

Ventricular tachycardia: ominous sign of devastating prosthetic aortic valve dehiscence

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Prosthetic valve endocarditis (PVE) is the most feared complication after valve implantation. It usually results in substantial morbidity and mortality in the postoperative period. An adverse effect on the annulus can cause conduction disturbances in the atrioventricular (AV) node, resulting in a high-degree AV block. This study describes a case of PVE that predominantly presented with sustained monomorphic ventricular tachycardia, which indicated a severe clinical course of PVE caused by a significant displacement of the aortic valve prosthesis. In our opinion, a very pronounced flap valve motion of the dehisced valve probably caused, in the critical moment, coronary artery blood flow limitation by means of coronary microembolization, which produced temporary ischemia and provoked sustained ventricular tachycardia. Furthermore, disturbances of rhythm such as ventricular tachycardia in the setting of endocarditis indicate a high-risk condition and should mandate fast and thorough noninvasive diagnostic procedures to obtain correct diagnosis even in the case of mild, slowly progressing disease.

In the era of the sophisticated heart valve design accompanied by a precise surgical technique that successfully encompasses even the transcatheter aortic heart valve implantation, the most severe complications of aortic valve replacement (AVR) are the ones after heart valve implantation.^{1,2} The most feared one is prosthetic valve endocarditis (PVE), which results in substantial morbidity and mortality in the postoperative period.¹ The bacteria adhere to the prosthetic surface and produce a biofilm that effectively protects them from the host defenses and antibiotics. In PVE, the infection usually affects the prosthetic suture line of the annulus.¹ The invasion of the suture line can cause weakening and necrosis, leading to the complications of abscess, fistula, or valve dehiscence.¹ The dehiscence of an aortic valve can present with a significant displacement of the prosthesis, causing major paravalvular leak at the level of the aortic valve.³ Patients with PVE and major paravalvular leak most frequently present with an acute chest pain or heart failure accompanied by fever and chills.³ An adverse effect on the annulus can cause conduction disturbances in the atrioventricular (AV) node, resulting in a high-degree AV block.^{1,4} This study

describes a unique case of PVE that predominantly presented with sustained monomorphic ventricular tachycardia, which indicated a severe clinical course of PVE caused by a significant displacement of the aortic valve prosthesis.

CASE

A 55-year-old male was referred by his family medicine physician to the emergency department (ED) because of chest pain and fever. One year earlier, the patient underwent AVR (St Jude A-29) for severe aortic stenosis. His medical history included a stroke with full recovery. The rest of his medical history was unremarkable. On physical examination, he was febrile (temperature 38.5°C) with diaphoresis, blood pressure of 110/50 mm Hg, a heart rate of 90 bpm, and normal oxygen saturation on room air. A combined systolic and diastolic heart murmur was noted. Crackles were present on the bases of the lung, but with no evident signs of severe heart failure. In addition, no typical signs of endocarditis on the skin were detected. Laboratory testing at admission indicated mildly elevated white blood cells ($13.6 \times 10^9/L$), C-reactive protein (CRP, 63.1 mg/L), and lactate dehy-

drogenase (LDH, 728) with signs indicative of chronic anemia (hemoglobin 99 g/L). Electrolytes and serum troponin were within normal laboratory ranges.

During short initial triage in the ED, the patient suddenly developed wide QRS complex tachycardia compatible with a sustained monomorphic ventricular tachycardia (Figure 1). He was successfully treated with amiodarone and synchronized electrocardioversion with 200 J. After a short period, the patient was admitted to the coronary care unit. Transthoracic echocardiography and transesophageal echocardiography were performed promptly. Principal echo findings included moderate, predominately paravalvular aortic regurgitation with left coronary Valsalva sinus aneurysm but no visible communication with the left atrium. The specific “rocking motion” of the aortic valve prosthesis partially limited the severity of aortic regurgitation. The left ventricle was spherical shaped and mildly dilated, with preserved ejection fraction. Moderate functional mitral regurgitation was found because of aortic regurgitation and increased left ventricular filling pressure. The imaging of the heart in the catheterization laboratory with fluoroscopy showed that the prosthetic aortic valve was significantly displaced with every heartbeat, causing an impressive “rocking motion” of the prosthesis (Figure 2). The patient underwent urgent operation. Intraoperative findings revealed highly advanced inflammatory process, which included locally advanced abscess of the aortic root and the sinuses of Valsalva. The aortic root and annulus were reconstructed with glutaraldehyde-fixed pericardium and root replacement after which the Bentall procedure was performed. However, severe inflammation in the tissues resulted in root and left coronary button dehiscence and massive intractable bleeding. The patient eventually died from irreversible hemorrhagic shock. Autopsy findings confirmed PVE caused by *Propionibacterium acnes* (PA). Coronary artery disease was excluded.

DISCUSSION

It is important to bear in mind that endocarditis of the prosthetic aortic valve is still rather uncommon, but it represents a very severe medical condition with a significant incidence.¹ It usually presents with a clinical picture of endocarditis with heart failure, which progresses to cardiogenic shock mainly due to severe aortic regurgitation.¹ Common rhythm disturbances primarily include AV node conduction disturbances resulting in different stages of AV block.^{1,4} Our review of published reports revealed no case of PVE that initially presented as sustained ventricular tachycardia requiring electrical cardioversion. Regarding the echocardiographic evaluation of the aortic valve regurgitation, it is important to recognize recently described “flap valve phenomenon” (FVP) in the situation of aortic valve dehiscence.^{5,6} The flap valve motion of the dehisced valve with each cardiac cycle prevented significant paravalvular regurgitation in a diastole.^{5,6} This phenomenon, which was also observed in this study, probably prevented progression to severe congestive heart failure or pulmonary edema in the patient. Furthermore, it is hypothesized that the flap valve motion was much more pronounced in the

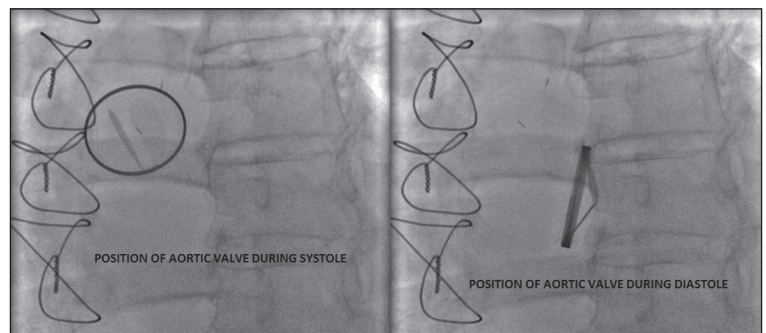


Figure 1. Presenting electrocardiogram monitor strip of the patient showing wide QRS tachycardia consistent with ventricular tachycardia.

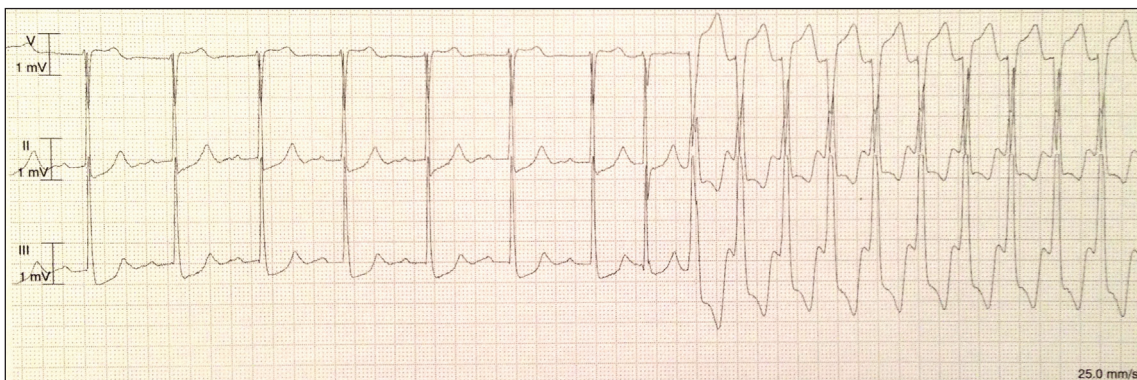


Figure 2. Dehiscence of the aortic valve showing impressive “rocking motion” of the valve during the heart cycle.

patient (Figure 2), and probably caused, in the critical moment, coronary artery blood flow limitation by means of coronary microembolization that produced temporary ischemia and provoked sustained ventricular tachycardia. A similar pathophysiological mechanism of acute total coronary obstruction resulting in myocardial infarction has been recently published.⁷ PA is an anaerobic, nonspore-forming, gram-positive bacillus, and is often part of the normal flora of human skin.⁸ However, it can rarely cause serious infections including infections of prosthetic valves, native valves, and annuloplasty rings, usually presenting as a slowly progress-

ing disease.⁸ It is unclear whether PA can cause such devastating forms of PVE, since only a few cases of PA PVE have been published and, by itself, it represents a very rare condition.⁸

In conclusion, disturbances of rhythm such as ventricular tachycardia in the setting of susceptible endocarditis indicate a high-risk condition and should mandate fast and thorough noninvasive diagnostic procedures to obtain correct diagnosis even in the case of a mild, slowly progressing disease. This is mandatory to increase the probability of surgical success and reduce high patient mortality.

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