

CLINICAL VIGNETTE

Urgent Re-operation for Prosthetic Aortic Valve Dysfunction

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Case Report

A 69-year-old woman with a history of a St. Jude's prosthetic aortic valve placement 9 years ago presented to cardiology with progressive chest - pressure and dyspnea. Her symptoms had worsened significantly over the two weeks prior to her visit. She also noted paroxysmal nocturnal dyspnea, lower extremity edema, and three-pillow orthopnea.

On physical examination, the patient was in mild respiratory distress, with mild tachypnea. Her neck veins were elevated. Cardiac auscultation revealed III/VI, crescendo-decrescendo systolic murmur with radiation to the carotids.

Valvular click was inaudible. Her lungs were clear with no dullness to percussion. She had 1+ lower extremity edema.

The patient was admitted to the hospital. Her ECG revealed sinus rhythm, poor R wave progression, ST/T wave abnormalities in the lateral leads and bi-atrial enlargement (Figure 1). Her troponin level was mildly elevated, and her INR was sub-therapeutic. She reported poor adherence with her warfarin due to intermittent epistaxis. Chest radiograph showed prominent pulmonary vasculature and mild cardiomegaly.

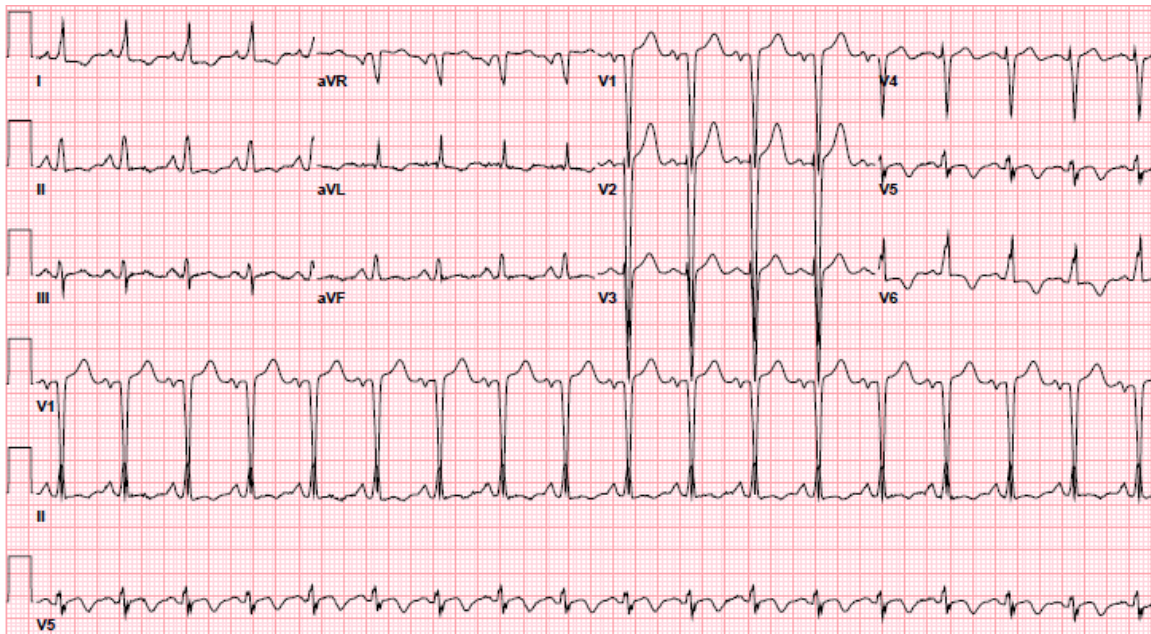


Figure 1: The initial ECG showing normal sinus rhythm, ST/T abnormalities, and bi-atrial enlargement.

Her symptoms improved with aspirin, intravenous heparin, and diuretics. Urgent echocardiogram showed dysfunction of her prosthetic aortic valve. There was an elevated transvalvular gradient with a velocity of

5.0 m/s, mean gradient of 52 mm Hg and a peak gradient of 99 mm Hg, consistent with significant stenosis. Cardiac catheterization showed mild coronary artery disease, and fluoroscopy confirmed restricted motion of the mechanical aortic valve leaflets (Figure 2).

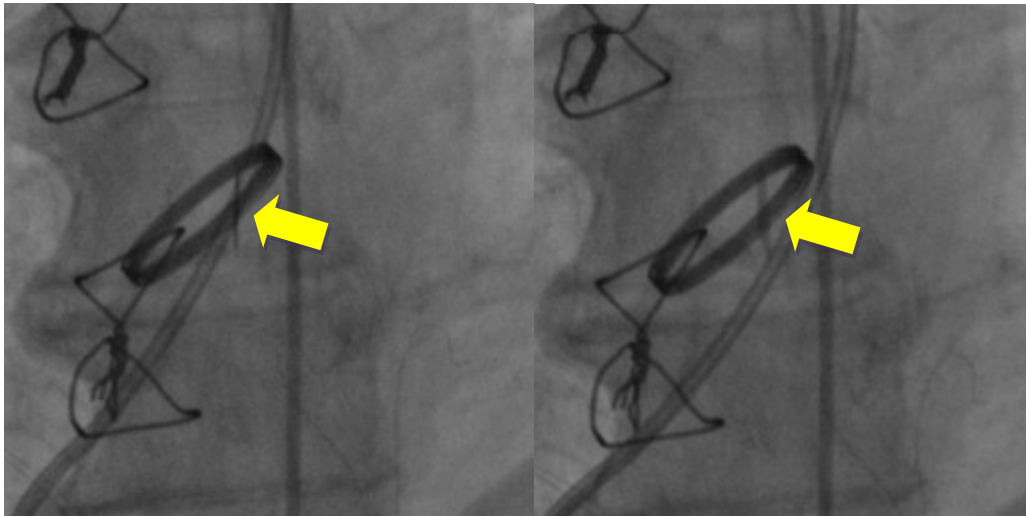


Figure 2: Fluoroscopic evaluation showing restricted leaflet motion (arrow) of the St. Jude's prosthetic valve during the cardiac cycle (left panel- systole; right panel- diastole).

She was referred for urgent surgery and underwent successful replacement of her dysfunctional prosthetic aortic valve with a bioprosthesis. The explanted prosthetic valve showed that valvular dysfunction was due to a combination of pannus and thrombus formation.

Background

Despite structural evolution in mechanical valves, prosthetic heart valves are associated with several complications. Structural deterioration, thrombus or pannus formation, endocarditis and hemolytic anemia occur to varying degrees based on the type of implanted valve and its position. Valvular complications have become less frequent with modern valves, resulting in increasing durability¹. For example, St. Jude's bi-leaflet valves have an estimated longevity of over twenty years.

Prosthetic Valve Thrombosis (PVT) is a rare condition that requires prompt cardiac care. PVT occurs in both bioprosthetic and mechanical heart valves with reported annual rates varying from 0.03% per year to as high as 5.7% per year². PVT should be suspected in patients with worsening cardiopulmonary symptoms, thromboembolic complications, and inadequate anticoagulation³.

Pathophysiology:

A single center, retrospective analysis of 152 patients who required re-operation after prosthetic valve implantation, identified the modes of valvular dysfunction that led to surgery. Most common was primary valve failure in 85 patients, followed by valvular thrombosis and valve-related thromboembolic episodes in 27 patients, paravalvular leak 25 patients, prosthetic valve endocarditis 14 patients, and hemolysis 1 patient⁴.

Early thrombotic events after surgery occur at a rate of 0.7% within thirty days after mechanical aortic valve implantation. Risk factors for early thrombotic events include heparin-induced thrombocytopenia and diabetes mellitus⁵. Late thrombotic events are most often due to sub-therapeutic anticoagulation². Pannus formation occurs over a prolonged time course, typically one to five years, and is caused by an inflammatory process and growth of granulation tissue around the valve. On ultrasound, pannus is typically localized to the suture line and, unlike thrombi, does not move independently of the valve apparatus. Fractured pannus may lead to a thrombotic cascade.

Presentation:

In the single center experience above, three patients presented in New York Heart Association functional Class II, 77 in Class III, and 61 in Class IV; the remaining 11 patients were emergencies and as a result, they were not classified.⁴ Prognosis is closely tied to the degree of congestive heart failure (CHF) at presentation, with those in Class III and IV CHF requiring immediate surgery per the ACC/AHA guidelines⁶.

Diagnosis:

Echocardiography is often the initial imaging tool utilized in assessing valvular structure and function. Fluoroscopy is a simple and accurate method to assess prosthetic valve leaflet mobility⁷. Although not widely used for the evaluation of prosthetic valves, cardiac CT may provide additional information due to its post-processing features. Nevertheless, differentiating thrombus versus pannus remains difficult, and current methods are often inconclusive.

Management:

Several factors should be considered when managing patients with PVT. These factors include local expertise, risks of surgery and thrombolytic drugs (hemorrhagic and embolic complications), functional class, and the probability of achieving valve re-opening^{3,8}. There are only few studies comparing fibrinolytic therapy versus surgical intervention in patients with PVT. Surgical intervention remains the treatment of choice in emergent settings, when patients present with shock from an abrupt decline in cardiac output. Furthermore, surgery is recommended in patients with left-sided PVT and Class III and IV CHF, and in patients with left-

sided PVT and a large clot burden⁶. On the other hand, thrombolysis has been gaining acceptance, especially when the thrombus burden is small⁶.

Thrombolysis may also be considered in situations when surgery is not available or the patient is not an optimal surgical candidate^{6,9}.

Prognosis:

Obstructive PVT is a serious complication in patients with prosthetic heart valves. In the single center experience listed above, the 30-day operative mortality was 14.5%.⁴ The 5-year and 10-year survival rates were 75% and 63%, respectively.⁴

Preoperative functional class and the mode of valve dysfunction significantly affected early and late mortality.⁴ In addition, multiple prior surgeries may increase mortality rate. Some recent studies have suggested a lower mortality with thrombolysis when compared to surgery.⁹ Thus, the optimal treatment modality remains controversial with current guidelines continuing to recommend surgery as first line therapy for left-sided PVT.

Conclusion:

In summary, PVT is a complication of prosthetic valves that requires emergent intervention, either with surgery or thrombolysis. Echocardiography and fluoroscopy are appropriate diagnostic modalities. A low index of suspicion should be maintained when patients with prosthetic valves present with heart failure symptoms and inadequate anticoagulation.

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