Intermittent cardiogenic shock in a man with mechanical prosthesis of the aortic valve

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Intermittent dysfunction of prosthetic aortic valve is a rare but life-threatening condition that may be difficult to recognize. Here, we present a case of 64-year-old man with a history of bicuspid aortic valve that had been replaced with a Medtronic-Hall prosthesis 15 years earlier. Until his current illness, he was fit, with good function of the valve and well maintained anticoagulation. After several days of progressive intermittent breathlessness, he experienced severe anginal chest pain, orthopnea, and dizziness that proceeded into an electromechanical dissociation. Circulation was restored after a brief resuscitation, intubation, and the administration of vasopressors by emergency medical services personnel. On admission to the intensive care unit, he had sinus rhythm with intraventricular conduction defect (Figure 1), stable blood pressure on vasopressors, and was ventilated for pulmonary edema (Figure 2). Nongated chest computed tomography scan ruled out an aortic dissection, and no particular abnormality was observed in the area of the aortic valve, although the resolution in the closest proximity of the prosthesis was attenuated by artifacts. Echocardiography showed normal right ventricular size/function, mild systolic left ventricular dysfunction, aortic valve prosthesis with a small systolic gradient (30/20 mm Hg) and negligible insufficiency (1/5), and no thrombus/mass within the prosthesis (Figure 3B). Troponin I was elevated (140 μg/L), but angiography (Movies I and II in the online-only Data Supplement) showed normal coronary anatomy and normal motion of the prosthetic disc. The cause of hemodynamic collapse remained unexplained. The condition of the patient improved, but the next day, after an attempt to taper norepinephrine infusion, he developed multiple episodes of sudden and profound hypotension, terminated by bolus-dosed norepinephrine (Figure 3D). During one of such episodes, we noticed an abrupt change of invasive arterial pressure waveform with diastolic flattening (Figure 3A), coinciding with a disappearance of audible click of the valve. At this time, bedside echocardiography confirmed massive aortic regurgitation from the incompletely closed aortic prosthesis (Figure 3C and 3D; Movies III and IV in the online-only Data Supplement). Transesophageal echocardiography (Movie V in the online-only Data Supplement; Figure 4) identified a nonobstructing rod-like protrusion bellow the valve plane formed by pannus (arrow, Figure 4), and small filiform thrombus, superimposed on the base of the pannus (visible in Movie V). These structures probably

Figure 1. Admission 12-lead ECG, showing sinus rhythm with intraventricular conduction defect.
Figure 2. Admission chest x-ray, showing bilateral pulmonary edema and tracheal intubation.

Figure 3. Femoral artery pressure waveform with flattened diastolic part, corresponding to episode of no audible valve click (A, left) due to transient massive aortic intraprosthesis regurgitation (C). Sudden reappearance of normal arterial pressure contour (A, right, arrows) with normal closure of the valve prosthesis (B). Eight-hour trend of invasive systemic blood pressure demonstrating 5 episodes of profound hypotension (arrows) due to valve dysfunction, terminated spontaneously or by epinephrine bolus administration (D).

Figure 4. Transesophageal echocardiography demonstrated nonobstructing rod-like pannus below the valve (arrow) and small thrombus superimposed on the base of the pannus (visible in movie V). LV indicates left ventricle; LA, left atrium; AO, aorta.
behaved as a backstop for the smaller part of the disc that tilts into the left ventricular outflow tract, trapping it in an open position. The intermittent, noncyclic character of the valve dysfunction could be explained by thrombus superposition on subvalvular pannus. The patient underwent valve replacement with pannus resection and made a full recovery with no recurrence of symptoms. The explanted valve prosthesis showed no intrinsic malfunction and the thrombus had already disappeared at the time of surgery.

In comparison with mitral position, intermittent intraprosthetic regurgitation in the aortic position is very rare. Proper valve closure is facilitated by high aortoventricular gradient, and, therefore, norepinephrine was helpful in abolishing the episodes of regurgitation. Single tilting disc prostheses are more prone to develop this condition, and its occurrence has been linked to subvalvular nonobstructing pannus that creates a niche for superposing formation of thrombus. Intermittent prosthetic aortic valve regurgitation can present as a severe acute ischemia from load/perfusion mismatch and as heart failure, so prompt recognition may be lifesaving. Given the fact that the valve may appear almost normal in between the episodes, valve malfunction may be difficult to identify. The loss of normal diastolic pressure decay in arterial pressure waveform, together with the loss audible click could indicate this problem, and it should be monitored by repeated careful echocardiographic interrogation of the valve during the episode of hemodynamic instability.

Disclosures

None.

References

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