enhancement of a systemic inflammatory and pro-coagulable state with further surgical intervention after TAVI may trigger valve thrombosis. In our case report, in spite of our initial suspicion of valve endocarditis, the prosthetic mass disappeared without residual prosthetic damage and after a short period of antibiotic treatment and heparinization. Additionally the location of thrombus is mostly in aortic prosthetic valve. This particular finding could suggest that the initially diagnosed mass was, more than a prosthetic valve endocarditis, a valve thrombosis.

Conclusion

In conclusion, in the light of the patient's complex comorbid profile, prosthetic valve endocarditis after TAVI is a medical challenge. Mimicking conditions, such as valve thrombosis secondary to inappropriate anti-aggregation, should be ruled out and eventually treated before embarking in more complex forms of intervention.

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Video 1. A mobile 18x7mm mass on the CoreValve® prosthesis via transesophageal echocardiography
Video 2. Ten days after initiation of antibiotic treatment and heparinization showing disappearance of the mass without any residual structural lesion of the CoreValve ® prosthesis via transesophageal echocardiography

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A favorable outcome of a post-myocardial infarction ventricular septal rupture

Miyokart enfarktüsü sonrası ventriküler septal rüptürün olumlu sonucu

Introduction

Usually, the ventricular septal rupture is a devastating complication of the myocardial infarction (1), leading to death, in case of the unoperated patient. Additional investigations are essential in the correct hemodynamic assessment, especially the first introduced in clinical practice for this disease, the echocardiography (2). Long-term mortality is reduced if the patient with acquired ventricular septal defect is emergently operated, if there is significant hemodynamic alteration (3).

The aim of our study is to reveal the spontaneous, rare evolution toward healing of a ventricular septal rupture, acquired after a myocardial infarction.

Case Report

A-45-year- old patient, previously diagnosed with Wolf-Parkinson-White syndrome (5 years ago), was hospitalized with subacute anteroseptal myocardial infarction (September 2011). One week ago, after excessive alcohol consumption, he had chest pain for 6 hours, exacerbated by physical effort. At the moment of admission, the serum biomarkers for myocardial infarction were normal, as well as other laboratory data, with the exception of the gamma-glutamil-transpeptidase - 150 IU/l (normal values: <40 IU/l). He also had echocardiographic kinetic changes-dyskinesia of the anteroseptal wall, hypokinesia of all the other left ventricular walls, ejection fraction - 30%. The electrocardiogram revealed only the Wolf-Parkinson-White syndrome: the delta wave was hiding the Q-waves because the conduction was via the accessory pathway, as Brackbill et al. (4) also remarked. He was conservatory treated (delayed admission – after one week). The epicardial coronary arteries were normal at angiography (the vasospasm was the incriminated mechanism for myocardial infarction). After discharge, he interrupted the medication and he performed inadequate physical efforts. He underwent a cardiological examination in October 2011 (4 weeks after the first admission), for small efforts dyspnea and palpitations. He had a left parasternal systolic murmur, produced by a ventricular septal defect, revealed by Doppler echocardiography (Fig. 1). Left ventricle was dilated with an altered ejection fraction (30%); this diminished myocardial contractility was explained by chronic alcohol consumption (50g per day; elevated gamma-glutamil-transpeptidase: 125 IU/l, normal values<40IU/l) and by the associated hypothyroidism (thyroid stimulating hormone: 7 IU/l, normal values: 0.5-4.5 IU/l). His electrocardiography presented the same aspect as 5 years ago: Wolf-Parkinson-White syndrome (Fig. 2). The patient refused the electrophysiological studies for ablative therapy. Repeated episodes of paroxysmal supraventricular tachycardia were detected on 24 hours electrocardiographic Holter recording. The medical recommended treatment consisted of: acetylsalicylic acid 100 mg/day, ramipril 5 mg/day, atorvastatin 80 mg / day, levothyroxine 100 µg/day. He did not come to reevaluation for one year, even if he was invited to an examination every month. In October 2012, it was an unexpected surprise to find that he had no systolic murmur at physical examination. Doppler echocardiography revealed that there was no ventricular septal defect anymore (Fig. 3). The same aspect of fibrotic scar with no

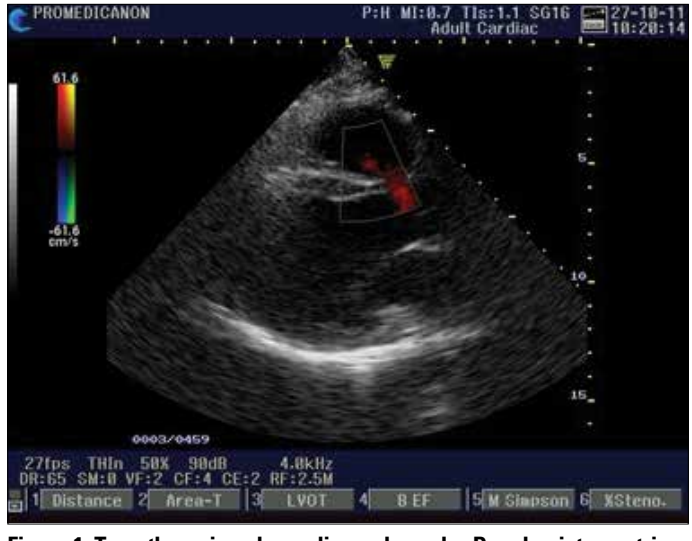

Figure 1. Transthoracic echocardiography: color Doppler, interventricular subaortic communication

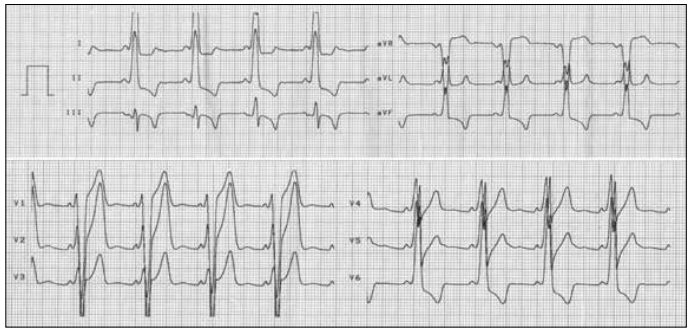

Figure 2. Electrocardiogram (Wolf-Parkinson-White syndrome)

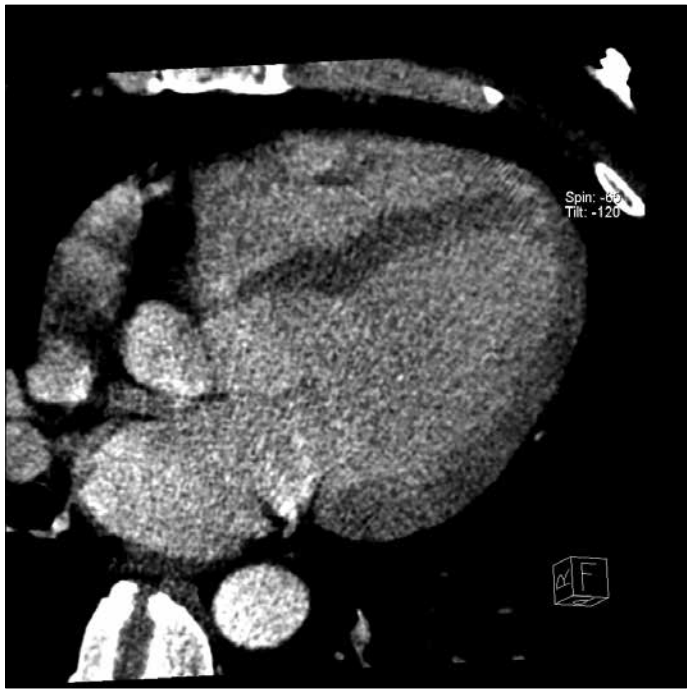

Figure 4. Thoracic computed tomography angiography scan: fibrotic scar of the subaortic ventricular septum

interventricular communication was documented through 3D echocardiography and transesophageal echocardiography (Video 1. See corresponding video/movie images at www.anakarder.com). Our team also performed a thoracic computed tomography angiography, which revealed the same fibrotic scar of the subaortic ventricular septum (Fig. 4). He still had arrhythmias and the echocardiographic aspect of dilated cardiomyopathy, but his effort tolerance was better. The patient respected the recommended cardiological treatment, but he continued to consume alcohol on a daily basis and refused to follow the endocrinological treatment for hypothyroidism.

Discussion

The spontaneous closure of post-infarction ventricular septal rupture is very rare. Only 5 patients were reported (5-7) until the presented case. The mechanism of the closure in these 5 cases was the formation of a clot. At the presented patient, the mechanism was probably the proliferation of the fibrous tissue, proven by the echocardiographic aspect of the damaged ventricular septum, after one year of evolution. This mechanism was mentioned as leading to a spontaneous closure of the congenital ventricular septal defects in young and middle-aged adults (8); therefore, it can be similar for the acquired post-infarction ventricular septal defect, where the clot was not involved in this closure.

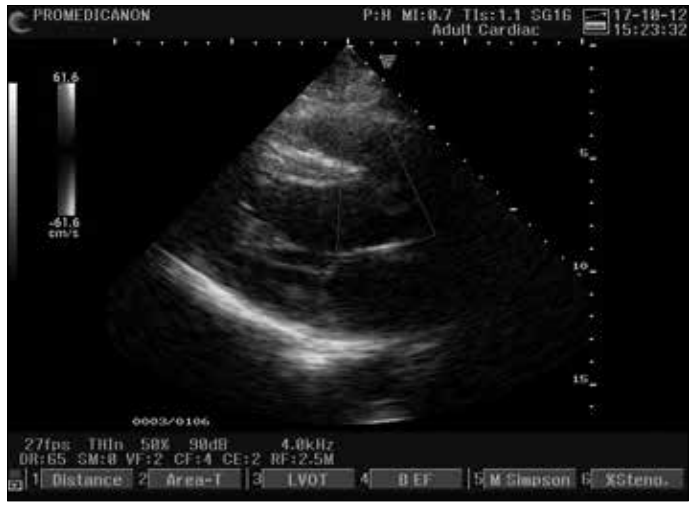

Figure 3. Transthoracic echocardiography after one year: no interventricular communication at rupture site

Conclusion

The case presented rare physiopathological mechanisms for the associated pathologies and their evolution: a myocardial infarction with normal coronary angiography, a mechanical complication of the myocardial infarction (ventricular septal rupture, spontaneously closed), recurrent arrhythmias, chronic alcohol consumption and hypothyroidism.

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Disclosure
The authors declare that they have no potential conflicts of interest to disclose.

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Video 1. 3D echocardiography and transesophageal echocardiography: fibrotic scar, without interventricular communication

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