva, but that the LCA arose from the main pulmonary artery (Fig. 2A–2B). Given the high risk of sudden cardiac death in these patients, even in adult patients with minor symptoms, we recommended immediate surgical correction. After transversal opening of the pulmonary artery, ostium of the left coronary artery was identified and closed with pericardial patch with 5–0 polypropylene running

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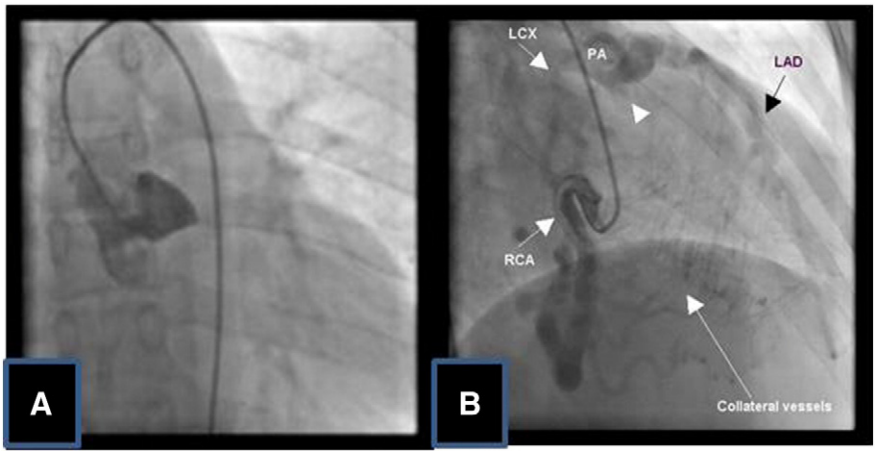

Fig. 1. Aortography shows that no coronary artery was originating from the left sinus of Valsalva (A), and right coronary artery angiogram shows an enlarged and torturous RCA filling left coronary artery via numerous collaterals and left coronary artery is drained into the main pulmonary artery (B). LAD: left anterior descending artery, LCX: left circumflex artery, RCA: right coronary artery, PA: pulmonary artery and arrowhead denotes the draining of contrast agent into the pulmonary artery.

suture (Ethicon, Somerville, NJ) and left coronary artery direct reimplantation of the LCA into the ascending aorta. TEE wasn't performed during the procedure. The clinical course after surgery was uneventful and the patient was doing well at the 6-month follow-up.

Discussion

The BWG syndrome is a very rare congenital anomaly and is usually fatal during the neonatal period. On the other hand, a recently published case report has shown that since many patients are asymptomatic until their death they remain undiagnosed.[3] The clinical manifestation of this condition is gradual and depends on the alterations in pulmonary circulation that occur after birth.[4] In the fetal period, systemic and pulmonary arterial pressures are equal, and from birth till about 2 months of age, because of the high resistance in pulmonary circulation, antegrade flow from the pulmonary artery to the LCA perfuses the left ventricle; thus sudden death is extremely rare in this age group. As pulmonary artery pressure gradually falls after 8 weeks of life, disruption of the left ventricular function begins because of low perfusion and low oxygen pressure. During this period, left ventricle perfusion or the extent of myocardial ischemia completely depends on the development of collateral circulation between the right and left coronary artery. If collateral flow is poor then the patient will die during the first year of life due to heart failure and/or sudden cardiac death.[5] However, patients with well-established collaterals survive through the neonatal- and childhood period into adulthood without any symptoms.[6,7] In our patient, no remarkable symptoms associated with heart failure appeared until the age of 32 years. Interestingly, she had three uncomplicated and successful pregnancies even though the pregnancies would have caused some degree of cardiac overload. We think that this patient remained asymptomatic until now due to an optimal balance between the dilated and tortuous RCA and LCA via well-established collaterals, and that the presenting symptoms are probably related to partial failure of this collateral circulation.

The diagnosis of BWG syndrome is made after a coronary angiography or a multi-slice CT, and three criteria must be met. These are: the LCA must not originate from the aorta, the LCA must originate from the pulmonary artery, and retrograde filling of the LCA from the RCA.[7] Other salient findings that are indicative of ALCAPA include left axis deviation, abnormal Q-waves in leads I and a VL, poor R-wave progression and ST/T wave changes on ECG.[6,7] An echocardiography may also uncover a previous anterolateral myocardial infarction, left ventricular hypertrophy, a dilated left ventricle with global hypokinesia, mitral insufficiency, and a reversal of flow from the LCA into the pulmonary artery, which constitutes a left-to-right shunt. The echocardiography can also reveal the presence of a turbulent diastolic flow within the

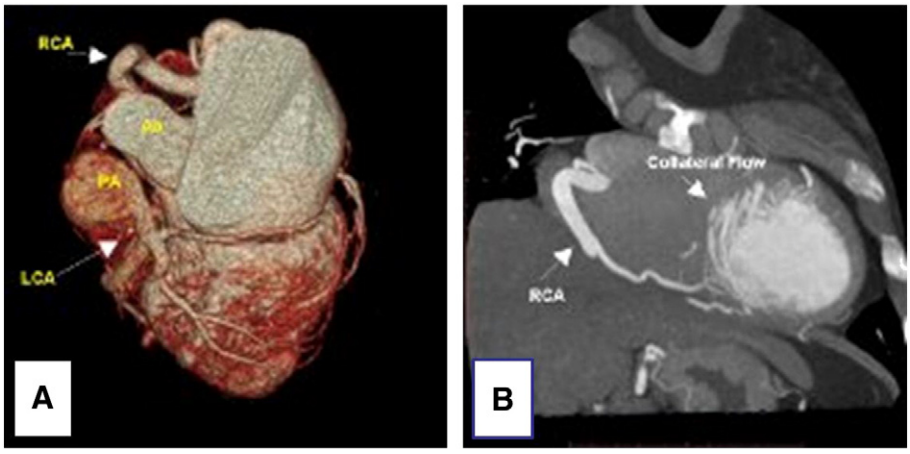

Fig. 2. 3D-multi-slice computed tomography (MSCT) shows that left coronary artery is arising from pulmonary artery and right coronary artery is arising from aorta (A), Also, MSCT shows the retrograde left coronary artery flow draining into the pulmonary artery (B) LAD: left anterior descending artery, LCX: left circumflex artery, RCA: right coronary artery, PA: pulmonary artery, Ao: aorta.

interventricular septum as a result of inter-coronary collaterals, and a dilated and tortuous RCA in the parasternal short axis.[8]

Sudden cardiac death is the most common cause of death in adults with ALCAPA and this may very well be its first clinical presentation. The presence of scar tissue from previous myocardial infarctions and the coronary steal phenomenon caused by inadequate perfusion are potential causes of malignant ventricular arrhythmias in these patients.[9] Therefore, immediate surgical correction after diagnosis is recommended in all patients, even in adult patients with only minor symptoms[10] as it has been shown that left ventricle function improves after a successful re-establishment of coronary circulation.[11]

The BWG syndrome is a very rare congenital anomaly and individuals can remain asymptomatic well into adulthood and even during recurrent pregnancies, as seen in this case report. There are about 58 cases reported of survival to adulthood in literature. It is important to be aware of this syndrome because of the high risk of sudden cardiac death, even though patients may be asymptomatic. Immediate surgical correction after diagnosis is recommended.

Comments

Case characteristics

An untreated 32-year old female patient with this congenital anomaly who has been asymptomatic till now even though she has had 3 uncomplicated pregnancies.

Clinical diagnosis

Physical examination was unremarkable. A 12-lead electrocardiograph (ECG), administered upon admission, showed a left anterior fascicular block and ST-segment depression in leads V3–V6.

Differential diagnosis

Angına pectoris, Valvuler heart disease.

Imaging diagnosis

The patient underwent coronary angiography we were unable to visualize the left coronary artery (LCA) in the aorta It was also noticed that the right coronary artery (RCA) was markedly dilated and tortuous, with retrograde supply to the LCA through numerous prominent collaterals. The contrast agent drained from the left main coronary artery into the pulmonary artery, thus permitting a diagnosis of anomalous LCA originating from the pulmonary artery.

Multi-slice computed tomography (MSCT) coronary angiography was performed which showed that the RCA arose normally from the right sinus of Valsalva, but that the LCA arose from the main pulmonary artery.

Treatment

The patient underwent surgical correction with direct re-implantation of the LCA into the ascending aorta.

Related reports

It was rare in the literature that an untreated 32-year old female patient with this congenital anomaly who has been asymptomatic till now even though she has had 3 uncomplicated pregnancies.

Experiences and lessons

Despite Given the high risk of sudden cardiac death in these patients rare cases can be survival to adulthood.

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Short communication

Percutaneous ASD and VSD closure of 4-month-old infant in the same session

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ABSTRACT

Percutaneous closure of septal defects is a successful treatment modality that has been used for a long period of time in children.
Our main objective in this case report is to discuss the transcatheter closure of atrial and ventricular septal defects of 4-month-old infant in the same session. As far as we know, this case is the smallest one in age that percutaneous VSD closure was done in the same session with ASD closure.
A 4-month-old boy with tachypnea, tachycardia diagnosed to have aneursymatic perimembranous ventricular septal defect (VSD) sized 4 mm and atrial septal defect (ASD) sized 8 mm. Anti-congestive treatment was started, but despite to the treatment, his symptoms continued and he was hospitalized 3 times for lower respiratory tract infections. Surgery was found as too risky because his lung parenchyma was not good and body weight was low. Therefore, transcatheter closure was planned. VSD was closed with 4 × 4 Amplatzer® Ductal Occluder II device, ASD with 9 mm-sized Amplatzer® Septal Occluder. In his first month control: his body weight increased to 6.2 kg.
In conclusion, percutaneous ASD, VSD closure is being done safely in children, but for the first time, percutaneous VSD closure was done in an infant with low body weight in the same session with ASD closure successfully. This case will be an encouraging example for the future.

Introduction

Percutaneous closure of septal defects is a successful treatment modality that has been used for a long period of time in children.

Our main objective in this case report is to discuss the transcatheter closure of atrial and ventricular septal defects of a 4-month-old infant in the same session. As far as we know, this case is the smallest one in age that percutaneous VSD closure was done in the same session with ASD closure, except hybrid procedures.

Case report

A 4-month-old boy; weighing 4.6 kg, was referred to our clinic for the heart murmur. He suffered from having frequent lower respiratory tract infections. His weight gain was insufficient. He had tachypnea (68/min), tachycardia (150/min), and 2–3°/6 pansystolic murmur in his physical examination. Aneurysmatic perimembranous ventricular septal defect (VSD) sized 4 mm and atrial septal defect(ASD) sized 8 mm (Fig. 1) were detected by transthoracic echocardiography. Anti-congestive treatment was started, but despite the treatment, his symptoms continued and he was hospitalized 3 times for lower respiratory tract infections. Also, bilateral renal calculi were found in abdominal ultrasonography.

Surgical closure of septal defects was planned, but since the lung parenchyma was not good and his body weight was low, it was accepted as too risky. Therefore, transcatheter closure was planned. Qp/Qs ratio was measured as 4, pulmonary artery pressure was 37/10 mean 29 mm Hg. VSD was closed with 4 × 4 Amplatzer® Ductal Occluder II device, ASD with 9 mm-sized Amplatzer® Septal Occluder (Figs. 2, 3). The VSD was passed through by using a right Judkins catheter or a partly cut pigtail catheter. A hydrophilic glide wire was passed across the defect into the right ventricle and then into the pulmonary artery or superior vena cava, where it was snared and withdrawn from the femoral vein thus establishing an arteriovenous loop. Subsequently, a sheath was advanced to the left ventricle via the arteriovenous circuit. An occluder was advanced into the delivery sheath. Then the occluder was positioned on the VSD. Intervention was guided transthoracic echocardiography in addition to floroscopy.

We have been following the patient for 1 year. We have seen him firstly 1 week later on; 1 month, 3 months, 6 months after the procedure. Each time we did his physical examination, checked his electrocardiogram, and controled valvular insufficiency and device position with transthoracic echocardiography, we have not faced with any problem even the vascular ones. In his first month control, his body weight increased to 6.2 kg, his cardiac functions were normal, no residual shunt was detected in echocardiographic examination. We also were not faced with conduction problems like AV block.

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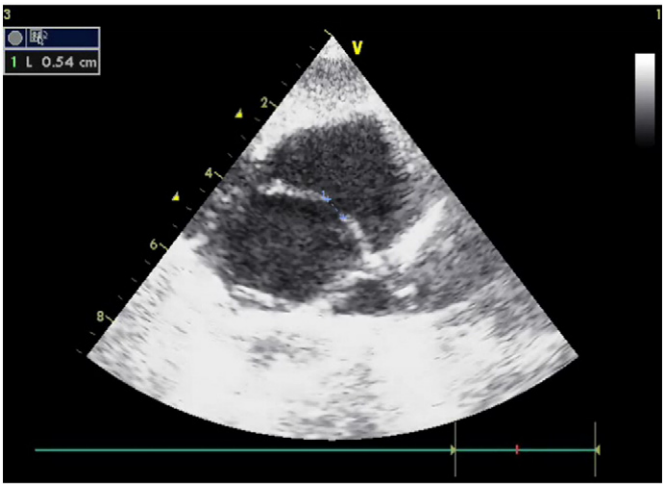

Fig. 1. Echocardiographic image of ASD.

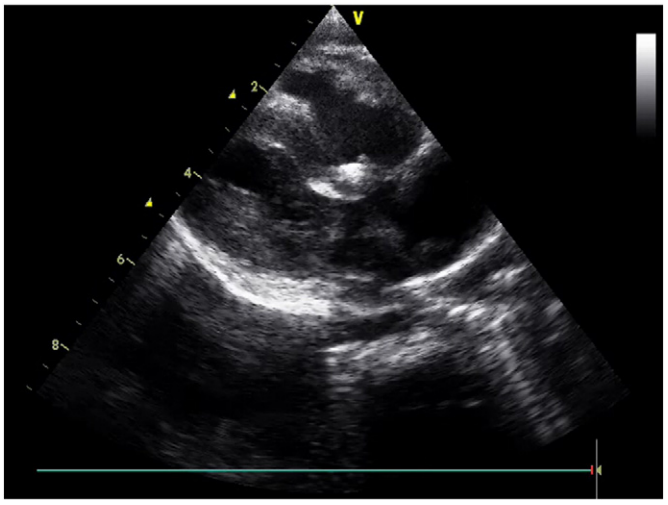

Fig. 3. Echocardiographic image of VSD after closure with ADO II device.

Discussion

Large atrial septal defects may lead to congestive heart failure, frequent respiratory infections, and growth retardation in infants.[1]

Also ventricular septal defects that have significant shunt, increase pulmonary blood flow, enlarge the left chambers of heart, cause cardiac dysfunction, arrhythmia and constitute a risk factor for infective endocarditis.

Surgical closure of septal defects is gold standard, it is safe and effective; however, the morbidity associated with sternotomy/thoracotomy, cardiopulmonary bypass, postoperative complications, and residual surgical scarring cannot be avoided.

Transcatheter closure of perimembranous VSDs constitutes special care because of greater technical difficulty arising from proximity to the aortic valve and conduction tissue. In order to minimize such risks; appropriate device should be selected according to the type, location, and the size of the defect. Amplatzer® Ductal Occluder (ADO) is actually designed for ductal closure. As we mentioned in previous studies, shape of aneurysmatic VSD resembled PDA. Therefore, it makes closure of VSD with septal aneurysm possible with the Amplatzer duct occluder. The left ventricular margin of the VSD is larger than the right one. The left ventricular margin resembles the aortic side and ampulla of a PDA while the right ventricular margin and septal aneurysm resemble the narrowing of a PDA at the pulmonary artery margin.[2] The design of the ADO II device is soft in nature with no polyester material that does not apply a direct force on conduction system. We have done 21 VSD closures with ADO II device in 3 years interval in our institution. We did not face with any complication and our success rate was 100%.

ADO II device are normally contraindicated in the patients less than 6 kg and less than 6 months of age. But it is frequently used for PDA closure for newborns less than 6 kg. We have not faced with a case less than 6 kg whose VSD was closed percutaneously in the literature.

New advances in pediatric cardiology enable us to apply interventions safely and effectively to the younger patients even the newborn. Interventions to multiple defects in the same session is a tough issue in children. More common usage of combined transcatheter procedures, lessened the ratio of surgery needed cardiac lesions and decreased the mortality and morbidity related to surgery. Mostly balloon angioplasty or valvuloplasty were done with ASD, VSD, or PDA closure.[3,4,5]

We have only faced a single adult case whose VSD and ASD closed in the same session percutaneously.[6]

In conclusion, percutaneous ASD, VSD closure is being done safely in children, but for the first time, percutaneous VSD closure was done in an infant with low body weight in the same session with ASD closure successfully. This case will be an encouraging example for the future.

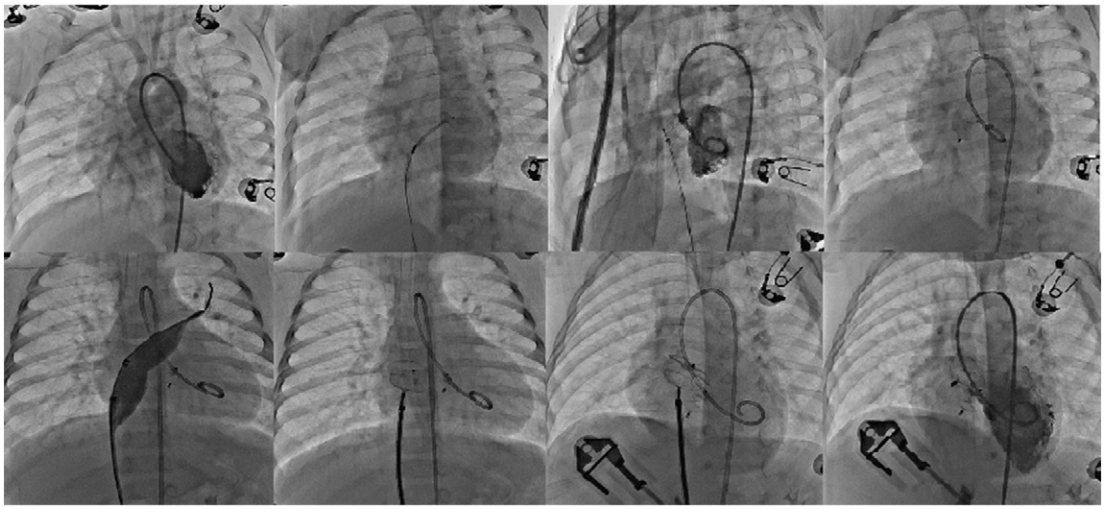

Fig. 2. Percutaneous ASD, VSD closure.

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Short communication

Amiodarone-induced exudative bullous lesion and hepatotoxicity in a patient with ventricular tachycardia

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ABSTRACT

Amiodarone is a potent, iodine rich, highly lipophilic class III antiarrhythmic drug widely used for the management of both supraventricular and ventricular arrhythmias. It tends to concentrate in tissues including fat, lung, liver cornea and skin. Several side effects have been reported in patients taking amiodarone. The mechanisms of amiodarone-induced side effects are poorly understood. Accumulation of amiodarone in tissues and organs has been suggested as a possible mechanism. The most frequent dermatologic side effects are photosensitivity, skin discoloration and erythema. This article presents the case of a patient who developed amiodarone-induced bullous skin lesions and hepatotoxicity.

Introduction

Amiodarone is considered as a class III antiarrythmic (Vaughan Williams classification). It possesses electrophysiologic characteristics of sodium, potassium and calcium channel blockage, as well as alpha and beta adrenergic blocking activity.[1,2] It is commonly used in the treatment of supraventricular and ventricular arrhythmias, such as atrial fibrillation and ventricular tachycardia.[3] It has strong antiarrythmic properties but may cause neurologic, pulmonary, thyroid, ocular, dermatologic, and hepatic toxicity in long-term or excessive use. The mechanisms of side effects are not well understood. The most common side effects of amiodarone are nausea and vomiting, which occur in approximately 25% of patients. Side effects of the skin are also common, occurring in approximately 15% of patients.[4] Patients frequently experience photosensitivity. Other dermatological side effects of amiodarone include skin discoloration and blue-gray or yellow-brown pigmentation. In this report, we present a case of ventricular tachycardia that developed erythematous exudative bullous skin lesions and hepatotoxicity during amiodarone therapy.

Case presentation

Sixty-one year old male patient was admitted to the emergency department with complaints of palpitation, dyspnea and chest pain. He had the same symptoms twice in the last 24 h. He denied any syncope or dizziness. He had a history of hypertension and a coronary angiography which was performed 2 years ago. The angiogram showed slow flow in the coronary arteries without any other coronary pathology. The patient was treated with acetylsalicylic acid (ASA) and antihypertensive medications.

The patient had a hemodynamically stable ventricular tachycardia. The remaining systemic examination was normal.

Electrocardiography (ECG) showed sinus rhythm in leads I–III, aVR, AVL, and AVF at initial recording. The rest of the ECG revealed monomorphic, monofocal ventricular tachycardia (VT). The rhythm on cardiac monitor was consistent with VT (Fig. A). Hemogram and biochemical parameters were within normal limits.

Two-dimensional (2-D) echocardiography was performed after medical cardioversion and cardiac measurements were determined by 2D, cross sectional M mode and Doppler study echocardiography. Echocardiographic measurements as follows: right ventricle diameter: 1.9 cm, left ventricular end-diastolic diameter: 4.5 cm, interventricular septum diastolic diameter: 1.4 cm, and the left ventricular posterior wall diastolic diameter: 1.2 cm. Left ventricular ejection fraction was calculated as 58% by using the modified Simpson technique. There was no valvular, supravalvular or infravalvular stenosis in the aorta and pulmonary artery.

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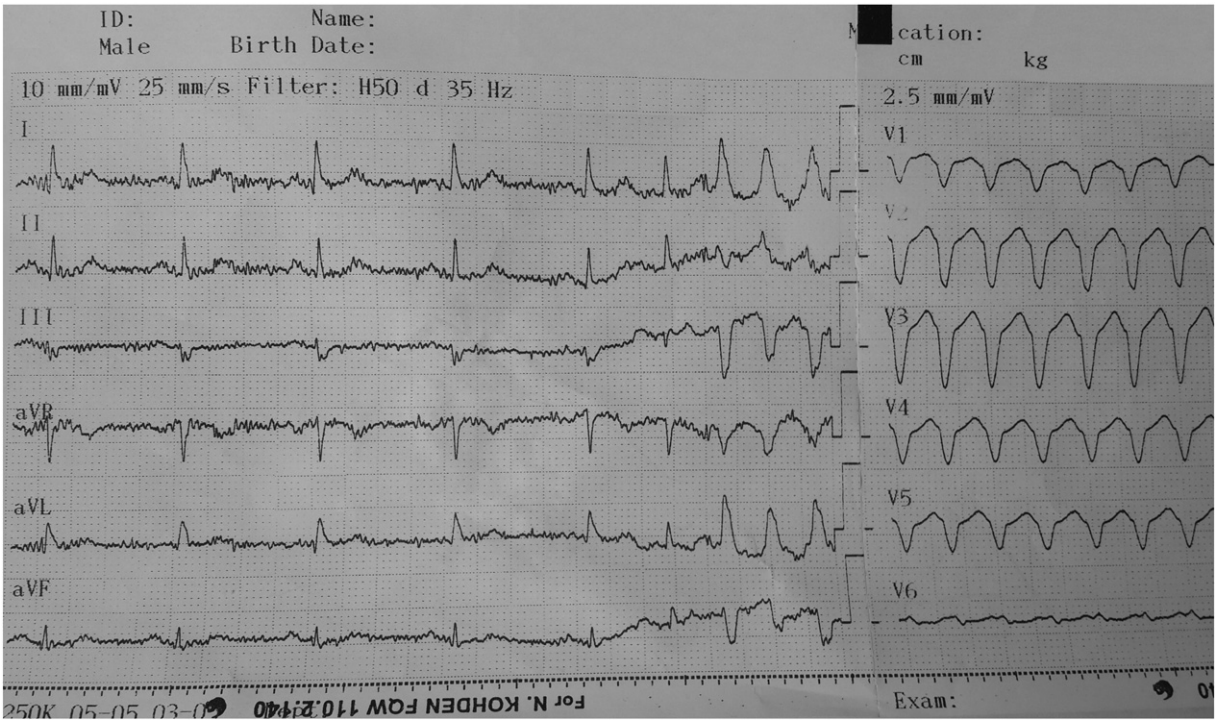

Fig. A. 12-Lead surface ECG during ventricular tachycardia.

The patient was diagnosed with sustained VT. Due to patient's stable clinical and hemodynamic parameters it was decided that a medical cardioversion was required. After loading dose of amiodarone, (150 mg in 100 mL dextrose (D5W) administered over 10 min), the patient was returned to sinus rhythm. His dyspnea and palpitations disappeared. The patient was taken to the coronary care unit and therapy was continued with a slow loading infusion containing 900 mg amiodarone injection in 500 mL of dextrose (D5W). The infusion was administered at a rate of 1 mg/min for 6 h followed by maintenance infusion at a rate of 0.5 mg/min over 18 h.

Coronary angiography revealed ectasia in the proximal and mid portions of the LAD and Cx with no critical obstructive lesions. There was also slow flow in both coronary arteries.

One day later, the patient had elevated liver enzymes and developed multiple bullous skin lesions over the anterior chest wall filled with exudative material. All bullae appeared on slightly erythematous base. Diameters of the largest and the smallest bulla were 2.3 × 1.5 cm and 0.6 × 0.7 cm respectively. Erythema was more prominent around the hair follicles and occurred both on the normal and involved skin (Figs. B and C). The biochemistry and hemogram results were as follows: Alanine transaminase (ALT) 99 U/L (normal < 41 U/L), aspartate transaminase (AST) 89 U/L (normal < 40 U/L), lactate dehydrogenase (LDH) 210 U/L (normal 135–225 U/L), monocyte 0.59 c/μL (normal 0.12–1.2 c/μL) and basophiles 0.08 c/μL (normal 0.00–0.02 c/μL). The results of other laboratory tests were within normal limits.

Amiodarone was stopped and the patient was started on propafenone (class Ic antiarrhythmic agent) orally every 12 h. During the follow-up, no VT or other arrhythmia was observed and liver enzymes returned to normal. He was discharged on the sixth day of hospitalization with prescriptions of acetylsalicylic acid 100 mg 1 × 1, propafenone 150 mg 2 × 1 and valsartan 160 mg 1 × 1. One month later, at the follow-up visit, the patient noted that his symptoms had subsided. The patient's biochemical, hemogram and ECG results were normal. Bullae healed leaving scar tissue (Fig. D). Twenty four hour holter monitoring showed no pathological findings.

Discussion

Amiodarone is probably one of the most potent antiarrhythmic medications that we have available. Unfortunately, it also has side effects; up to 40% of patients quit amiodarone within 2 years due to those side effects.[1,5,6] It is metabolized in the liver by cytochrome p450 enzymes and excreted in the bile. Less than 1% of the dose excreted in the urine.[7] Its main metabolite, desethylamiodarone, has also multiple side effects.[8] The mechanisms of side effects are not fully understood. Side effects include photosensitivity (25% to 75%), blue-gray or yellow-brown skin discoloration located mostly on the face, ears, and palms of the hands (4% to 9%), hepatotoxicity (15% to 30%); optic neuropathy/neuritis (1% to 2%), corneal microdepsits (>90%), pulmonary toxicity (1% to 17%), hypothyroidism (6%), hyperthyroidism (0.9% to 2%), tremor and ataxia (3% to 35%).[3] Peripheric neuropathy, insomnia, memory loss and delirium are uncommon side effects that have been reported in the literature. Life-threatening anaphylaxis or anaphylactoid reaction, pulmonary alveolitis and fibrosis have been reported.[2,9]

Our patient had elevated liver enzyme levels and multiple bullae. Bullae filled with no purulent fluid resembling exudates. Light microscopic skin examination revealed normal epidermis. The Hematoxylin-Eosin-Saffron stained sections demonstrated few yellow brown granules within cytoplasm of histiocytes in the mid dermis. These pigmented histiocytes clustered around capillaries. Minimal inflammation was observed. Histochemically the pigments were strongly positive by Ziehl–Neelsen, mild to moderate positive by PAS and negative by turnbull blue stainings. [10,11]

Electron microscopy revealed polygonal to fusiform shaped histiocytes with large cytoplasm, dispersed between dermal collagen fibers. Spheroid and extended nucleolus with dense chromatin network and large cytoplasm contained dense bodies ranging in size from 250 to 2500 nm, dense granules and eosinophilic materials. No difference

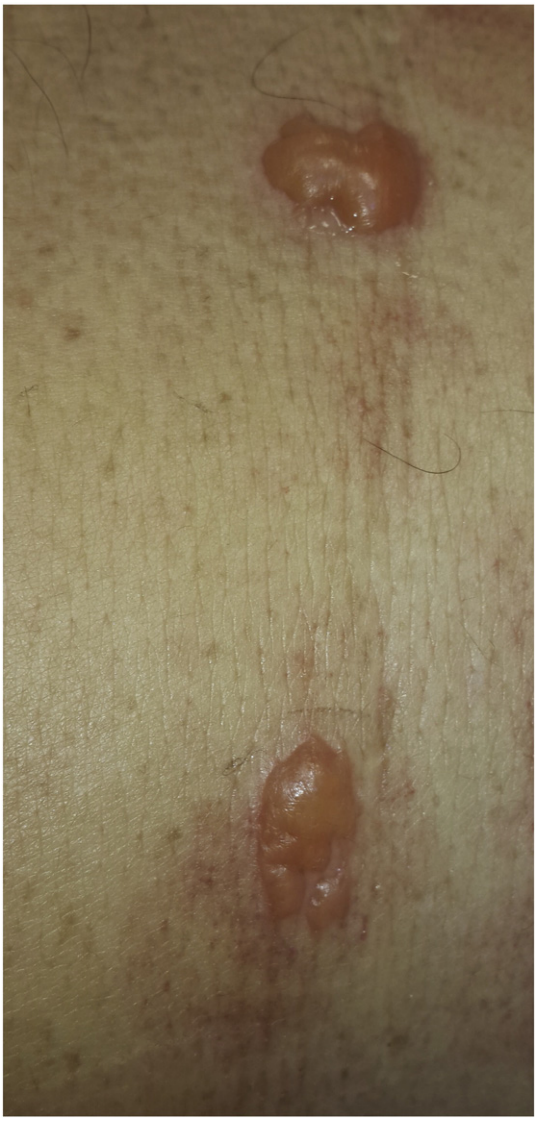

Fig. B. Multiple bullous lesions filled with material resembling exuda.

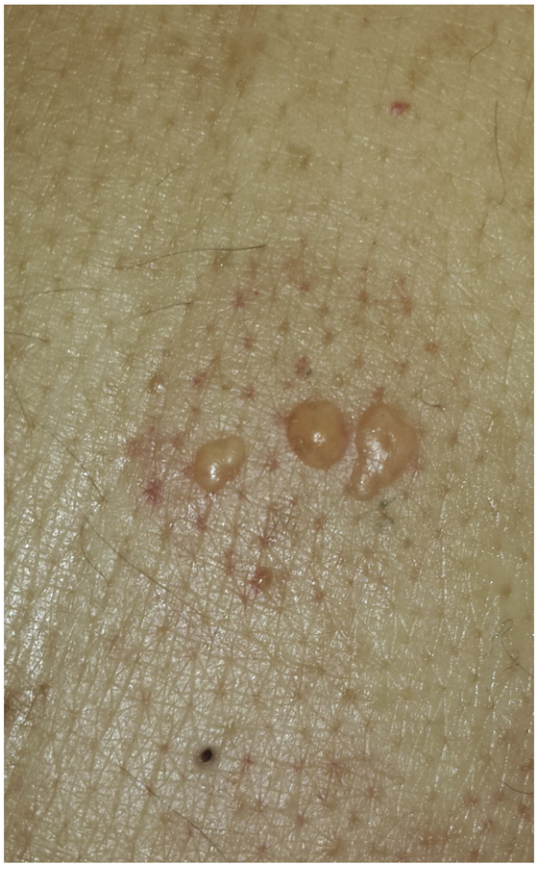

Fig. C. Multiple bullous lesions filled with material resembling exuda.

was reported in the other organelles including mitochondria, endoplasmic reticulum and free ribosomes.[10,11]

Dermatological complications of amiodarone are time-and dose-dependent. In most cases adverse effects are reversible with discontinuation of therapy. The main skin changes induced by amiodarone are phototoxic and photoallergic reactions, as well as hyperpigmentation. More than 10% of patients have been reported to have sunlight sensitivity. The symptoms begin in several minutes after exposure to the sunlight, continue up to 24 h and usually subside in approximately 48 h, but in some cases they persist up to 72 h. Phototoxic and photoallergic reactions might even occur a few months after the withdrawal of amiodarone due to its long elimination time.[12] During long-term or high-dosage amiodarone therapy, patients should be recommended to protect themselves against photosensitivity. Patients should wear lightweight clothing that covers most surface areas of skin and use sunscreen to protect themselves from harmful UV-A/UV-B radiation.[3]

Blue-gray discoloration of the skin is an uncommon adverse effect caused by the accumulation of lipofuscin and melatonin in dermis.[13] The appearance of the discoloration depends on the dosage of amiodarone and duration of use, and may regress upon cessation of the treatment.[14] Biopsy confirms skin impregnation of iodine with deposition of brown pigment within dermal microphages. Occurrence of skin lesions does not require discontinuation of therapy. Treatment should be stopped if liver enzyme levels exceed three times the upper limit of normal.[15]

The amiodarone molecule contains a large amount of inorganic iodine, which may exacerbate certain skin diseases, such as dermatitis herpetiformis and psoriasis. There have been reports of post-amiodarone pseudo-purulent changes, such as acne or blister-purulent lesions characteristic of iodism.[12,16] Less common dermatological complications of amiodarone include hives, pruritus, erythema nodosum, purpura, and the most severe variant of erythema multiforme-toxic epidermal necrolysis.[17,18]

Amiodarone can induce allergic reactions in sensitized patients. Although the mechanisms of allergic reaction to amiodarone are poorly understood, it is believed to occur due to the 37.3% iodine that is present in the amiodarone molecule. The incidence of amiodarone induced hypersensitivity reactions in hospitalized patients with a listed allergy to iodine or iodinated contrast agents was found to be 0.4%.[19] Physicians should be cautious while prescribing amiodarone for such patients.

Conclusion

Amiodarone is a potent antiarrhythmic agent that is used to treat ventricular and supraventricular arrhythmias. Side effects due to amiodarone are related to dosage and route of administration. Skin lesions are common side effects of amiodarone therapy and do not require discontinuation of treatment. Treatment should be stopped if patients develop hepatotoxicity or liver enzyme elevations greater than the triple normal limit. It should be kept in mind that exudative bullous skin lesions may occur during amiodarone therapy.

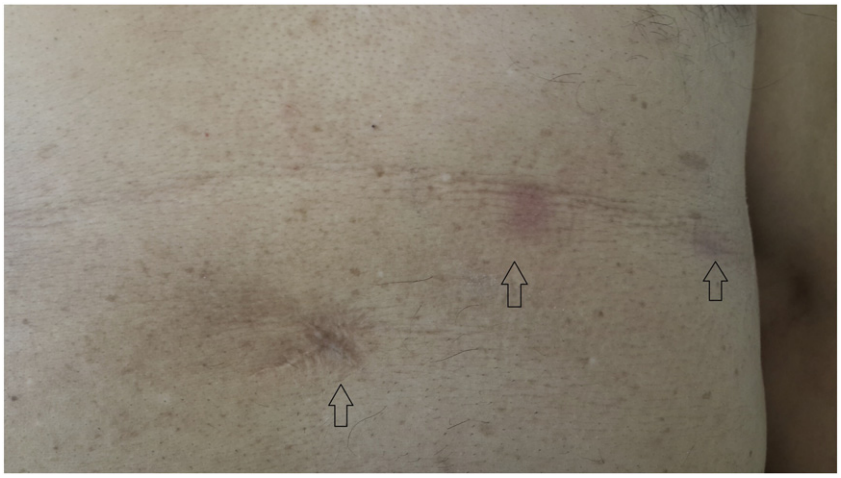

Fig. D. At one month follow-up visit, the patient showed healed bullae with scarring (black arrows).

Conflict of interest

None declared.

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Short communication

Primary spontaneous coronary dissectİon in a young male and the role of intravascular ultrasonography for diagnosis and treatment

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ABSTRACT

Primary spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome and is observed especially during gestation or the postpartum period of young healthy female patients. SCAD is much more rarely seen in young male patients. A 32 year-old male was admitted to our clinic with a typical anginal complaint that had begun after a verbal discussion in a family environment. The patient was hospitalized with a diagnosis of acute myocardial infarction without ST elevation. During coronary angiography, a lesion seen as a dissection in the proximal region of the left anterior descending artery (LAD), 30% stenosis in the circumflex artery and plaque in the right coronary artery were observed. In the LAD osteal region, a dissection flap and intimal rupture was observed at the 12:00–2:00 o'clock position using intravascular ultrasonography (IVUS). Our case was very young and had no atherosclerotic risk factors except for a history of smoking. It was suggested that intense emotional stress was the triggering factor for coronary dissection. The gold standard imaging method for spontaneous coronary dissection is coronary angiography. Recently, imaging methods like IVUS have made important contributions to the diagnosis of dissections that cannot be detected by coronary angiography. Treatment must be individual since there is no standard protocol. Medical therapy, percutaneous coronary intervention and coronary artery bypass surgery are the main treatment options.

Introduction

Primary spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome and sudden cardiac death.[1] It is observed especially during gestation or the postpartum period of young healthy female patients who do not have classical cardiovascular risk factors.[2] SCAD is much more rarely seen in young male patients. Early diagnosis is highly important to be able to start available treatment procedures.[3] Intravascular ultrasonography (IVUS) is a helpful imaging method that provides early diagnosis and appropriate stent size implantation.

In this paper, a case of a 32-year-old is presented, who had a stent implantation after emotional stress acute coronary syndrome because of left descending artery dissection detected by coronary angiography and IVUS.

Case presentation

A 32-year-old male was admitted to our clinic with a typical anginal complaint, which had begun after a verbal disscussion in a family environment. His history included smoking 10 packs of cigarettes per year. There was no disease or medication use in his history. There was also no significant condition in his background.

A sinus rhythm was detected in electrocardiography with an ST segment depression at V1–V6 derivations. HemoglobineThe initially evaluated Troponin I level was 5.3 ng/ml (reference value: 0–0.6 ng/ml). An ejection fraction of 50% with mild aorta insufficiency and mild hypokinesia of the anterior wall were detected in echocardiography, while all other valvular structures were normal.

The patient was hospitalized with a diagnosis of acute myocardial infarction without ST elevation and coronary angiography (CAG) was planned. During coronary angiography, a lesion seen as a dissection in the proximal region of the left anterior descending artery (LAD), 30% stenosis in the circumflex artery and plaque in the right coronary artery were observed. The dissection imaged lesion in the LAD was passed with a 0.014 mm guidewire and imaging with an Atlantis SR Pro 40 MHz (Boston Scientific, Freemont, CA) intravascular ultrasonography

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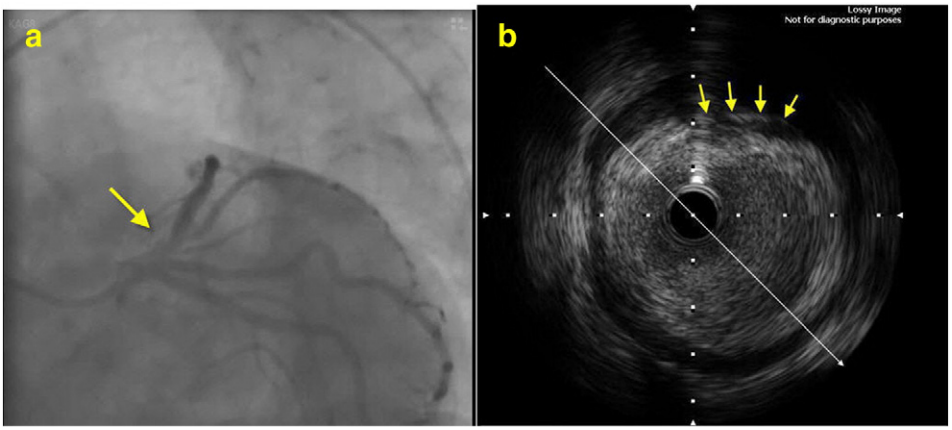

Fig. 1. (a) The dissection imaged lesion in LAD was shown in coronary angiography (CAG). (1b) In LAD osteal region, a dissection flap and intimal rupture was observed at the 12:00–2:00 o'clock position by imaging via intrvascular ultrasonography (IVUS).

(IVUS) catheter. In the LAD osteal region, a dissection flap and intimal rupture were observed at the 12:00–2:00 o'clock position (Fig. 1). The maximum LAD diameter was 4.8 mm in the osteal region as measured by IVUS. A stent of 5.0* 12 mm in size was implanted by inflating at 12 atmospheres pressure to the dissected lesion in the LAD osteal region. Imaging by IVUS after the procedure showed that the stent apposition was not appropriate. For this reason, the balloon was again put through the stent and inflated at 16 atmospheres pressure. Control imaging by IVUS showed an exactly appropriate apposition of the stent structure (Fig. 2). The patient was discharged with a prescription of acetylsalicylic acid, clopidogrel and atorvastatin.

Discussion

Spontaneous coronary artery dissection is defined as a rupture in the coronary artery wall caused by traumatic and non-iatrogenic reasons. This process results in myocardial ischemia caused by disruption of blood flow in the real lumen because of pseudo lumen formation and intramural hematoma.

Many etiologic conditions causing non-atherosclerotic SCAD exist. These are mainly fibromuscular dysplasia, gestation, connective tissue disorders, systemic inflammatory dieases, hormonal therapy and idiopathic causes. The reasons triggering SCAD include intense exercise, intensive emotional stress, birth labor, conditions with powerful valsalva maneuver such as heavy coughing, vomiting, and drugs such as cocaine and amphetamine.[4] Spontaneous coronary artery dissection is more common in the female population. This fact makes us consider that hormonal factors may play an important role. It is well known that hormonal factors have a role in medial degeneration of collagen synthesis. No descriptive data exist for male cases. Our case was very young and had no atherosclerotic risk factors except a history of smoking. It was suggested that intense emotional stress was the triggering factor for coronary dissection.

The number of case reports presenting post-emotional stress coronary artery dissection is very low. Firstly, in 1998, Pamar et al. detected coronary artery dissection by coronary angiography in 2 of 4 patients with no coronary artery disease, but who experienced horrible nightmares.[5] Hendir et al. reported only one emotional stress–related SCAD case in 1100 acute myocardial infarction patients.[6] Mayr et al. have described a 51-year-old woman who had SCAD at work after intense emotional stress.[7] Kaya et al. described acute inferior myocardial infarction due to spontaneous coronary artery dissection in a 21-year-old medical school student who experienced examination stress.[8] The fact that our case experienced severe angina pectoris after an intense argument in the family made us consider that emotional stress might be a triggering factor in development of the spontaneous coronary dissection.

Coronary angiography is the most commonly used imaging method in the diagnosis of SCAD.[9] SCAD can be diagnosed during CAG by demonstration of a radiolucent intimal flap frequently associated with contrast staining on the vessel wall. Although CAG can clearly show the lumen of the vessel wall, it cannot depict the vessel wall. Therefore, it is difficult to diagnose SCAD with development of intramural hematoma. Recently, IVUS has been able to provide novel diagnostic insights in patients with SCAD. Coronary lumen and vessel walls are nicely depicted with this technque. IVUS can demonstrate clearly dissected membrane, and true and false lumen, which cannot be detected on coronary angiography in patients with silent SCAD.[10] IVUS can provide clear pictures of intramural hematoma and false lumen thrombosis,

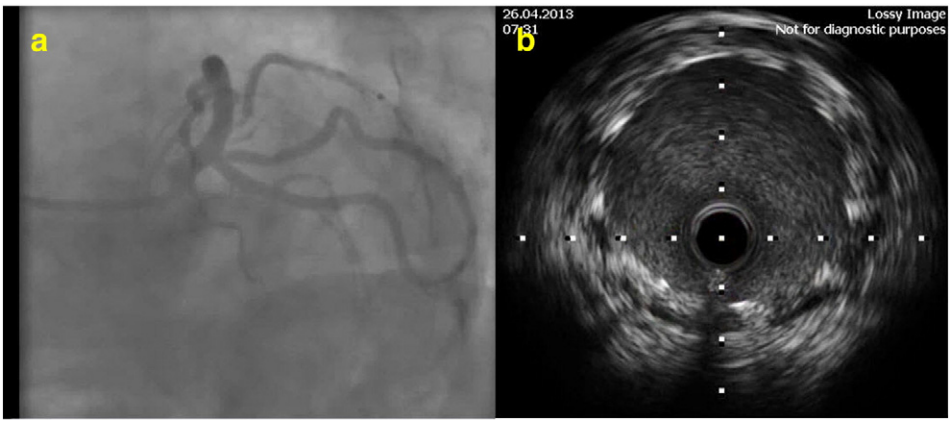

Fig. 2. (a) Dissected lesion in LAD was closed after stent implantation. (2b) IVUS image showed the apposition of stent structures.

which can be misdiagnoesd as atherosclerotic narrowing on CAG. Although we could see a lesion like an intimal flap in the proximal region of the LAD during CAG, SCAD was confirmed clearly with IVUS by demonstrating a dissected flap, and also the exact localization of the lesion could be depicted.

IVUS has also been used to optimize the results of stent implantation in patients with SCAD requiring coronary intervention. Stent size and length may be selected by considering the true vessel size and the true longitudinal extension of the dissection, thereby ensuring optimal stent appositon to the vessel wall.[11] In our case, we determined the stent size according to the IVUS measurement. Indeed, insufficent stent expansion and apposition could be depicted after reevaluating with IVUS. Therefore, it was necessary to postdilate with a balloon catheter. After postdilatation, optimal stent expansion, stent appositon and complete closure of the dissection line can be ensured with IVUS guidance.

Treatments must be individual because there is no standard protocol. Medical therapy, percutaneous coronary intervention and coronary artery bypass surgery are the main treatment options.[12] Percutaneous coronary intervention was preferred in our case because of the continuous anginal complaint, LAD osteal region dissection development and wide ischemia of the myocardium. We continued dual antiplatelet therapy for 1 year after stent implantation. Prophylactic statin therapy was commenced due to detection of non-critical coronary stenosis in other vessels

Conclusion

Although spontaneous coronary dissection is a rare cause of acute coronary syndrome and sudden cardiac death, it must be considered and investigated in cases with low atherosclerotic risk factors but typical anginal complaints, and IVUS imaging must be applied if necessary.

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Short communication

A case of atrial fibrillation leading to syncope after an electric injury in a patient with twin pregnancy

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ABSTRACT

The heart is one of the most affected organs during electric injuries. In electrical injuries, mechanical complications such as myocardial rupture, valvular rupture, pericardial effusion as well as a variety of arrhythmias ranging from electrocardiographic changes to ventricular fibrillation may occur. In this paper, we have presented a female patient with twin pregnancy at the 26th week of gestation, in whom syncope due to electric shock occurred and atrial fibrillation was detected on admission to the emergency department and have discussed treatment methods.

Introduction

In modern life, electrical accidents are becoming increasingly more frequent. In individuals exposed to electrical current, a variety of conditions ranging from burn lesions in soft tissues to cardiac complications, neurological damages and even death can occur.[1] In the present case report we aimed to draw attention to atrial fibrillation with rapid ventricular response in a patient with twin pregnancy at 26th week of gestation, who was admitted to the emergency department with complaints of loss of consciousness and subsequent palpitations.

Case report

A 26 year old female with twin pregnancy at 26th week of gestation according to the last menstrual period (LMP), was brought to the emergency department with complaints of loss of consciousness due to electric shock. Two hours prior to admission, the patient had been exposed to electrical current passing through the left hand because of touching the light source unit with a wet hand while taking her clothes out of the washing machine. The patient received an electric shock in her left hand and found unconscious by her relatives. On admission to the emergency department, the patient was conscious with ongoing palpitations. The patient had no cardiac risk factors or previous history of cardiac disease. The physical examination revealed that the patient was in good general health condition and conscious, with an arterial blood pressure of 110/70 mm Hg and an irregular pulse of 134/min. No pathology was detected in other system examinations. Laboratory tests revealed no abnormalities except for anemia (hemoglobin: 8.8 g/dl). Cardiac specific enzymes (CK, CK-MB, troponin I) were normal. Thyroid function tests showed no abnormalities. The electrocardiography revealed atrial fibrillation with rapid ventricular response (Fig. 1). Echocardiography revealed no pathology other than minimal tricuspid regurgitation. The patient was administered 2 l of O_2 per min., fluid infusions (150 ml/h). Anticoagulation was not considered. The patient was transferred to the intensive care unit and kept under monitoring. 5 mg of metoprolol was administered intravenously within 15 min. The rhythm was converted to sinus rhythm in 5 min after administration (Fig. 2). During her follow-up, no rhythm disturbances were observed. On 24-hour Holter recordings performed the next day, no atrial fibrillation and/or premature beats were detected. The patient was referred for gynecologic consultation and no problems which may threaten the pregnancy were detected. Three days later, the patient was discharged from the hospital without treatment.

Discussion

Electrocution is an uncommon cause of death and occurs commonly due to the accident. Electricity-caused occupational accidents were determined by certain authors to vary in a wide range from 26.47% to 81%.[2] The average age of the victims was 35.25 years with a significant prevalence of the male sex (74.07%).[2]

Electrical injuries can cause myocardial or valvular rupture, pericardial effusion, structural changes in coronary arteries whereas life-threatening arrhythmias and electrocardiographic changes such as temporary ST-segment elevation and QT prolongation are commonly encountered.[3] Arrhythmias caused by electrical injury include premature ventricular contractions, atrial tachycardia, sinus tachycardia, atrial fibrillation, ventricular fibrillation, ventricular tachycardia, bundle branch blocks

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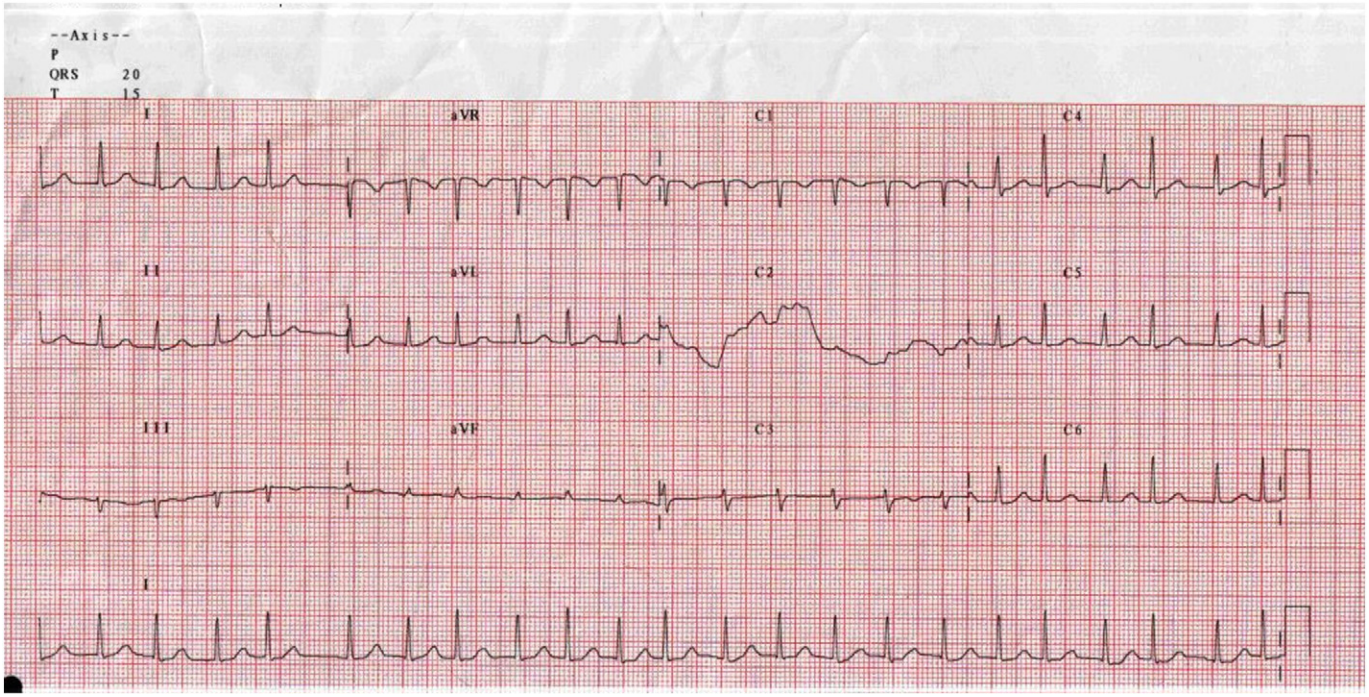

Fig. 1. Electrocardiography showing atrial fibrillation on admission to the hospital.

and complete heart block.[4] According to a study, the rate of arrhythmia development following electric shock ranges between 10–36%.[4,5] The electric current mainly affects the sinus node in the conduction system. In addition, it also exerts a direct effect on myocardial cells, leading to minor areas of myocardial necrosis, which may then undergo fibrosis and become arrhythmogenic foci.[5] The electric current also affects Na^+–K^+ pump activity, leading to an increase in K^+ contractions at cellular level, and thus, arrhythmias can be triggered.[4] However, electric current can cause a change in the myocyte membrane permeability.[6] In the present case, normal levels of cardiac specific enzymes suggest that atrial fibrillation may have resulted from the fact that sinus node had been affected, rather than because of myocardial damage.

The most common source of published data on accidental maternal electrocution, however, has often been cumulative case reports, which suggest a fetal mortality of 76%.[6] Fetal injuries have been reported to include sudden deaths, cardiac arrhythmias, intrauterine growth retardation, oligohydramnios, and abortion.[7] Several parameters have been proposed to influence the seriousness of fetal effects following

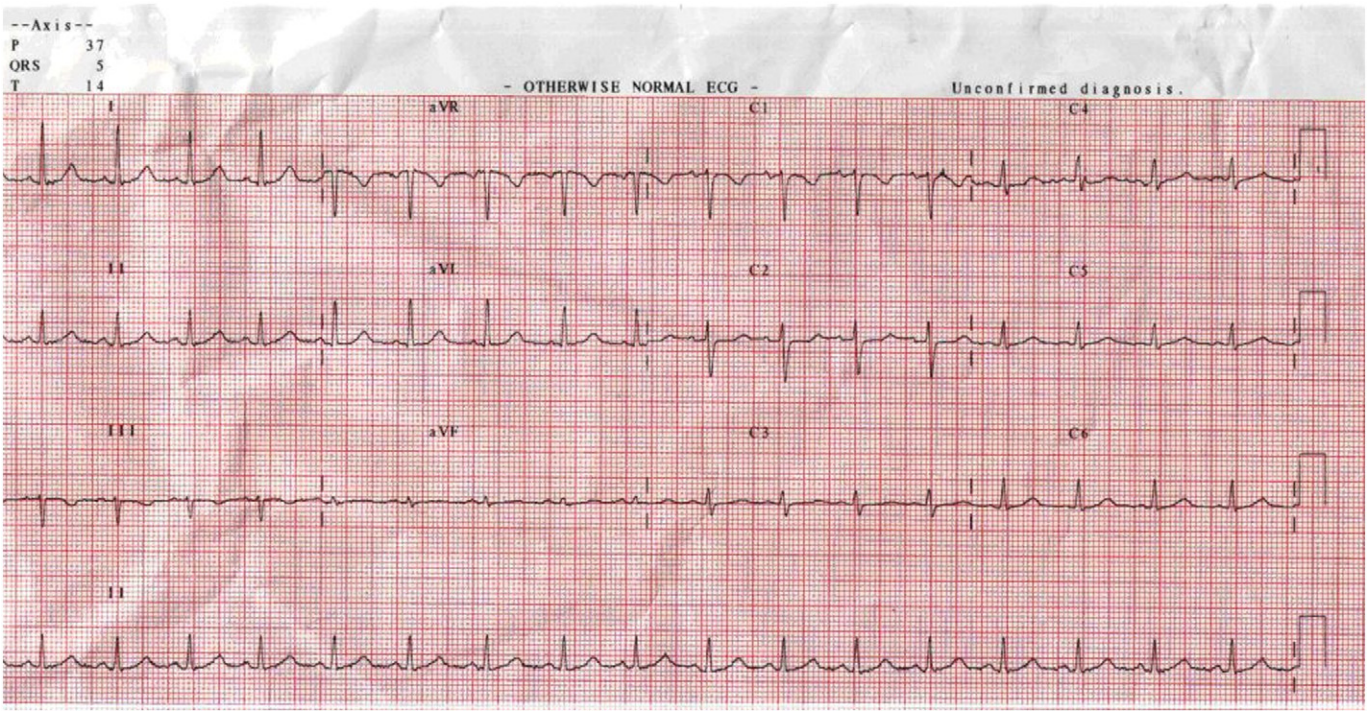

Fig. 2. Electrocardiography showing sinus rhythm after administration of 5 mg metoprolol infusion.

maternal electric shock. The characteristics of electricity, magnitude of current and the pathway of current flow both appear to be important determinants of fetal risk during accidental maternal electrocution. Besides amniotic fluid transmits current effectively, and this could increase risk of spontaneous abortions and fetal burns or death.[7,8]

Purdue et al., recommend cardiac monitoring for patients with loss of consciousness, recorded arrhythmia, abnormal electrocardiogram on admission and arrhythmia in the emergency department.[9] In the treatment of atrial fibrillation in pregnancy, the primary aim should be to restore normal sinus rhythm. In the absence of hemodynamic instability, digital, and beta blockers can be used. After irregular heartbeats are controlled with intravenous forms of these drugs, maintenance treatment can be initiated with oral forms of these drugs. However, cardioversion should be considered for the treatment of first choice in cases with hemodynamic instability. Anticoagulants should be used to avoid thromboembolic risk.[10] Since atrial fibrillation did not last more than 48 h we did not consider to anticoagulate the patient. Because the patient had normal hemodynamics, we preferred to perform pharmacological cardioversion after monitoring and used beta blockers as the drug of choice. The patient's normal sinus rhythm was restored in a short period of time and oral treatment was not planned because her heart rate was stabilized.

In conclusion, electrical injuries can cause arrhythmias such as temporary atrial fibrillation. The patient should be kept under close monitoring with respect to life-threatening arrhythmias such as syncope.

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Conflict of interest

No potential conflict of interest relevant to this article was reported.

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Short communication

Beeping ICD device: Case report

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ABSTRACT

A 56-year-old man with history of coronary bypass 6 years ago and ICD implantation 5 months ago was admitted to hospital after hearing a beeping from the ICD. On chest x-ray, the tip of the lead had migrated out of the heart silhouette. Percutaneous lead extraction was performed under close monitoring and fully equipped for pericardiosynthesis and resuscitation with on standby surgical backup. Although right ventricle perforation is a rare complication of pacemaker implants, regardless of the lead fixation mechanism, the possibility of perforation should always be considered.

Introduction

The expanding indications for cardiac pacemaker (PM) and implantable cardioverter defibrillator (ICD) therapy have led to a substantial increase in device implant for the last years. Lead perforation of the right ventricle (RV) is a rare and potentially lethal complication that may occur during, shortly or late after implant.[1–3]

Case report

We present a 56-year-old man with history of coronary bypass 6 years ago. The patient had ICD (Maximo II VR, Medtronic Inc. Minneapolis, USA) with dual-coil *active fixation lead* with *DF-4* terminal pins (Sprint Quattro, Medtronic Inc. Minneapolis, USA) implant 5 months ago for secondary prevention of sudden cardiac arrest. The patient was admitted to hospital after hearing a beeping from the ICD. Device interrogation showed a decrease in sensed R wave amplitude to 0.4 mV also RV pacing lead impedance decreased to 494 Ω from 780 Ω and pacing threshold increased to 5 V at 0.40 ms pulse width. There was also a lead integrity warning. The decline in lead impedance was suggestive of insulation defect, whereas the decrease in R wave sense and increase in pacing threshold was suggestive of a lead fracture. The chest x-ray demonstrated that the tip of the lead had migrated out of the heart silhouette. Computed tomographic (CT) scan confirmed the perforation and minimal pneumothorax (Fig. 1A–B). Transthoracic echocardiography revealed no pericardial effusion. Percutaneous lead extraction was performed in the coronary angiography room under close monitoring and fully equipped for pericardiosynthesis and resuscitation with on standby surgical backup. Soft stylet inserted in the lead and active fixation tip was retracted, then the lead was removed manually with simple traction through the left subclavian vein under fluoroscopic guidance. No complications occurred after removal. After a week, a new passive fixation ICD lead was implanted and post implantation chest x-ray and CT scan confirmed that the pacemaker lead was in RV apex. (Fig. 1C–D).

Discussion

Pacemaker lead perforation is a rare complication of pacemaker implantation, ranging from 0.1% to 0.8% in PM ventricular leads and 0.6–5.2% in ICD leads.[1] Acute lead perforation occurring during or soon after the procedure usually manifests as cardiac tamponade due to acute pericardial effusion, while late cardiac perforation can be asymptomatic. The late presentation is a less recognized complication but can have serious consequences if unrecognized. Lead perforation may be attributed to a combination of factors including patient characteristics, lead tip position, use of oral steroids, implant technique and the design characteristics of the lead.[4] Patients can admit with different symptoms; pericardial pain, dyspnea, syncope, ICD shock, poor sensing, pericardial effusion, hemothorax or like in our patient ICD alarm. ICDs have inbuilt alarm systems to notify patients of the need to seek assistance.

The data on how to manage delayed RV-lead perforation, whether to extract the lead or not, and if so, whether to extract perforating leads percutaneously or surgically, are very limited.[5,6] There are reported cases of tamponade occurring with late RV perforation treated with

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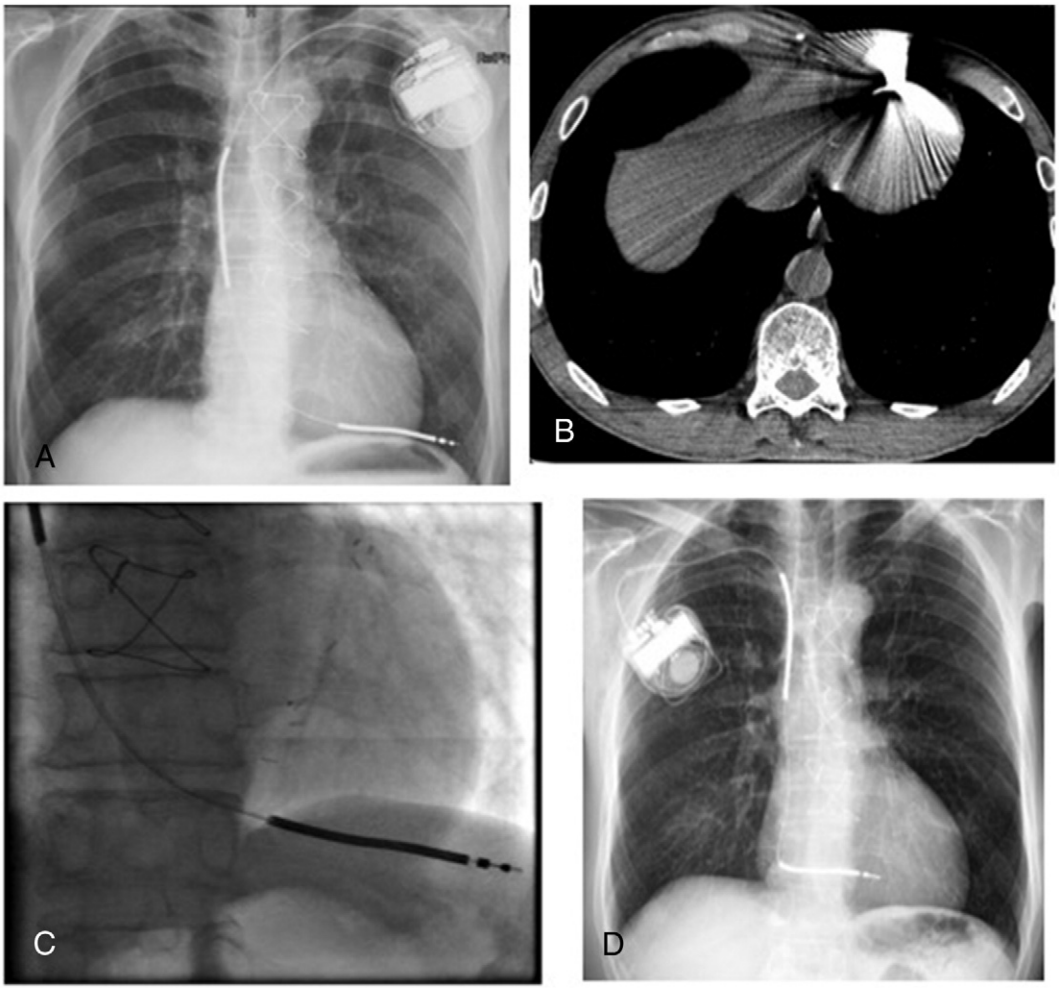

Fig. 1. (A) Chest x-ray demonstrating RV perforation by ICD lead. (B) Computed tomography confirmed RV-lead perforation. (C) Fluoroscopy showed displacement of the ICD lead (active fixation), with its tip located outside the cardiac silhouette. (D) The perforating lead was successfully removed by direct traction under fluoroscopy, in the absence of any complications and a new lead was placed.

percutaneous lead extraction by simple traction, but this complication is rare.[3,7] Not removing dysfunctional lead in a patient without infective endocarditis can sometimes be an option, however, veins can only accommodate a limited number of leads due to space constraints, and also it is more difficult to extract old leads after more time in the body.

In our case no complication accords after simple direct manual traction of the RV lead under fluoroscopic guidance. It may be possible that withdrawing the perforating lead did not cause significant bleeding in the pericardium because of the myocardium self-sealing properties and the pericardium might have been stiff due to previous cardiac operation. However, because of the possibility of acute life-threatening pericardial bleeding, the procedure should be carefully scheduled and a surgical backup should be mandatory.

Conclusion

As pacemaker lead perforation can be during or early after implantation, it can occur late. In most patients, percutaneous lead extraction by simple traction is a safe and effective management approach that may be performed in the electrophysiology room with surgical backup.

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Short communication

Renal failure and acute coronary syndrome due to use of Cannabis in a 26-year-old young male: A case report

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Introduction

The chemical substance cannabis, which is produced from the leaves of the plant *Cannabis sativa*, is commonly used by youth in Europe and in our country.[1] Although heroin, ecstasy, and cocaine have been known to be associated with adverse health effects, there is a common belief in society that cannabis is a relatively benign substance, just like alcohol and tobacco. In addition to its pleasure-inducing actions, *C. sativa* has also some side effects in various organ systems, particularly the cardiovascular system, and may sometimes threaten life.[2] In this paper, we reported a 26-year-old male patient presenting to emergency department with acute coronary syndrome and acute renal failure, who had used various forms of cannabis for a long time and had a number cardiovascular risk factors. The mechanism of action of cannabis was also discussed herein.

Case report

A 26-year-old male with no known chronic disease presented to the emergency department with exertional dyspnea and chest pain. He stated that his complaints had been recurring for 4–5 months but they had recently increased in severity and frequency. He had been smoking for 15 years and using illicit drugs for 12 years. Questioning about his medical history revealed no acute rheumatic fever or glomerulonephritis in the past. His family was free of coronary artery disease and the patient was not taking any medications on a regular basis. He admitted that he had used marijuana for 6 years, kubar (a substance preparation produced by pulverizing leaves and seeds of female *C. sativa* and mixing them with pulverized marijuana) for 4 years, and bonzai (a type of synthetic cannabinoid) for the last 2 years. His blood pressure was 160/100 mmHg and his pulse rate was 88 bpm. On physical examination, his skin was cold and pale. Cardiovascular examination revealed a 1/6 systolic murmur in aortic focus. Respiratory and other system examinations were normal. An ECG taken in the emergency department was also normal. Creatinine level was 9 mg/dL, urea was 173 mg/dL, and troponin level was 0.5 ng/dL. Troponin elevation was attributed to markedly abnormal renal function and the patient was taken into dialysis via a right femoral catheter. Following dialysis, he again had chest pain, and thus was consulted with the cardiology department. On ECG, there was a 0.5 mm St segment depression on leads DII, DIII and aVF (Fig. 1). Troponin level was found to be 0.7 ng/dL. Therefore, he was considered to sustain a high-risk unstable angina pectoris and admitted to coronary care unit. Mid and apical septal segments were hypokinetic on echocardiography. His left ventricular diameters increased and ejection fraction was around 50%. He also had mild mitral insufficiency. Clopidogrel 600 mg was loaded along with acetylsalicylic acid 300 mg, metoprolol 25 mg, enoxaparin 0.4 mg, atorvastatin 80 mg, and intravenous nitrate infusion. A coronary angiogram was performed, which showed a total occlusion in the right coronary artery (RCA) and a 50% stenosis with a haziness appearance in left anterior descending artery (LAD) (Fig. 2). Circumflex artery was normal. After balloon angioplasty 80% diffuse lesion appeared (Fig. 3). A stent was implanted to the RCA with complete vessel opening. LAD lesion was decided to be medically managed. Detailed laboratory tests were done during his intensive care stay. His fasting blood glucose was 76 mg/dl, and the lipid parameters were as follows: total cholesterol 282 mg/dL, LDL-C219 mg/dL, HDL-C 41 mg/dL, and triglyceride 107 mg/dL. Hemoglobin level was 7.9 and anemia was consistent with iron deficiency anemia. The patient underwent a regular dialysis program and was administered 1 unit of erythrocyte suspension. His medical therapy was arranged and he was transferred with a plan to visit the nephrology department for continuation of the dialysis program. A renal biopsy was scheduled but postponed due to fear of bleeding from dual antiplatelet therapy given after percutaneous coronary intervention. At the control examination 2 months after the first admission, he was still on the dialysis program.

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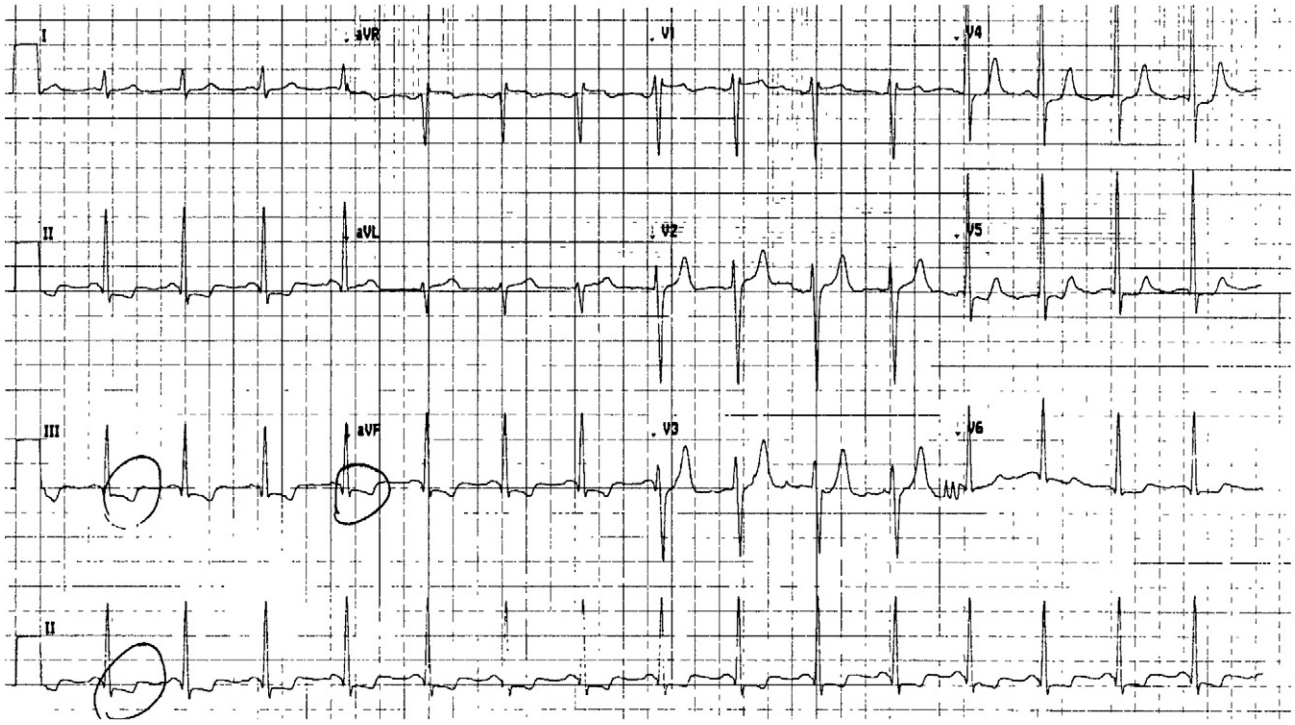

Fig. 1. Figure showing 1 mm downslopping ST segment depression on leads DII, DIII and aVF.

He had no chest pain or acute ECG changes. He quit using cannabis and smoking. He was on clopidogrel, acetylsalicylic acid, metoprolol, amlodipin, and statin medication.

Discussion

Cannabis is produced from leaves of the plant *C. sativa* and is known to be the most abused substance in rural and urban centers both in Europe and in our country. It has various preparations worldwide, including marijuana, hash, ganja, kubar, and bonzai. Its main effects are through cannabinoid receptors in the heart, brain, spleen, blood vessels, and immune system.[3] Δ9- tetrahydrocannabinol (THC) is the molecule that is responsible for pharmacological actions of cannabis. THC acutely exerts a vasoconstrictor effect and, additionally, cardiac ischemia as a result of an increase in cardiac workload, and postural hypotension. Similarly, increased blood carboxyhemoglobin levels are responsible for inadequate myocardial blood supply. In addition, cellular stress resulting from oxidant gasses activates thrombocytes and increases the amount of oxidized LDL and factor VII activity. Thus, it exerts chronic adverse cardiovascular effects by inducing inflammatory responses.[4] It has also been shown to induce thrombus formation within the coronary artery lumen.[4] For all these reasons, cannabis has been implicated for the development of coronary artery disease and triggering acute coronary syndromes.[4] As a result of these marked vascular actions, cannabis-containing cigarettes may worsen angina.[5] Literature data

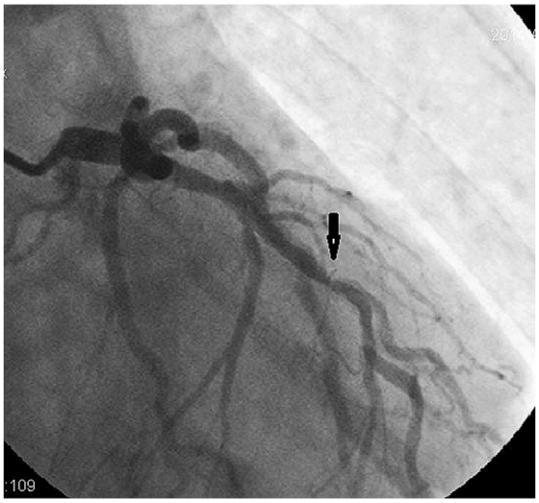

Fig. 2. 50% stenosis in left anterior descending artery with a hazziness appearance.

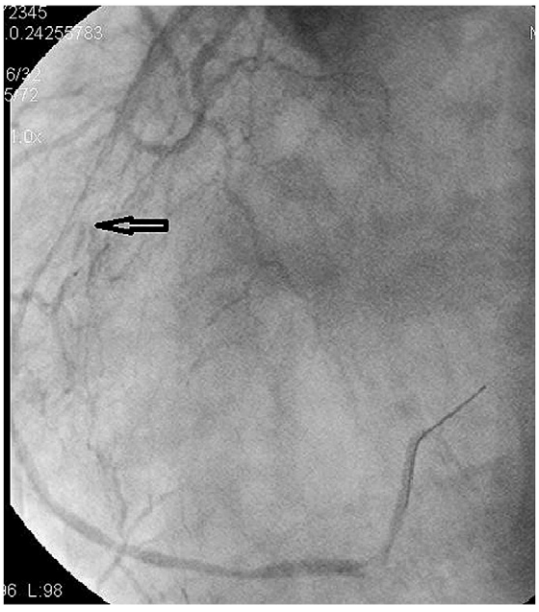

Fig. 3. Figure showing diffuse lesion appeared after baloon angioplasty in right coronary artery.

suggest that cannabis may trigger acute coronary syndrome in addition to marked sinus tachycardia and frequent premature ventricular depolarizations independent of whether atherosclerotic coronary artery disease is present or not.[6] Cannabis use can lead to severe cardiovascular events, not only in patients with traditional cardiovascular risk factors, but also in young people without any risk factors.[7] Our patient had used marijuana for 6 years, kubar for 4 years, and bonzai for 2 years. He presented to our clinic with acute coronary syndrome. We also think that the acute coronary syndrome was a direct result of cannabis consumption earlier that day in our case. It is known that cannabis increases heart rate and blood pressure in low doses, while it lowers the two in higher doses. Although our patient used bonzai on the day of admission, his hemodynamic profile was within normal limits.

Literature data about the renal side effects of cannabis is quite limited. It has been reported that cannabis formulations, especially its intravenous forms, cause transient nonoliguric renal failure.[8] Bhanushali et al. reported 4 cases of acute renal failure as a result of synthetic cannabinoid use.[8] Although we could not prove histopathologically, we considered that acute renal failure was chronic. And we think that low hemoglobin count was due to the chronic renal failure. The patient was included in the hemodialysis program and he was on hemodialysis at the latest visits.

There are various researches investigating the relationship between cannabis use and metabolic parameters. Muniyapa et al[9] and Berard et al[10] found lower HDL cholesterol levels in patients with cannabis abusers compared to the controls. LDL cholesterol levels were similar between cannabis abusers and controls in both of the investigations. In our case, LDL cholesterol levels were higher probably due to the genetic or dietary habits of the patient.

Conclusion

Cannabis use has been increasing worldwide. Thus, coronary heart disease, acute coronary syndrome, and acute renal failure as a complication of cannabis use are likely to increase in the future. We believe that our case suffered acute coronary syndrome and acute renal failure not only because of conventional atherosclerotic risk factors such as hyperlipidemia and smoking, but also possibly because he had used cannabis for an extended period before admission. Use of cannabis or other illicit drugs should always be questioned in young cases presenting with acute coronary syndrome and/or acute renal failure.

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Conflict of interest

No potential conflict of interest relevant to this article was reported.

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