CASE REPORT

Acute obstruction of a mechanical aortic valve in a young woman: case report and review of the literature

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Summary

Acute aortic valve obstruction is a medical and surgical emergency necessitating intensive care unit admission. The differential diagnosis includes thrombosis, pannus formation or vegetations. The diagnosis should be obtained as soon as possible, with possible orientation towards the cause. Different diagnostic modalities exist nowadays. Notably, the transesophageal echocardiography (TEE) offers a diagnosis and a guide for management. Surgical treatment remains of choice despite growing evidence about a benefit of combined thrombolytic and anticoagulation line of management.

Learning points:
- Acute management and resuscitation of acute valvular obstruction.
- Differential diagnosis and role of echo in the diagnosis.
- Management options.

Background

Acute prosthetic valve obstruction represents a challenging medical emergency. The difficulties are in both diagnosis and treatment. We present a case of acute prosthetic aortic valve obstruction which had been managed successfully.

Case presentation

A 27-year-old woman with Marfan’s syndrome, who had a replacement of her ascending aorta and insertion of a mechanical aortic valve in 2006, presented to a district hospital with hypoxia and acute cardiogenic pulmonary oedema. She had a history of a small stroke, confirmed by CT scan, in 2009 from which she had fully recovered. There was a 2-week history of rhinorrhea, increasing dyspnoea and central chest pain referred to the back. Over the last two nights, she started to have night sweats but neither fever nor rigors. Her only drug therapy was warfarin and according to the patient’s clinic records, the international normalised ratios (INRs) had been within the recommended therapeutic range. Transthoracic echocardiography (TTE) demonstrated a mean transvalvar gradient of 88 mmHg. The patient had been diagnosed as with cardiogenic pulmonary oedema and Furosemide 40 mg was administered intravenously and she was transferred immediately to a tertiary cardiac intensive care unit.

Investigation

On arrival, the patient was fully conscious but with cardiogenic pulmonary edema. Her respiratory rate was
50 cycles/min, with coarse crepitations heard over all lung zones; arterial saturation was 85–88%. A sinus tachycardia at a rate of 145 beats/min was present and the blood pressure was 109/65. Blood tests showed international normalized ratio (INR) 1.3, activated partial thromboplastin time (APTT) ratio 0.95, Troponin I was 4289 ng/l ($n<50$), creatinine 51 μmol/l, ALT 115 ($n=0–40$ U/l) and CRP 67. Full blood count showed Hb 146 g/l, WBC $28.2\times10^9$/l (neutrophils $25.4\times10^9$/l) and platelets $233\times10^9$/l.

A repeated transthoracic echocardiogram was performed with good imaging windows. There appeared to be severely limited excursion of the bileaflet discs with no obvious thrombus or vegetations. The peak gradient across the valve was 128 mmHg and the left ventricle was hyperdynamic and non-dilated but severely impaired. Arterial oxygen saturation was 96% and lactate was 1.4 mmol/l. The chest X-ray showed a picture of pulmonary oedema (Fig. 1). Owing to the degree of respiratory and haemodynamic compromise, transoesophageal echocardiography (TEE) was deferred until the patient was anesthetised with a fully prepared theatre team.

The TTE (Fig. 2A, B and C; Videos 1 and 2) demonstrates a bileaflet mechanical St Jude prosthesis with severely limited excursion of the leaflets. The differential diagnosis was thrombosis, pannus formation or least likely endocarditis. The history, in particular the acute presentation, suggested prosthetic thrombosis. Furosemide 80 mg was administered intravenously and continuous positive airway pressure was applied via face mask, producing a reduction in both dyspnoea and respiratory rate. Discussion centered around i.v. thrombolysis, but was not performed due to lack of identifiable thrombus and the history of stroke. Blood cultures were taken, i.v. antibiotics and heparin infusion were commenced and emergency surgery was scheduled as soon as a theatre and team could be assembled.

**Treatment and outcome**

The patient was anesthetised within 4 h. Haemodynamic stability was maintained at induction and a TEE probe

![Figure 1](image1.png)

**Figure 1**

AP chest X-ray of the patient on admission to the ICU, showing bilateral infiltrate due to cardiogenic pulmonary oedema.

![Figure 2](image2.png)

**Figure 2**

(A and B) TTE PLAX showing prosthetic aortic valve in systole and diastole with shadowing, note that the assessment of prosthetic aortic valve is difficult on TTE. (C) Apical four-chamber view with spectral continuous wave Doppler (CWD) across the aortic valve showing a maximum velocity across the valve of 5.4 m/sec and a peak gradient of 119 mmHg.
was inserted. The images thus obtained did not show thrombus or vegetations, but confirmed severe restriction of the leaflets (Fig. 3 A, B and C; Videos 3, 4, 5 and 6). The TEE confirmed the high transvalvular gradient obtained by TTE. Following this confirmation of the TTE findings, a secondary median sternotomy was performed and division of adhesions, cardiopulmonary bypass was established uneventfully. The St Jude valve was exposed and extensive pannus and granulation tissue were revealed overlying the valve and interfering with the movement of the leaflets. There was no evidence of infection, but a tissue sample was sent for a gram stain. All granulation tissue was removed along with the valve. A 25 mm ATS bileaflet mechanical valve was then inserted and the aorta was closed. The patient was then taken off bypass with a minimal inotropic support and transferred to the intensive care unit (ICU). There were no complications and the patient was discharged home on day 10. None of the samples sent for culture returned any positive microbiological growth.

**Discussion**

The diagnosis was acute aortic prosthetic valve obstruction by pannus. In this case, the presentation initially resembled an acute thrombotic occlusion of the prosthesis prosthetic valve obstruction (PVO) is an infrequent but serious complication and is associated with significant morbidity and mortality (1). Historic studies from 1980s and 1990s showed an incidence rate ranging from <0.5 to 4.5% per
patient year (2). The possible causes include thrombus formation, pannus formation and, rarely, vegetations. Pannus and thrombosis may be present alone or in combination causing acute or subacute valve obstruction (3). Pannus is a fibro-connective tissue ingrowth from the sewing ring that occurs many years after valve implantation. Moreover, routine anticoagulation does not prevent its formation (4). In a study included 112 obstructed mechanical valves, thrombus alone or with a little pannus formation was the most common cause and found in 77.7%, while pannus formation was the underlying cause in 10.7% of valves. Pannus in combination with thrombus formation was the most common cause and found in 77.7%, while pannus formation was the underlying cause in 10.7% of valves. Pannus in combination with thrombus was present in 11.6% (2). Nevertheless, pannus formation is more common in the aortic position (70% vs 21%) (4). More specifically, failure of St Jude Medical mechanical aortic prosthetic valves due to pannus formation is rare, in one study estimated to be in 0.73% of patients (5). The time window from valve implantation to pannus formation varies and has been reported to occur between 3 months and 23 years after implantation (6, 7).

In our patient, the presenting symptoms and signs were confusing. The preceding symptoms of viral-like infection followed by night sweats but without fever raised a suspicion of endocarditis-associated prosthetic valve dysfunction. The differential diagnosis also included the possibility of thrombus formation, as the initial INR measured in our hospital was less than the target INR for this type of valves. TTE done in the referring hospital confirmed a valve dysfunction with high mean gradient; a finding which had been re-confirmed by repeating the TTE on admission to our cardiothoracic ICU. Differentiation of pannus from thrombosis by TTE is challenging. Data from previous prospective studies and case reports suggested using a combination of duration of symptoms, anticoagulation status, and lastly qualitative and quantitative ultrasound intensity of the obstructing mass to differentiate pannus from thrombus formation (4). A video-intensity ratio of <0.70 had a positive predictive value of 87% and a negative predictive value of 89% for thrombus in one study, but this parameter is not in current use in a standard echocardiographic studies (4).

Other possible diagnostic modalities include a real-time 3D TEE (8). According to the AHA/ACC and ESC guidelines, if no thrombus is clearly identified and the patient had NYHA grade i.v. symptoms, then a TEE should be performed and emergency surgery scheduled (9, 10). The guidelines also recommend fluoroscopy to identify the mechanical leaflet movement. As we were able to identify the limited leaflet excursion confidently on TTE and we did not wish to place the patient in supine in view of the dyspnea. Fluoroscopy was not performed in this instance. In general, TEE is less useful to assess mechanical aortic valves (due to reverberation artifact) than mitral or tricuspid mechanical valves, whilst TEE of bioprostheses or homografts provides better image quality. According to the recommendations of the American Society of Echocardiography (ASE), thrombus is associated with a short duration of symptoms and with a history of inadequate anticoagulation (INR <2) in comparison to pannus formation (11). A combination of a soft density on the prosthesis and an inadequate INR may have a positive and negative predictive values of 87 and 89%, respectively, for thrombus formation (4). According to the 2009 ASE guidelines, the distinction between thrombus and pannus is essential if thrombolytic therapy is to be considered and a combination of TEE along with clinical parameters should be assessed (11). Thrombi are in general larger with soft ultrasound density, similar to that of the myocardium, and the detection of abnormal prosthetic valve motion by TEE is more commonly seen in thrombosed valves. In contrast, pannus formation is visualised as a small dense mass that in 30% of cases may not be distinctly seen. Again, pannus formation is more common in the aortic position (4, 11).

A retrospective single-centre study comparing 263 cases of surgery (136 cases) vs thrombolysis (127 cases) showed no significant mortality difference but more complication in the thrombolysis group (haemodynamic stability and embolic manifestations). The long-term mortality, complication and recurrence rates were higher in the thrombolysis group. Interestingly, the study showed an increased trend towards surgical management in the time period after the introduction of TEE to guide patients’ management (12). Nevertheless, the author’s recommendations based on reported higher mortality with patients presenting with NYHA class IV (overall perioperative mortality of 12.3% in comparison with a mortality rate of 17.5% in patients with functional class IV and 4.7% for functional classes I–III) (2) proposed a rescue fibrinolysis in case if the operation cannot be performed urgently (critically ill patients with low cardiac output) or if contra-indicated (12).

Declaration of interest
The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of this case report.

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Author contribution statement
Dr A Roshdy as intensive care doctor admitted the patient, and wrote the case report and the discussion. Dr M A I Sarsam and Dr G T Karapanagiotidis are surgeons, wrote the surgical intervention and revised the article. Dr G T Karapanagiotidis had taken the patient’s consent. Dr S N Fletcher was the ICU consultant, admitted the patient, reviewed the TTE and TEE images, and reviewed and modified the article.

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