Successful thrombectomy of a stuck mechanical prosthetic mitral valve
guided by perioperative transesophageal echocardiography and cinefluoroscopy

Trombectomia com sucesso de obstrução de prótese mecânica mitral, guiada por
ecocardiografia transesofágica e cinefluoroscopia perioperatória

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ABSTRACT
We describe the case of a 53-year-old man with past history of rheumatic valvular disease who developed acute decompensated heart failure due to thrombosis of his mechanical mitral valve prosthesis. The diagnosis was established after a combined and complementary approach of echocardiography and cinefluoroscopy. Because of the severe heart failure at presentation, the patient was taken to surgery. The intraoperative transesophageal echocardiography was critical to guide a successful thrombectomy procedure. Postoperative pathological findings revealed the presence of thrombus and fibrotic tissue (pannus) in the surgical specimens removed from the valve. The success of this case and the treatment choice are supported by the most recent literature data on prosthetic valve thrombosis. We highlight the use of three diagnostic approaches in our patient: echocardiography, cinefluoroscopy and pathology.

Keywords: Heart valve prosthesis; Thrombosis; Fluoroscopy; Echocardiography, transesophageal; Thrombectomy

INTRODUCTION
Prosthetic valve thrombosis (PVT) is a lifelong risk for recipients of mechanical prosthetic heart valves. Acutely it is a rare, but life-threatening condition⁵. The incidence of PVT ranges from 0.03 to 5.7% patient-years, depending on the generation and biocompatibility of the prosthesis used, the location of the valve, and inadequate or discontinued anticoagulation therapy. Treatment is controversial and includes several options such as conservative approach with anticoagulation only, thrombolysis or surgery⁶. We report a case of a prosthetic mitral valve thrombosis that underwent a detailed diagnostic process, including echocardiography, cinefluoroscopy and pathological analysis, resulting in a successful surgical thrombectomy.

Study carried out at Veterans Affairs Medical Center (VAMC) of University of Tennessee Health Sciences Center – Memphis, Tennessee, USA.
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CASE REPORT
A 53-year-old male with previous history of rheumatic fever and a prior mechanical mitral valve replacement (St. Jude type) was admitted to hospital due to acute shortness of breath over the past six hours. Patient reported that he was not under appropriate anticoagulation during three weeks prior to admission.

At presentation, patient was in severe respiratory distress. His blood pressure was 130/78 mmHg, heart rate was 102 bpm with regular pulse and sinus rhythm on the monitor. His oxygen saturation was 91% in room air with respiratory rate of 32 breaths per minute. Cardiac auscultation revealed regular S1, S2 but muffled heart sounds. There were no murmurs, gallops or rubs. There were crackles over two thirds of both lung fields. Peripheral pulses were normal, equal and symmetrical bilaterally. There was no peripheral edema or signs of deep venous thrombosis. Chest X-ray showed acute pulmonary edema. Electrocardiogram showed sinus tachycardia and no signs of acute myocardial ischemia.

Patient was immediately placed on non-invasive mechanical ventilation, and given intravenous loop diuretics with gradual relief of symptoms. He was admitted to cardiac intensive care unit with diagnosis of acute decompensated heart failure, acute pulmonary edema, NYHA Class III-IV symptoms. At this moment laboratory tests were ordered.

All his biochemistry panel was within normal limits, cardiac enzymes were negative, BNP level was 400 pg/ml.

Transthoracic echocardiogram was not diagnostic, but suggestive of increased transmitral pressure gradient. Cinefluoroscopy and transesophageal echocardiography (TEE) showed prosthetic dysfunction (leaflet immobility) secondary to a large thrombus (Figures 1A, 1B). Due to the severity of his heart failure (NYHA Class III), presence of a large clot burden and having a left sided prosthetic valve thrombosis, patient was taken to the operating room for surgical management. Surgical thrombectomy was performed with removal of about 2 cm of apparent thrombotic material (Figure 2). Efficacy of thrombectomy was confirmed by intraoperative TEE, which showed complete recovery of valve functioning. Eventually, pathological analysis revealed the presence of a thrombus and fibrotic tissue (pannus) in the surgical specimens (Figure 2). Postoperative cinefluoroscopy and follow-up TEE showed normal mechanical valve functioning (Figures 3A, 3B). Patient had uneventful recovery, resumed appropriate oral anticoagulation and was discharged home six days later, without any bleeding complications.

Figure 1. (A).TEE 2 chamber view shows a large thrombus over the prosthetic mitral valve. Left atrial appendage (LAA) free of abnormalities; (B) fluoroscopy shows immobility of prosthetic valve leaflet

Figure 2. Pathological analysis showing the presence of a thrombus (hematoxylin eosin) and pannus (Masson Tricromic)
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Long term follow-up visits (at 3, 6 and 12 months) revealed patient compliance with oral anticoagulation regimen and use of warfarin. His INR goal was kept between 3.0 and 3.5 with monitoring every four weeks. He had neither recurrent thrombotic events nor bleeding complications.

**DISCUSSION**

Prosthetic valve thrombosis (PVT) occurs with similar frequency in patients with bioprosthetic valves and in those with mechanical valves who are treated with anticoagulants. The reported annual incidence of PVT ranges from 0.03 to 5.7%, with some reports showing higher rates in patients with mitral prostheses and in those with subtherapeutic anticoagulation(1).

Left-sided PVT has a variable profile of clinical presentation and may also be presented as cerebral vascular accident, transient ischemic attack, or peripheral embolism. However, the most common presentation is hemodynamic compromise and/or occurrence of decompensated heart failure(1) due to valve obstruction or insufficiency, as illustrated in our patient.

The gold standard for the diagnosis of PVT is transthoracic (for assessment of hemodynamics) and transesophageal echocardiography (TEE) and/or cinefluoroscopy to assess both valve motion and clot burden(2).

Traditionally, TEE has been chosen as the first diagnosis approach due to its diagnostic ability and possibility of intraoperative application(3). Cinefluoroscopy had been underused in the past, but recent studies have shown its ability to allow rapid and easy evaluation of mechanical prosthetic valve function, and, in most cases, allowing a distinction to be made between normal and dysfunctional prostheses. Some authors report cinefluoroscopy as superior to echocardiography in identifying disc motion, and capable of rapidly providing valuable information which is complementary to that obtained by echocardiography(4).

There are three options for the treatment of PVT: systemic anticoagulation only, surgery and fibrinolytic therapy. Fibrinolytic therapy for a left-sided prosthetic valve obstructed by thrombus is associated with significant risks (cerebral emboli in 12 to 15% of cases) and is often ineffective. This therapy is reserved for those patients in whom surgical intervention carries a high risk and those with contraindications to surgery(2).

Controlled data for the management of PVT are not available and both surgery (perioperative mortality) and fibrinolytic therapy (bleeding and systemic embolization) have important limitations(5).

The 2006 ACC/AHA (American College of Cardiology/American Heart Association) guidelines(2) on the management of valvular heart disease included recommendations for the treatment of PVT. The approach is based on a variety of factors such as the site of valve thrombosis, symptom status as determined by NYHA functional class, the size of the clot burden, the patient’s risk and availability of surgery.

These guidelines state as Class IIa recommendation that emergency surgery is indicated for thrombosis in a left sided prosthetic heart valve in patients with NYHA class III-IV symptoms and in the presence of a large clot burden.
Such recommendations were accurately followed in our patient. Intraoperative transesophageal echocardiography was critical for the surgical decision in preservation of prosthesis with performance of thrombectomy rather than a complete valve replacement procedure.

Finally, pannus formation due to fibrous tissue ingrowth is a less common cause of valve obstruction than thrombosis. Its presence prevents efficacy of fibrinolytic therapy; therefore, distinction between these two causes is important and may be achieved by echocardiography\(^2,6\) or only with the analysis of surgical specimens, as shown in our case.

Our patient resumed oral anticoagulation and did not have any hemorrhagic postoperative complication.

In conclusion, we described a successful thrombectomy of a left-sided (mitral) PVT guided by a combined and complementary diagnostic approach by transesophageal echocardiography, cinefluoroscopy and pathology. PVT is not a frequent emergency (incidence less than 5% per year), but remains as a true medical challenge. Decision-making process is difficult and should be based on clinical presentation and on the findings of diagnostic imaging approaches (echocardiography and/or cinefluoroscopy).

REFERENCES


