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## Obstructive Thrombosis of a Mitral Valve Prosthesis and Live Three-Dimensional Transesophageal Echocardiography

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**CASE REPORT**

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**KEY WORDS:** *statins, percutaneous coronary intervention, acute coronary syndromes*

A 67-year-old woman with a bileaflet mitral valve prosthesis since 2007 due to mitral stenosis and chronic atrial fibrillation was admitted because of severe shortness of breath and diarrhea. She had been discharged from the hospital one week earlier on clarithromycin due to lower respiratory tract infection. She gave a history of increasing shortness of breath on exertion ever since, with the recent addition of diarrhea over the last 24 hours. Her past medical history includes previous ischemic stroke in 2004.

On clinical examination the prosthetic first heart sound was reduced in intensity and on auscultation of the lungs she had coarse crepitations bilaterally and bronchial breathing over the left lower lung field. Performed chest X-ray revealed consolidation of the left lower lobe and bilateral pleural effusion. Her ECG demonstrated atrial fibrillation with a rate of 90 bpm and the arterial blood gas indicated mild metabolic acidosis (PH= 7.33, HCO<sub>3</sub>= 19 mEq/Lt, pO<sub>2</sub>= 85 mmHg, and pCO<sub>2</sub>= 40 mmHg). White cell count and C-reactive protein were increased as expected and INR was 1.5. Patient was immediately started on unfractionated heparin. Bedside transthoracic echocardiogram revealed increased velocities across the prosthetic mitral valve (MV peak vel= 2.8 m/s, peak gradient= 35 mmHg, mean gradient= 25 mmHg) (Figure 1). Patient deteriorated significantly despite an adequate anticoagulation achieved within 6 hours. Transesophageal echocardiography with live 3D was performed which confirmed the presence of a thrombus on the atrial side of the prosthetic valve resulting in an immobile lateral leaflet and a severely restricted anterior leaflet (Figures 2 & 3). Furthermore there was a large thrombus in the left atrial appendage extending into the left atrium.

Patient underwent urgent redo mitral valve replacement. The mitral valve prosthesis was severely covered with thrombus and the cusps were fixed. A bioprosthetic St Jude medical mitral valve was implanted with good results.

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**DISCUSSION**

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Prosthetic heart valve thrombosis with obstruction remains a significant problem causing increased morbidity and mortality. It occurs in 1% to 3% of cases annually which is somewhat lower than the annual risk of bleeding due to anticoagulation therapy (2.7%)<sup>1</sup>. According to the recent guidelines<sup>2</sup>, in hemodynamically unstable patients immediate action is warranted and surgical treatment is preferred over thrombolysis

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OBSTRUCTIVE THROMBOSIS OF A MITRAL VALVE PROSTHESIS AND LIVE THREE-DIMENSIONAL TEE

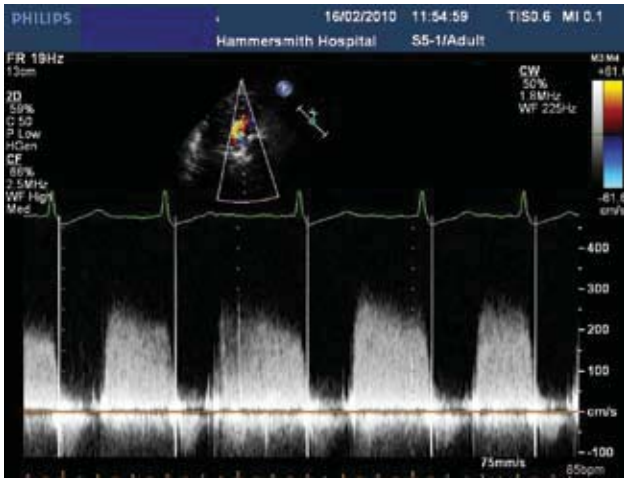


FIGURE 1. Transthoracic study, continuous wave LV inflow. Peak transmitral velocity is 2.8 m/s, peak gradient is 36 mmHg and mean gradient 26 mmHg, indicating severe stenosis of the prosthetic valve.

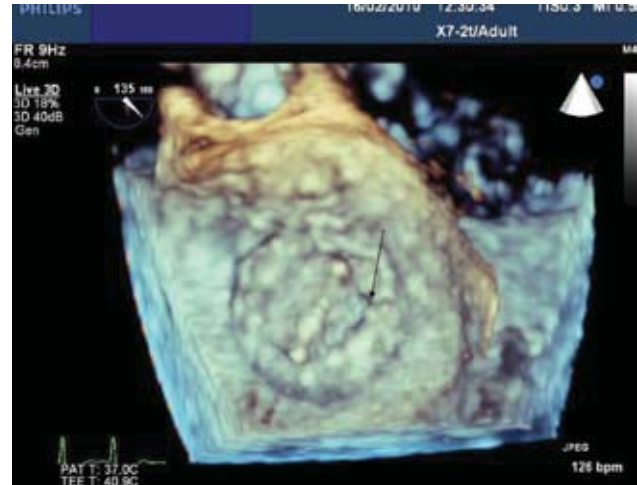


FIGURE 3. 3D transesophageal echo (TEE) study picture of the atrial side of the mitral prosthesis in diastole. The valve barely opens (see arrow).

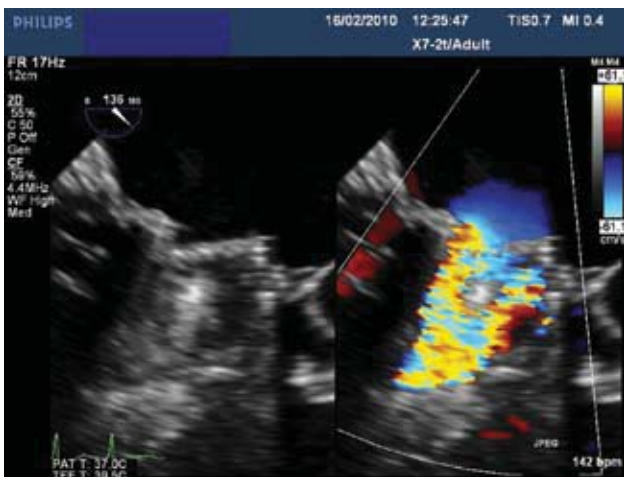


FIGURE 2. Transoesophageal study. Immobile lateral disk and restricted opening of the anterior disk resulting in highly turbulent flow across the valve during colour flow mapping.

when readily available or there are no serious contraindications or comorbidities precluding its use. Moreover, in the presence of thrombus in the left atrial appendage, as in this case, thrombolysis adds to the risk of systematic embolization<sup>3</sup>. A repeat operation carries a significant risk, with mortality rate ranging between 10-15%, which may be two- or three- fold higher in critically ill patients<sup>4</sup>. The single most important risk factor that contributes to prosthetic valve thrombosis is inadequate anticoagulation, followed by the position of the prosthesis, mitral being more thrombogenic than aortic, the type of the prosthetic valve, presence of atrial fibrillation, low

cardiac output and hypercoagulable states such as pregnancy, dehydration or inflammation.

Recent history of increasing shortness of breath or an embolic event along with findings on clinical examination such as reduced intensity of first prosthetic heart sound or its absence should alert the clinician towards the possibility of prosthetic mitral valve thrombosis. Suboptimal INR and transthoracic or transesophageal echocardiograms confirm the diagnosis.

It may be possible to demonstrate the type of mitral prosthesis as well as the restricted motion of disks or occluder from a transthoracic approach but Doppler echocardiography is more important since it can define the increased transmitral peak and mean gradient as well as the pressure half time (PHT) and mitral valve area with PHT. Prosthetic obstruction considerably prolongs the PHT, and this may be a very useful sign especially if the image quality is not good. A PHT longer than 200 ms is unusual in a normal functioning prosthetic valve especially when peak velocity is above 2.5 m/sec.<sup>5</sup> A transesophageal echo (TEE) study allows better visualization of the prosthetic valve and its motion throughout the cardiac cycle and defines whether it is restricted or not. Masses attached to the valve or the ring are relatively easily defined as well as their size and extent in the left atrium. TEE also allows the differential diagnosis between thrombus and pannus formation based on the size and the echogenicity, thrombi are usually larger, extend into the left atrium and have a soft echodensity similar to that of the myocardium<sup>6</sup>. Furthermore, live 3-D TEE offers the opportunity to inspect the atrial size of the prosthesis allowing a comprehensive assessment of the size and nature of attached masses and a direct high quality 'surgical' view of the prosthesis.

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