

AS was of rheumatic cause, with commissural fusion and little calcification. The aortic annulus and sinuses of Valsalva diameters were 22 and 30 mm, respectively. Systolic pulmonary artery pressure was 60 mm Hg. Coronary angiography showed normal epicardial coronary arteries, the calculated logistic EuroSCORE was 21. She was declined for surgery on the basis of prior cardiac surgery and poor left ventricular function.

The technique was similar to that described by the previous case. During deployment, accelerated right ventricular pacing with 140 bpm and an oversized (29 mm) CoreValve prosthesis were used. Only one attempt was necessary to achieve the optimal result without any technical issues (Video 5, 6. See corresponding video/movie images at www.anakarder.com). Follow-up echocardiography showed a well functioning prosthesis, with a mean gradient of 8 mm Hg, respectively. Mild paravalvular leak was present. The patient was clinically stable at 30 days follow up after the procedure.

Discussion

The use of TAVI is considered a relative contraindication in non-calcified valves (5). Calcium seems mandatory for anchoring the stent-valve and prevent pop-out, dislocation and migration of the prosthesis. In rheumatic AS, there is little or no calcification. However, our cases show that TAVI could be safe, feasible and effective treatment in patients with rheumatic AS.

The concept of TAVI is based on crushing the usually heavily calcified native valve leaflets against the aortic wall by implanting a metallic stent-frame. Since calcification of the native valve leaflets is presumably essential for fixation of the stent-frame, TAVI is indicated in patients with calcified AS. Indeed, TAVI in patients with only marginal annular calcifications may lead to dislocation of the bioprosthesis into the left ventricle (2, 4). The unique pathological features of rheumatic AS, with lack of calcium, commissural fusion and pliable leaflets, can make it unsuitable to TAVI.

The CoreValve prosthesis might anchor solidly even in the absence of calcification when oversized due to engineering properties (2), and may offer treatment for rheumatic AS without dislocation and migration of the prosthesis. During deployment, to prevent pop-out, embolization and migration of the prosthesis we performed accelerated right ventricular pacing between 140-180 bpm. In addition, oversized, self-expandable (CoreValve) valves were selected.

Conclusion

This report shows that TAVI could be safe, feasible and effective treatment in patients with rheumatic AS in selected no-option patients. Embolization of the valve may become an issue, and could be a drawback to this approach.

Video 1. Transesophageal echocardiography demonstrates thickening and commissural fusion of the aortic valve with little calcification in basal short-axis view in case 1

Video 2. Transesophageal echocardiography demonstrates thickened and little calcified aortic valve and concomitant rheumatic involvement of the mitral valve in long-axis view of the left ventricle in case 1

Video 3. Positioning of the CoreValve in the aortic valve annulus in case 1

Video 4. Aortography shows mild aortic regurgitation after deployment of the CoreValve in case 1

Video 5. Positioning of the CoreValve in the aortic valve annulus in case 2

Video 6. Aortography shows mild aortic regurgitation after deployment of the CoreValve in case 2

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A case of unusual looking prosthetic mitral valve thrombosis treated with low dose slow infusion tPA

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Introduction

Prosthetic mitral valve thrombosis (PVT) is a serious complication of valve replacement which carries a high risk of mortality. However, the optimal treatment method for PVT remains controversial. Here, we report a case of PVT with an echoluscent-structured thrombus diagnosed on a mechanical mitral valve and the use of a low-dose tissue plasminogen activator (tPA).

Case Report

A 37-year old woman was admitted to our clinic with complaints of respiratory distress and fatigue. Her history included mitral valve replacement surgery a year ago due to rheumatic heart disease. The

patient, who had been using warfarin and receiving regular INR testing, had targeted INR values ten days ago. A week ago, she used oral prednisone for lower back pain which she had stopped taking two days ago. On physical examination, rales were heard bilaterally on the basal regions of the lungs. The haemoglobin and leukocyte count were normal. The erythrocyte sedimentation rate was 60 mm/hour, CRP was 40.8 mg/L (0-6 mg/L) and INR was 1.58. Transthoracic echocardiography (TTE) revealed a mobile non-obstructive mass attached to the mechanical mitral valve. The maximum and mean transmitral diastolic gradients were determined to be 24/10 mm Hg, mitral valve area was measured as 1.4 cm², and it was assumed that the high gradient was related secondary to the high rate. Patient blood samples were obtained for microbial analysis, and antibiotic treatment directed at infective endocarditis (IE) was administered. Subsequently, transesophageal echocardiography (TEE) was performed. A 14x9 mm sized non-obstructive, echoluscent-structured, mobile mass was detected which was attached to the annulus posterior to the prosthetic mitral valve and mild physiological mitral insufficiency was observed (Fig. 1, Video 1. See corresponding video/movie images at www.anakarder.com). The medical council concluded that the mass on the prosthetic valve was compatible with a newly formed thrombus, and that thrombolytic therapy (TT) should be applied to the patient. The patient was administered an intravenous infusion of 25 mg tPA in 12 hours. The 24 hour control TEE revealed that the thrombus had disappeared, and that in its place only a residue was visualized (Fig. 2, Video 2. See corresponding video/movie images at www.anakarder.com). No complications occurred and the patient was discharged after the antiaggregant therapy was re-regulated.

Discussion

PVT is a rare but serious complication of valve replacement. The most frequent cause of PVT is insufficient anticoagulation. Surgical technique, type of valve used, localization of the valve, and presence of a pannus and the hemodynamic situation of the patient are other important reported causes of thrombus formation on mechanical valves (1). Although PVT can present acutely as a fresh thrombus, it is most often a subacute or chronic phenomenon. Thrombi are typically formed of different clot layers, with varying degrees of organisation (2). TEE is the gold standard in the confirmation of diagnosis (3). Since the margins of the thrombus were prominent and the internal structure was translucent on TEE, we concluded that the thrombus had newly formed.

Different recommendations for the surgical or TT of PVT are given in the guidelines. Since TT has a risk of embolism, the 2007 European valvular heart disease guideline primarily recommends surgical treatment. According to these guidelines, TT is recommended only in PVTs of the right side, and in left side PVTs that are inoperable (4). In the guideline prepared by Lengyel et al.(5), in obstructive PVT, the rates of success, systemic embolism and mortality of TT treatment were reported to be 82%, 12% and 6% respectively, and if not contraindicated, it was recommended that TT be the first step in therapy. It has been reported in literature that TT should not be performed in the presence of left atrial thrombus due to the high risk of embolism (5). Furthermore, a multicenter PRO-TEE study has reported that the risk of embolism was higher with TT in patients with a thrombus area of >0.8 cm² (6).

There is no consensus on the proper dose and duration of TT in PVT. In the high-series study of Özkan et al. (7) (TROIA study), different TT protocols were compared in patients with PVT, and no difference

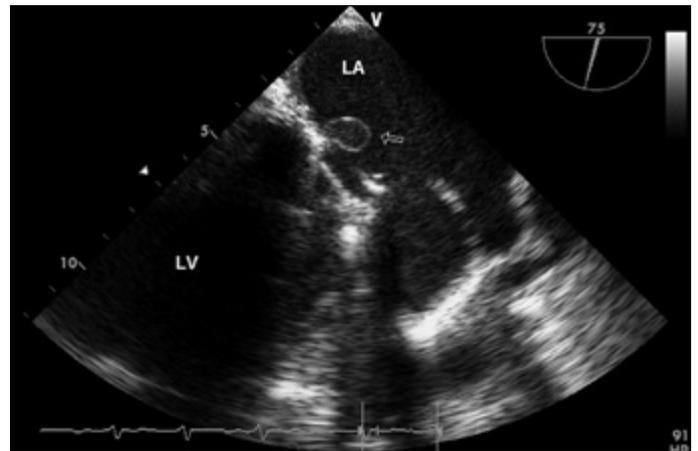


Figure 1. TEE on admission: Nonobstructive, echoluscent-structured mobile mass was detected which was attached to the annulus posterior to the prosthetic mitral valve

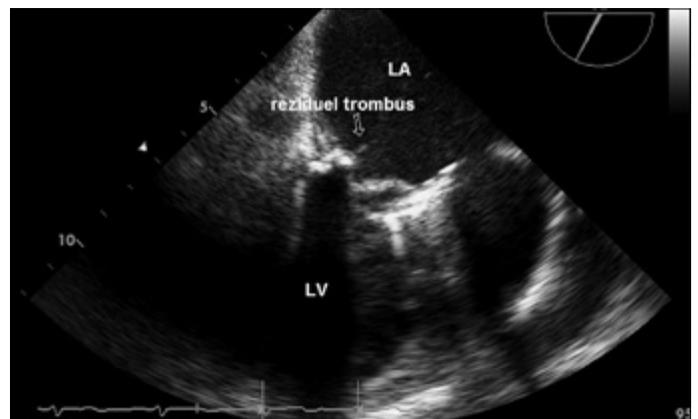


Figure 2. TEE after treatment: The thrombus had disappeared, and in its place only a residue was visualized

was observed between the success rates of different agents (streptokinase, tPA). Moreover, the lowest complication rate was reported in the low-dose and slow infused (25 mg/6 hours) tPA group. In the study, repeated infusions of were required in the majority of patients given low-dose tPA. In our case, the very short duration of subtherapeutic warfarin use and dissolution of the thrombus after a single dose administration of tPA 25 mg supported our assumption that the thrombus was a very newly formed one.

Conclusion

This case was presented in order to highlight the effective and reliable treatment option of low-dose TT by slow infusion in patients with PVT with special thrombus morphology.

Video 1. TEE on admission: Nonobstructive, echoluscent-structured mobile mass was detected which was attached to the annulus posterior to the prosthetic mitral valve

Video 2. TEE after treatment: The thrombus had disappeared, and in its place only a residue was visualized

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Misleading aspect of left atrial appendage membrane: an incidental echocardiographic finding

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Introduction

The left atrial appendage (LAA) is a blind-ending, muscular extension of the left atrium and is of clinical importance in as much as the LAA is a place where a thrombus could be formed when the left atrial (LA) function decreases (1). However, it should routinely be analyzed as part of a transesophageal echocardiographic (TEE) examination (2). The LAA cavity might very rarely have membranes. Indeed, only less than ten cases of a membrane involving the LAA have been described in the literature. The origin of membranes involving the LAA is not clear. The most likely explanation for the origin of these membranes would appear to be a congenital anatomic variation (3).

We report a case of a non-obstructive membrane at the orifice of the LAA on TEE, mimicking a mobile thrombus attached to it.

Case Report

A 42-year-old woman, with no history of cardiovascular disease, presented with palpitations and dyspnea. A 12-lead electrocardiogram

showed atrial flutter with an acceptable ventricular rate, and a two-dimensional echocardiogram was normal except for a mildly dilated LA. A pre-cardioversion TEE examination illustrated a linear, membrane-like structure traversing the orifice of the LAA (Fig. 1, Video 1. See corresponding video/movie images at www.anakarder.com). Color Doppler did not demonstrate flow acceleration across this membrane (Fig. 2, Video 2. See corresponding video/movie images at www.anakarder.com). Pulsed-wave Doppler confirmed low-flow velocities across the membrane, indicating no obstruction (Fig. 3) but a mobile linear particle (4 mm) mimicking a thrombus attached to the LAA. Accordingly, the mobile particle structure was considered thrombosis and anticoagulation therapy before cardioversion was recommended.

After six weeks with the patient on good anticoagulation, a second 2D and 3D-TEE examination yielded similar images and configurations (Fig. 4, Video 3, 4). The moving particle attached to the LAA membrane was, therefore, deemed a structural variant, and electrical cardioversion was performed successfully. After electrical cardioversion, the patient recovered sinus rhythm and was discharged on standard therapy.

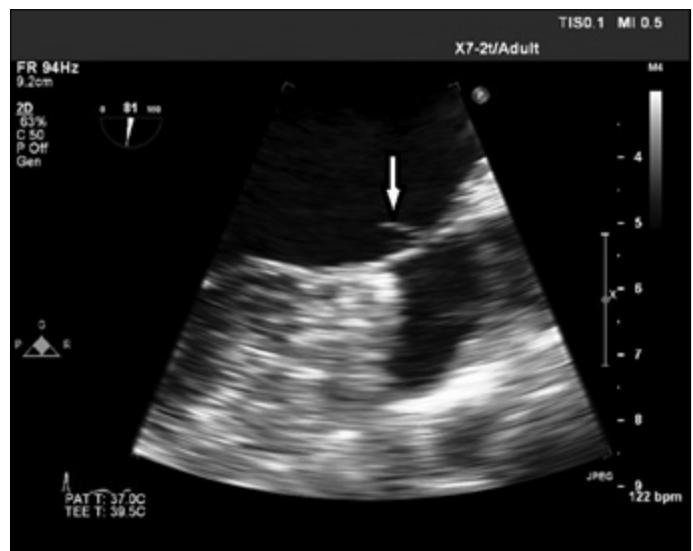


Figure 1. A membrane-like structure traversing the orifice of the LAA with a mobile linear particle mimicking a thrombus attached to the membrane (white arrow)

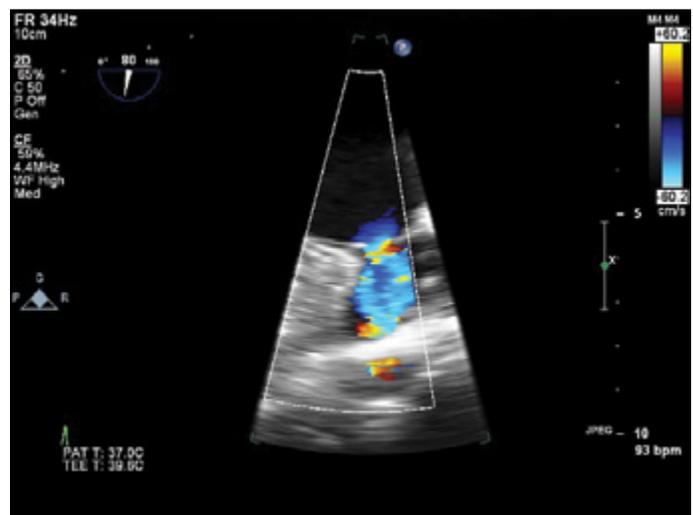


Figure 2. Color Doppler study demonstrates no flow acceleration across the LAA membrane