

Prosthetic valve thrombosis in a patient with mitral valve replacementMuhammad Bilal,¹ Muhammad Shahzeb Khan,² Abdul Haseeb,³ Abdul Bari Khan⁴**Abstract**

Prosthetic valve thrombosis within one year after mitral valve replacement is rarely seen in patients on warfarin therapy and without any risk factor. Here, we describe a case of a 39-year-old female, who presented with dyspnoea and shortness of breath 11 months after mitral valve replacement. The echocardiogram revealed severe valvular stenosis due to presence of clots on the mitral valve and restricted motion of the mitral leaflets. As a result of deterioration of general condition and haemodynamic un-stability, plan was made to re-operate for her valve replacement surgery. This case report highlights the diagnosis, prevention and management of patients with prosthetic valve thrombosis following mitral valve replacement.

Keywords: Prosthetic valve thrombosis; Mitral valve replacement.

Introduction

Prosthetic valve thrombosis (PVT), a rare complication of valvular replacement, is associated with significant morbidity and mortality. The exact incidence of valvular thrombosis in patients with prosthetic heart valves is unknown. However, the incidence of PVT on routine echocardiograph surveillance is reported to be as high as six percent.¹ The condition usually arises in patients who discontinue their oral anti-coagulants.² Prompt diagnosis of this lethal condition is important as it can rapidly progress to pulmonary hypertension and left heart failure (LHF).

Here, we present the case of a woman diagnosed with clots in the mitral valve one year after its replacement. The purpose of there port is to emphasise the need for prevention and successful management of this fatal condition.

Case Report

A 39-year-old female presented to Civil Hospital Karachi in August, 2014, with shortness of breath, low blood pressure (BP) and sign of low cardiac output. According to the patient, there was progressive exacerbation of symptoms

over the preceding one week. Her medical history for diabetes mellitus (DM) and hypertension (HT) was unremarkable. Past surgical history included mitral valve replacement (MVR) (Medtronic valve 29mm) 11 months earlier due to severe mitral stenosis and regurgitation as a result of rheumatic heart disease (RHD). She was prescribed anti-coagulants (warfarin) to be taken regularly with the therapeutic international normalised ratio (INR) set at 2.5.

Initial vital included BP 90/50mmHg, a regular pulse of 130 beats/min and a respiratory rate of 28 breaths/min. Electrocardiogram (ECG) revealed normal sinus rhythm with ST elevation in leads V1 to V4. Auscultation of the chest revealed fine crepitation on both sides of the middle and lower zones on the back of chest. Transthoracic echocardiogram (TTE) revealed ejection fraction (EF) of 65%, severe mitral valve stenosis and mitral regurgitation, with a mean gradient of 8.5 mmHg across the mitral valve, restricted motion of mitral leaflets and thrombus across mitral valve. Blood investigations revealed haemoglobin (Hb) of 8.4 mg/dl, platelet count (PLT) 430×10^9 /L and total leukocyte count (TLC) 12.9×10^9 /L. Clotting profile showed an INR of 2.20 while serum urea concentration and serum creatinine concentration were 16 and 0.7 mg/dl respectively. Due to the deterioration of general condition and haemodynamic un-stability, plan was made to re-operate her for valve replacement surgery.

The surgical procedure was carried out by redoing the midline sternotomy. No native valve- or chordal-sparing procedures were performed during the replacement. Immediately after adhesionolysis and aortic and bicaval cannulation, the patient was put on cardiopulmonary bypass. Moreover, the aorta was clamped and blood cardioplegia was applied. Left atrium was opened and clots were found on the prosthetic mitral valve. The thrombus was removed and cavity was washed with sterile saline solution. In order to quantify the volume of thrombus, it was put in galli pot of 60ml which was half full (30ml). After de-aeration and clamping off, the patient was taken off the cardiopulmonary bypass. With the application of protamine, cannulation was removed and a drain was placed which led to secured haemostasis. Finally, the sternum was closed with steel wire and wounds were also closed in layers. Aseptic

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dressings were applied and the patient was shifted to the intensive care unit (ICU). The patient then followed adequate anti-coagulation treatment with warfarin and the INR was maintained above 2.5. No episode of atrial fibrillation and other risk factors for thrombus formation were identified.

The post-operative course was unremarkable and TTE showed proper functioning of the prosthesis. Post-operative echo revealed normal function of mitral valve with a mean gradient of 2mmHg, pulmonary arterial systemic pressure reduced to 65mmHg from 90mmHg after surgery, no left atrial/left ventricular (LA/LV) clot or vegetation and normal LV function. Moreover, the patient had improved exercise tolerance, and was discharged on indefinite warfarin therapy after achieving an INR of 2.5. Follow-up every 6 weeks was recommended.

Discussion

The risk factors that promote formation of bio-prosthetic valve thrombosis include large left atrium size, atrial fibrillation leading to decreased transvalvular flow, decreased LVEF, prior history of thromboembolic events and a hypercoagulable state.³ In the current case, two risk factors were present, including enlarged LA measuring 4.80cm and a hypercoagulable state. The clotting profile of the patient revealed INR of 2.2. However, the patient was on continuous warfarin therapy after MVR, which had occurred 11 months earlier, but she still had sub-therapeutic INR which became the main reason for the development of thrombus in the replaced mitral valve. These outcomes illustrate that patients on prolonged anti-coagulation therapy can also develop significant thrombi and valve dysfunction prior to becoming symptomatic.

Moreover, according to the 2007 European Society of Cardiology guidelines,⁴ once valve thrombosis reaches clinically significant obstruction, management between anti-coagulation and surgery rests on the level of critical illness. Surgery is performed in critically ill patients otherwise fibrinolysis is performed. The anti-coagulation treatment is carried out by heparin and aspirin, and the clot size is reassessed by echocardiography. However, in critically ill patients surgery is performed immediately if thrombus size is >10 mm, otherwise it is delayed. The clot size in our patient was 12mm and the patient presented with severe exaggeration of LHF symptoms, including shortness of breath and fatigue. Therefore a repeat sternotomy was performed. Upon removal of clots, the leaflets were found to be mobile and, therefore, the valve of the patient was not replaced.

Prompt diagnosis of valvular embolism is essential as thrombosis of mitral valve leads to occlusion of left main artery. An acute occlusion of the left main artery can lead to cardiac death and myocardial infarction (MI) accompanied by cardiogenic shock.⁵ Since the advent of prosthetic valvular surgery, a common source of coronary emboli is the fragments of the prosthetic material in mitral valve. The first case of coronary embolism arising from mitral prosthesis was reported in 1964.⁶ The consequences of occlusion depend upon the size of the clot and the lumen of the artery it occupies. Therefore a rescue surgery was performed in our case and clots were removed from the stuck mitral valve to prevent further complications. For prevention, cessation of smoking must be advised as tobacco increases fibrin, leading to a clot.⁷ Moreover, excess alcohol should be avoided as alcohol leads to formation of fatty plaques. The rupture of these plaques causes aggregation of platelets which leads to formation of thrombus.⁸ Additionally, high glycaemic food should be restricted as elevated blood sugar can cause severe endothelial damage. It is important to note that our patient despite having no risk factors still developed PVT.

Conclusion

Thrombus in mitral valve secondary to MVR is an uncommon finding in a patient with continuous warfarin therapy. Therefore, post-operatively patients must undergo TTE on a regular basis to detect subclinical PVT in asymptomatic condition.

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