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Suspected endocarditis after CoreValve® implantation: a word of caution

CoreValve® implantasyonu sonrası ortaya çıkan şüpheli endokarditis: Değinilmesi gereken bir uyarı

Introduction

Transcatheter aortic valve implantation (TAVI) has been recently popularized as a safe and valuable alternative in patients deemed at too high risk for conventional surgery. Management of suspected endocarditis after TAVI should be pondered and surgical intervention should be advocated only when the diagnosis is certain and conventional medical treatment has failed. We herein report a case of suspected prosthetic valve endocarditis resolved after adequate medical treatment.

Case Report

An 80-year-old male patient was referred to our Institution for fever of unknown origin, disorientation, and recent onset of dyspnea (Euroscore: 10). Four months previously he had undergone a transfemoral TAVI with a CoreValve® prosthesis (Medtronic Inc., Minneapolis, Minnesota, USA). After the procedure he was discharged on oral double anti-platelet therapy (aspirin and clopidogrel) as prevention for valve thrombosis. He was then submitted 2 months later, in another hospital, to a trans-urethral prostatectomy and cystostomy for a benign prostate hypertrophy. In order to perform the procedure, the anti-aggregation was interrupted, no prophylactic heparin was given, and single anti-aggregation (aspirin) was restarted few days later. Infective endocarditis prophylaxis was administered before the procedure.

At admission the patient presented in good hemodynamic compensation. Standard blood and urine analysis documented elevated phlogosis indexes (C-reactive protein 13.0 mg/dL) and leucocyturia (WBC 500/µl). Blood cultures resulted positive for Enterococcus faecalis and a targeted antibiotic therapy with vancomycin was started. In spite of maximal medical treatment, the patient remained febrile. At this stage, a transesophageal echocardiography (TEE) was performed documenting a mobile 18x7mm mass on the CoreValve® prosthesis (Fig. 1. Video 1. See corresponding video/movie images at www.anakarder.com). Heart and prosthesis function were within normal limits. After collegial discussion with the cardiac surgeons, and in consideration of the high surgical risk profile of the patient (Euro-SCORE: 45%), it was decided to manage the condition medically. To exclude the possibility of a prosthesis thrombosis, a therapeutic regimen of sub-cutaneous low molecular weight heparin was coupled with the oral aspirin. The patient improved slowly and a control TEE performed 10 days after initiation of the targeted antibiotic therapy showed complete resolution of the mass and confirmed normal function of the prosthesis (Fig. 2. Video 2. See corresponding video/movie images at www.anakarder.com). After 6 weeks of antibiotic therapy, a second TEE documented a normally functioning prosthesis without any vegetation and without signs of structural degeneration or lesion. The patient was eventually discharged home in good hemodynamic and general condition.

Discussion

Endocarditis after TAVI has been previously reported and successfully treated with conventional surgery (1). It should be emphasized that in high-risk patients previously rejected to surgery, emergent intervention for eradication of prosthetic endocarditis carries a heavy morbidity and mortality burden. For this reason, certainty of the diagnosis and adequate medical treatment should be advocated before referring the patient for a more invasive intervention. Furthermore, the endocarditis prophylaxis should be administered before surgery and emergent intervention in patient after TAVI.

Moreover, the adequate anti-aggregation regimen for patients after TAVI remains controversial. Discontinuation of anti-aggregation and

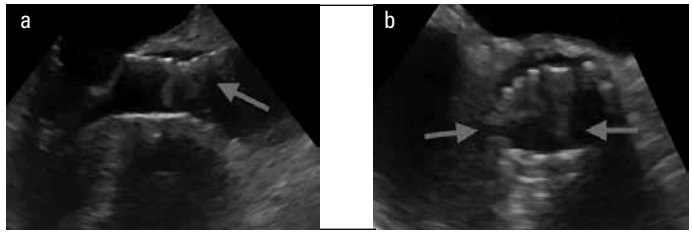

Figure 1. Transesophageal echocardiography showing a mass (18x7mm) on the CoreValve ® prosthesis (white arrows). a) long-axis view; b) short-axis view

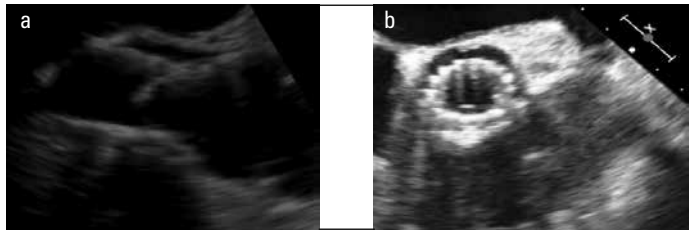

Figure 2. Transesophageal echocardiography 10 days after initiation of antibiotic treatment and heparinization showing disappearance of the mass without any residual structural lesion of the CoreValve ® prosthesis (white arrows). a) long-axis view; b) short-axis view

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

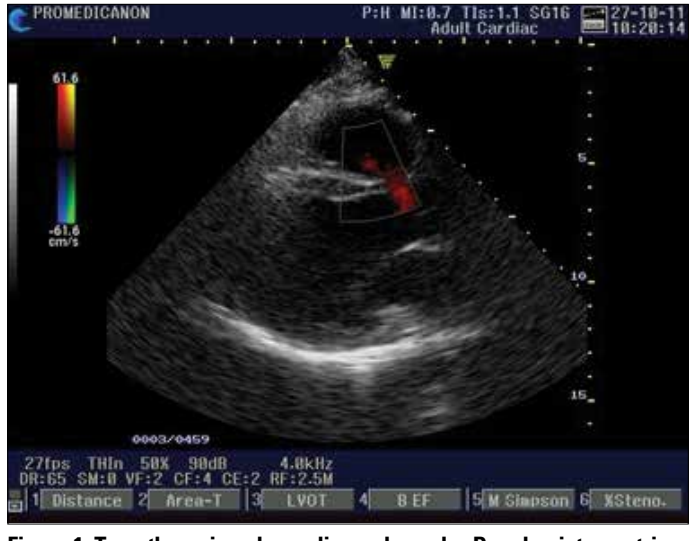

Figure 1. Transthoracic echocardiography: color Doppler, interventricular subaortic communication