Tricuspid valve arrest

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DESCRIPTION

A young woman with a medical history of endocarditis status postporcine tricuspid valve replacement, intravenous drug abuse and seizures presented to the hospital with shortness of breath. The patient reported progressively worsening dyspnoea over 2 days with bilateral lower extremity swelling. On presentation, the patient was in acute distress, hypoxic and hypotensive. Minutes following her presentation she had a seizure which progressed to status epilepticus. The patient was intubated and started on norepinephrine for shock. Chest X-ray and CT of the chest revealed multiple diffuse opacities in the bilateral lung fields which were concerning for multifocal pneumonia versus septic embolisation. Transthoracic echocardiogram revealed an acute occlusion of the tricuspid valve from a heavy burden of thrombotic versus infective material, which engulfed the prosthesis and extended into the subvalvular space and right ventricle. Doppler interrogation noted only faint flow through the valve with a mean gradient of 41 mm Hg noted (video 1). The right atrium was severely dilated indicating an acute on the chronic process, with mass effect on the left atrium indicating significant pressure overload of the right heart. The right ventricle, right ventricular outflow tract and the left ventricle (LV) were severely under-filled. LV function was noted to be hyperdynamic (figure 1, video 2). The patient was emergently transferred to the ICU, norepinephrine was initiated for haemodynamic support and cardiothoracic surgery was contacted. The patient was evaluated for possible surgical intervention versus thrombolytic therapy given the patient's critical condition. However, before any intervention could take place

Figure 1 (A) Transthoracic echocardiogram, apical four chamber view showing a completely occluded prosthetic tricuspid valve with overwhelming thrombotic burden (red arrow), in addition to severe right atrial enlargement causing mass effect into the left atrium (star). (B) Transthoracic echocardiogram, short-axis view at the level of the aortic valve revealing heavy thrombotic burden and with total occlusion of tricuspid valve (arrow) with massive right atrial enlargement (star). (C) Transthoracic echocardiogram, right ventricular inflow tract showing heavy thrombotic burden with total occlusion of tricuspid valve (arrow) with massive right atrial enlargement (star).

the patient went into cardiac arrest. The patient was noted to be in ventricular fibrillation so advanced cardiac life support was initiated. Unfortunately, the patient was unable to be resuscitated and died in the medical intensive care unit.

Complete prosthetic heart valve obstruction (PHVO) is exceedingly rare, however can quickly lead to refractory obstructive/cardiogenic shock if not promptly acted on.¹ It is imperative to have this on your differential in patients with refractory shock, especially those with a history of mechanical

Video 1 Transthoracic echocardiogram, short axis view at the level of the aortic valve with colour Doppler, revealing severely decreased flow through the tricuspid valve due to nearly complete occlusion due to large clot burden.

Video 2 Transthoracic echocardiogram, apical four chamber view showing a completely occluded prosthetic tricuspid valve with overwhelming clot burden, in addition to severe right atrial enlargement causing mass effect into the left atrium. Hyperdynamic left ventricle appears severely underfilled.

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valve replacement. This phenomenon has reported incidence of 0.1%–5.7% per patient-year. It can involve any mechanical valve (aortic, mitral, tricuspid and pulmonic) although most studies/observations include aortic or mitral, as they are far more commonly replaced.¹ Diagnosis is primarily made with echocardiogram (transthoracic echocardiogram, transoesophageal echocardiogram), usually at the bedside as these patients will be critically ill. Optimal management of PHVO remains controversial.¹ anticoagulation (AC) is often used for smaller thrombi (<5 mm) however for those >5 mm or causing complete obstruction more aggressive therapy is warranted. A second option for therapy (especially when complete obstruction is noted) is thrombolysis. Thrombolytic therapy has a success rate of 70% and a mortality rate of 9%–11%; it is more efficacious for aortic valve thrombosis than for mitral or tricuspid valve thrombosis as well as for patients with symptoms of less than 2 weeks duration. Since thrombolytic therapy carries a considerable risk of embolisation (up to 19%), it should be reserved for critically ill patients whose operative risk is high.² Even though there are no consensus guidelines, surgery is often recommended for patients

with PHVO.¹ Surgery is associated with better clinical outcomes over thrombolysis.¹ However, the mortality rate associated with surgical therapy for valve obstruction is approximately 15% but may be substantially higher for emergency operations in patients with haemodynamic instability.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

Learning points

- ▶ Complete prosthetic valve obstruction is exceedingly rare but can quickly lead to refractory shock if not acted on quickly.
- ▶ Prompt diagnosis with bedside echocardiogram can be life-saving.
- ▶ Surgery remains the gold standard in treatment, however thrombolysis is an option for critically ill patients.

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