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Mitral Prosthetic Valve Obstruction and Its Complications

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ABSTRACT

Prosthetic Valve Obstruction (PVO) is a serious complication which is associated with increased morbidity and mortality. This could result from thrombus formation, development of pannus, or a combination of both. Patients with this complication often present with symptoms and signs of heart failure, systemic embolism, acute cardiovascular collapse, and sudden death. Transesophageal echocardiography and cine fluoroscopy play a vital role in diagnosis of this potentially lethal condition. Herein, we reported a 56-year-old male patient who presented with severe heart failure and was found to have obstructed ATS27 bileaflet mitral prosthetic valve. Thrombolysis and redo surgery are two important options for treating this condition although guidelines for choosing between the two are not very definite.

►*Implication for health policy/practice/research/medical education:*
PVO prognosis and outcome depend on early diagnosis of the complication and prompt initiation of adequate therapeutic procedures. At present, the right decision regarding the treatment options depends on the decision of the heart team, including a cardiac surgeon, an interventional cardiologist, an echo specialist, and an anesthesiologist. At times, lack of proper road map in treatment of PVO puts the treating doctor in agony. This case report gives a clear idea about how to approach a PVO.

1. Introduction

Patients with prosthetic valve experience various types of complications. Prosthetic Valve Obstruction (PVO) is associated with high morbidity and mortality (1, 2). PVO occurs due to thrombus formation, pannus formation, and pannus, thrombus and vegetation in 54%, 6%, and 39% of the cases, respectively (3). PVO depends on several factors, including inadequate anticoagulation, type of valve, position, atrial fibrillation, ventricular dysfunction, hypercoagulable condition, and previous thromboembolism (3). Prosthetic Valve Thrombosis (PVT) is mostly seen in mechanical prosthesis, but pannus may be seen in both mechanical and bioprostheses (4). PVT has an incidence rate of 0.1 - 6% per patient in left sided valves and up to 20% in tricuspid valves (5). Bioprosthetic valves are less prone to thrombosis; only 0.03% per year mainly seen in the first months following surgery (6, 7).

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2. Case Presentation

A 56-year-old male driver known to have Rheumatic Heart Disease (RHD), Mitral Stenosis (MS) post Mitral Valve Replacement (MVR) with 27 ATS bileaflet prosthetic valve on 29th Oct 2007, Coronary Artery Disease (CAD), post Coronary Artery Bypass Graft (CABG), Atrial Fibrillation (AF), Transient Ischemic Attack (TIA) in Jan 2007, and mild Pulmonary Artery Hypertension (PAH) was admitted with the complaints of progressive dyspnea for 2 weeks duration, New York Heart Association (NYHA) Class III-IV, and pedal edema. The patient had no past history of Type II Diabetes Mellitus (T2DM), systemic hypertension, and dyslipidemia. He also had no family history of CAD, did not smoke, and was not ethanolic.

On examination, he was conscious, oriented, and afebrile. He showed no angina, mild dyspnea, elevated JVP, pedal edema, heart rate of 70 beats per minute, blood pressure of 130/80 mmHg, respiratory rate of 24 per minute, and saturation of 98%. His other features included CVS: S1 and S2 present, pan systolic murmur: 2/6, and Chest: bibasal crepitations present.

2.1. Investigations

The patient's laboratory evaluation showed Hb: 14.2 g/dL, Na: 148 mEq/L, K: 3.4 mEq/L, Urea: 60 mg/dL, Creatinine: 3.6 mg/dL, HbA1c: 6.2%, TSH: 1.3 micro IU/mL, BNP: 214.9 ng/L, PT: 18.8 sec, and INR: 1.38. Besides, the results of chest X-ray showed mild cardiomegaly and enlarged LA shadow in shadow. The findings of electrocardiogram were as follows: normal sinus rhythm, rate 80/min, no significant ST-T changes, and normal QTc. Additionally, transthoracic echocardiography revealed good Left Ventricular (LV) systolic function, no regional wall motion abnormality, dilated left atrium (EF = 55%), prosthetic valve at the mitral position, mitral valve area by planimetry of 0.86 cm^2, mitral valve area by PHT of 0.9 cm^2, mild to moderate mitral regurgitation, mild aortic regurgitation, mild pulmonary hypertension, right ventricular systolic pressure (45 + 10 = 55 mmHg), no clots or effusion, intact IAS/IVS, and normal inferior venacava. Transoesophageal echocardiography also showed good LV systolic function, no regional wall motion abnormality, dilated left atrium (EF = 55%), and bileaflet prosthetic valve at the mitral position. In addition, one disc of prosthetic valve was fixed and did not move at all (Figure 1).

Mitral valve area by planimetry was estimated as 0.88 cm^2, with mild mitral regurgitation, mean gradient = 18 mmHg, aortic valve thickened, mild aortic regurgitation noted, severe tricuspid regurgitation present, right ventricular systolic pressure = 80 mmHg, intact IAS/IVS, and no clots or effusion. Spontaneous echo contrast was seen in the left atrium. The results of fluoroscopy were as follows: mitral ATS bileaflet valve in situ, one moving leaflet, and the other leaflet stuck in the closed position (Figure 2). Besides, coronary angiogram indicated normal Left Main Coronary Artery (LMCA). However, Left Anterior Descending (LAD) coronary artery showed 30% proximal stenosis. Diagonal (D) 1 and 2 were normal. Left Circumflex (LCx) and right coronary arteries were normal, as well. On the

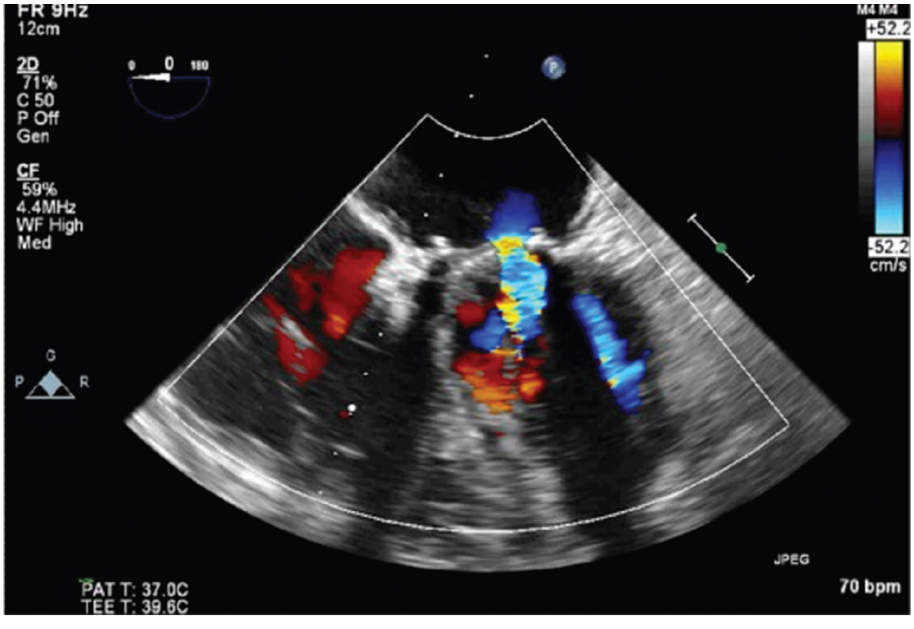

Figure 1. Transoesophageal Echocardiography. Fixed and Immobile Leaflet of Prosthetic Valve at Mitral Position with Mild Mitral Regurgitation

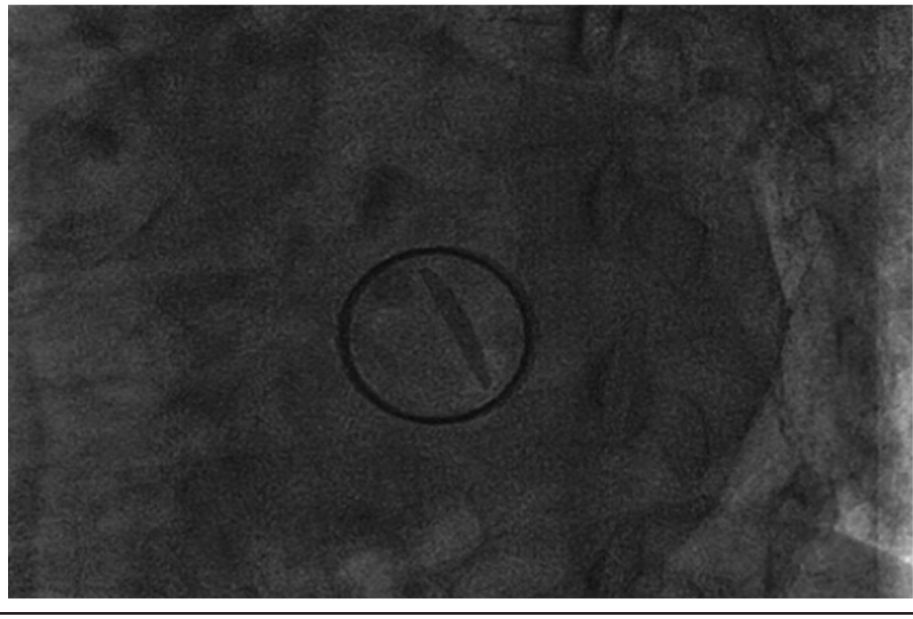

Figure 2. Stuck Mitral Prosthetic Leaflet, One in Open and the Other in Closed Position

other hand, saphenous vein graft to LAD had 30% stenosis.

2.2. Treatment

Ascending aorta and both vena cava were cannulated and cardiopulmonary bypass was established. The sewing cuff of the removed ATS valve was covered with pannus and red organized thrombus was observed on both sides of the valve (Figure 3). MVR was done with #25 tissue valve and the patient was weaned off the cardiopulmonary bypass. After that, intra-aortic balloon pump support was initiated. The postoperative course was uneventful.

3. Discussion

The ATS valve is a pylolytic carbon, open-pivot, and low-profile cardiac valve prosthesis. Its two leaflets are constructed to rotate on spheres projecting into the orifice. The mechanism of the valve causes blood stasis or turbulence. A multicenter study on the ATS valve demonstrated 2.1% early deaths in 1.8 ± 1.3 years, while this measure was obtained as 96% in the Aortic Valve Replacement (AVR) and 94% in the MVR groups at 5 years (8).

3.1. Thrombosis

PVT is the commonest cause of obstruction in prosthesis, which usually involves non-infective thrombotic material. The pathogenesis of PVT may be due to molecular interactions between artificial surface of the valve and corpuscular blood components, plasma, etc. The process starts with attachment of fibrinogen to the valve surface followed by platelet adhesion. The transprosthetic blood flow results in the rise of shear stress (> 5 – 10 dyne/cm2) in a structurally damaged endocardium and metabolically accelerates the probability of thrombus formation. Thrombus appears as a soft mass on the valve similar to myocardium.

3.2. Pannus Formation

The inflammatory reaction to a foreign body is the main cause of pannus formation. Pannus has a lot of histological features (9). Differentiating pannus from thrombus is vital when you suspect an obstructed prosthetic valve because in case of pannus, surgery is the only option. Duration of symptoms, anticoagulation records, and mass quantification by echocardiography are the few absolute discriminators between pannus and thrombus. Besides, extension of the mass into the left atrium is the typical marker of thrombus (10). On the other hand, pannus has soft echo density.

3.3. Clinical Presentation

The typical clinical finding in PVO is muffled prosthetic valve clicks. Patients with valve obstruction usually present with symptoms of heart failure and thromboembolism. Patients may also present with dyspnea and pulmonary edema with or without hypotension.

Among the prosthetic valves, mitral valves get affected more often due to poor anticoagulation treatment following valve replacement.

3.4. Diagnosis

Transthoracic Echocardiographic Assessment (TTE) is the basic and vital diagnostic tool for examining a patient suspected of valve obstruction (11, 12). For assessing the transvalvular gradient, Doppler is the ideal method. Many conditions, such as anemia, pressure recovery phenomenon, severe prosthetic regurgitation, and patient prosthesis mismatch, can mimic PVO. Transoesophageal Echocardiography (TEE) can help decide about the type of treatment and also gives a clear idea about the location, severity, and etiology of obstruction (11). TEE also provides excellent images of the prosthetic valve in the mitral position due to its anatomical position in relation to the transducer. In addition, TEE has high sensitivity and specificity for identifying PVO at mitral position. Thrombolysis can be initiated in left-sided obstructive PVT if thrombus area is less than 0.85 cm^2 (13). Real-time three-dimensional TEE is an excellent modality for accurate diagnosis of both thrombus and pannus (14). Moreover, cinefluoroscopy gives better images compared to TEE for visualization of leaflet motion of the mitral prostheses. With limited radiation exposure, it allows correct evaluation of the opening and closing angles and motion of the base of the prosthetic valve ring (11). Furthermore, Multidetector Cardiac Computed Tomography (MDCT) provides exact function of the leaflets and is more

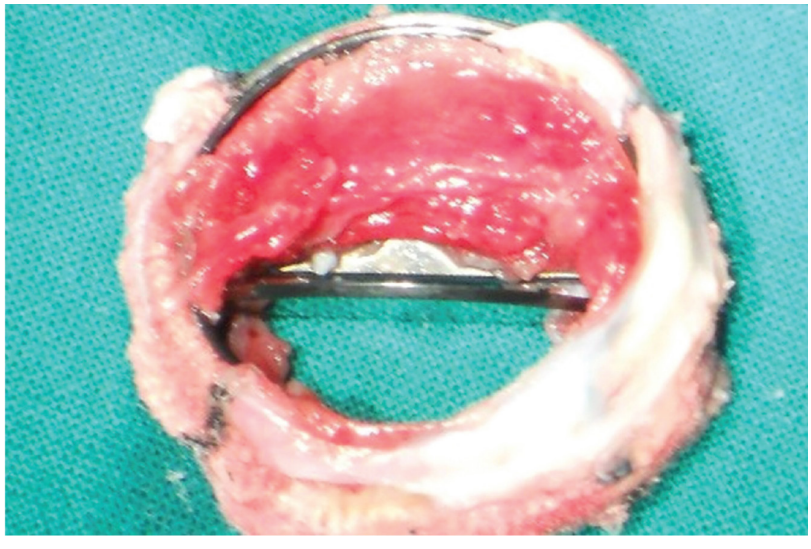

Figure 3. ATS Bileaflet Valve Was Covered with Pannus and Red Organized Thrombus

potent in identifying both thrombus and pannus. Image wise MDCT is far superior to TTE and TEE.

3.5. Treatment Options

The 2008 American College of Cardiology/American Heart Association (ACC/AHA) guidelines and the 2007 European Society of Cardiology (ESC) surgery is the treatments of choice for left-sided PVO (7). Surgery is associated with an operative mortality rate of 5 - 18% (4, 15). Bileaflet valves are usually more prone to thrombosis than to pannus, and obstruction is often confined to one leaflet (16). In comparison of the effectiveness of surgery and thrombolysis, thrombolytic therapy gains favor (15). Yet, thrombolysis is accompanied by the risk of embolism and always stays as an incomplete mode of treatment in case of pannus formation and vegetation (17). Surgery is the only option in case of pannus growth and large thrombus.

3.6. Conclusion

It is really difficult to identify the mechanism contributing to PVO for a given patient and to make a right decision regarding the treatment options. Early diagnosis and prompt initiation of adequate therapeutic procedures are required for the successful management. Inadequate anticoagulation, not preserving the subvalvular tissue during MVR, pannus-induced obstruction, and mitral chordal remnants that can interfere with proper disc or leaflet motion are the main culprits behind obstruction. If sutures are not cut short enough or become unraveled, they can be caught in the valve housing and cause sticking. LV outflow tract obstruction can occur with retention of the anterior mitral leaflet during mitral valve repair. The typical clinical finding in PVO is muffled prosthetic valve clicks. PVO may be suspected if the Doppler-derived gradients are twice as high as empirically found in 'normal' prostheses. If going for thrombolysis, patients should also be well informed about the involved risks, especially embolism. Real Time 3DTEE can be a useful technique for anatomic evaluation of PVO as a result of pannus overgrowth. Yet, management depends on thrombus burden and location, NYHA functional class, presence of embolism, availability of surgery, possible contraindications of each therapeutic option, and clinician's experience. Lack of proper roadmap in management of patients with PVO underlines the need for large clinical trials based on etiology-specified prevention and effective treatment.

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Authors' Contribution

Rajesh Rajan participated in data acquisition, data analysis, and preparation and drafting of the manuscript. VV Krishna participated in data analysis and manuscript preparation. Balachandran and Bahuleyan participated in data acquisition, data analysis, and manuscript preparation. All the authors had access to data and took responsibility for the integrity of data and the accuracy of data analysis. All the authors have read and approved the manuscript.

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