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Case Report

A rare cause of dysfunction of mechanical aortic valve prosthesis in a young adult

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Abstract

A mechanical prosthetic valve dysfunction carries a high risk of hospitalization and patient death. It may show up as stenosis of the valve prosthesis or intra/paraprosthetic regurgitation. Prosthetic stenosis, in turn, may be caused by granulation tissue (pannus), thrombosis, a combination of pannus and thrombosis, a prosthesis mechanical defect, or a unique surgical technique. Establishing the specific cause of prosthetic stenosis is of crucial importance for the therapeutic behavior. Prosthetic thrombosis requires conservative intensification of anticoagulation/fibrinolysis or surgical treatment in an accelerated/emergency manner. When the cause of the thrombosis is pannus, a mechanical defect, or surgical technique, the only treatment option is cardiac surgical reintervention.

The manifestation of granulation tissue (pannus) is indicative of a non-immune reaction to the prosthetic valve and suture material following prosthetic valve replacement. The basis of this phenomenon is the growth of fibroelastic tissue associated with an increase in the expression of transforming growth factor beta.[1] In a small proportion of patients with implanted prosthetic valves, the growth of pannus leads to subvalvular obstruction of blood flow and results in prosthetic stenosis. Despite the technological advancements in imaging diagnostics, visualizing pannus remains challenging and not always feasible. Consequently, we employ a multifaceted approach that incorporates anamnestic, clinical, and imaging methods to reach a diagnosis. Operative exploration is the only method by which definitive confirmation can be obtained.

The objective of this article was twofold: first, to present a rare case of stenosis of a mechanical aortic valve prosthesis due to a circumferential shaft of granulation tissue (pannus) forming a kind of de novo subaortic membrane; and second, to outline an algorithm for the diagnosis of prosthetic valve stenosis and differential diagnosis between thrombosis and pannus as the cause of valve dysfunction.

The following case study presents a patient who met the surgical treatment indications due to stenosis of a mechanical aortic valve prosthesis. The clinical information and imaging studies indicated that the prosthetic dysfunction was likely due to the presence of granulation tissue (pannus). The intraoperative finding revealed a circumferential shaft of granulation tissue, forming a novel subaortic membrane.

Keywords

mechanical prosthetic valve dysfunction, pannus, transthoracic echocardiography, transesophageal echocardiography.

Introduction

Globally, valvular heart disease affects about 100 million people. Aortic and mitral valve disease affects approximately 2.5% of the general population, with the incidence reaching 10% in those over the age of 75. The aging of the population will result in an exponential increase in the number of patients suffering from valvular heart disease in the coming decades. For the past 70 years, replacing or repairing the damaged valves through surgery has been the accepted treatment for valvular disease. There are two types of prosthetic valves: mechanical heart valves (MHVs),

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which are more durable but more thrombogenic, and bioprosthetic heart valves (BHVs), which have more physiological hemodynamic parameters but are less durable over time.[2] All implanted foreign bodies, including prosthetic valves, are thrombogenic and require short-term (BHVs) or long-term (MHVs) anticoagulation to prevent valve thrombosis, which can lead to severe hemodynamic disturbances and debilitating or even fatal ischemic stroke.[3] As a rule, mechanical prosthetic valves are implanted in younger patients and patients without significant comorbidities, in whom life expectancy is long, since the implantation of BHVs in this population would mean almost mandatory re-intervention in the midterm.[2]

All patients with implanted prosthetic valves are subject to long-term follow-up by a cardiologist, which routinely includes transthoracic echocardiography to assess the structure and function of the prosthetic valves and exclude prosthetic dysfunction.

Objective

The objectives of the study were to present a clinical case of a patient with dysfunction of a mechanical prosthetic valve – prosthetic stenosis due to a subaortic membrane made of granulation tissue (pannus) – and to outline a diagnostic and therapeutic algorithm for stenosis of a mechanical prosthetic valve.

Case report

We hereby present a case of a 25-year-old male patient who underwent cardiac surgery for aortic valve dysplasia in 2006 at the age of 8. The patient had been implanted with a mechanical prosthetic valve (AVR Medtronic Advantage Size 23, bileaflet mechanical heart valve). He was subsequently monitored by a pediatric cardiologist until 2020, and the parameters of the mechanical valve remained within the normal ranges established by the manufacturer. The patient meticulously monitored his intake of Sintrom, with the time spent in the therapeutic range exceeding 70%, as indicated by laboratory test results. Since 2020, routine echocardiographic studies have demonstrated a consistent increase in the peak systolic velocity (Vmax) of the transaortic blood flow, the peak systolic gradient (PSGr), and the mean systolic gradient (MnSGr) through the aortic valve prosthesis. In 2024, the patient was referred to the Department of Cardiac Surgery for evaluation of the valve function and assessment of indications for surgical treatment.

From the medical history, it became clear that in the last 12 months, the patient had stopped amateur sports due to a notable reduction in his functional capacity, but there were no complaints during daily activities. It was assumed that he had heart failure, NYHA class I. His physical status and clinical examination were normal, including a characteristic click of a mechanical prosthesis on auscultation. The transthoracic echocardiogram showed preserved dimensions and volumes of all cardiac chambers, preserved left ventricular systolic and diastolic function, and normal function of the mitral, tricuspid, and pulmonary valves. Abnormalities included:

- *The size of the tubular ascending aorta: 48 mm, with normal dimensions of all other aortic segments* ***(Fig. 1)****.*
- *Doppler parameters of the aortic valve prosthesis were as follows: V max: 3.8 m/s, PSGr: 58 mmHg, MnSGr: 35 mmHg, AVR acceleration time (AT): 107 ms, AVR dimensionless valve index (DVI): 0.2, ejection time: 303 ms, AVR velocity time integral: 85 cm, AVR EOA: 0.8 cm^2, AVR acceleration time/ejection time: 0.4, LVOT VTI: 16 cm, LVOT diameter: 23 mm. Due to the hinges of the mechanical prosthesis and as anticipated with this prosthesis design, aortic regurgitation outside of the so-called wash jets was not recorded.*

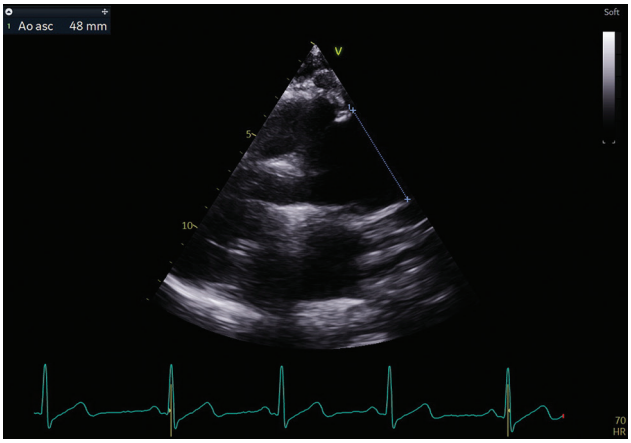

Figure 1. Transthoracic image displaying an aneurysm of the tubular part of the ascending aorta.

A review of the manufacturer's information on the prosthetic valve showed the normal parameters for this size and model. Specifically, the effective orifice area (EOA) is 2.2±0.3 cm^2, and the mean systolic gradient is 10.4±3.1 mmHg, which suggested clearly that the patient's prosthetic valve parameters were not within the normal range. The differential diagnosis included prosthetic valve stenosis, prosthesis/patient mismatch, hyperdynamic circulation, and pressure recovery phenomenon. In accordance with the established algorithm for evaluating increased transaortic prosthetic blood flow velocity, the following criteria were met: Vmax>3 m/s, DVI<0.25, and AT>100 ms. Based on these findings, it was hypothesized that the primary cause of the observed increased velocity was prosthetic aortic valve stenosis.

Thrombosis of the mechanical prosthetic valve and pannus, reducing the amplitude of the leaflet opening, were discussed as potential causes of the stenosis.

As clarifying methods, the following were used:

- *Transesophageal echocardiography – accelerated transvalvular blood flow was confirmed, especially in close proximity to the hinges of the prosthesis* ***(Fig. 2)****, aortic regurgitation and pathological formations captured by*

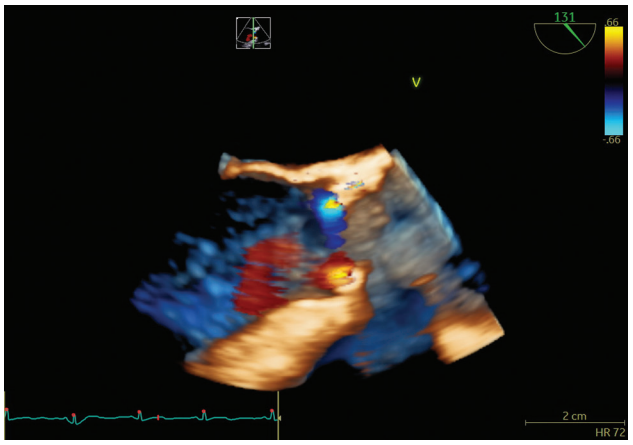

Figure 2. A 3D transesophageal image showing acceleration of flow in the LVOT.

the valve prosthesis were not visualized. The movement of the leaflets was not visualized due to the phenomenon of acoustic shadowing, due to the profile of the mechanical valve prosthesis.

- *Cinefluoroscopy – normal amplitude of movement of the leaflets of the mechanical aortic valve prosthesis was demonstrated (**Fig. 3**, systole; **Fig. 4**, diastole).*

Due to the long interval between the index surgery and prosthetic dysfunction (17 years), the gradual increase in gradients, the patient's excellent compliance with Sintrom treatment, the absence of thrombotic masses on transesophageal echocardiography, and the normal amplitude of movement of the leaflets of the mechanical aortic valve prosthesis on cinefluoroscopy, it was assumed with a high probability that the prosthetic dysfunction was due to pannus. Due to contraindications for thrombolysis in suspected pannus, AVR EOA 0.8 cm², a diameter of the tubular part of the ascending aorta >45 mm, and symptoms of heart failure, a decision was made for surgical re-intervention—operation a modo Wheat was performed: re-replacement of the aortic valve with a mechanical prosthesis and prosthesis of the ascending aorta.

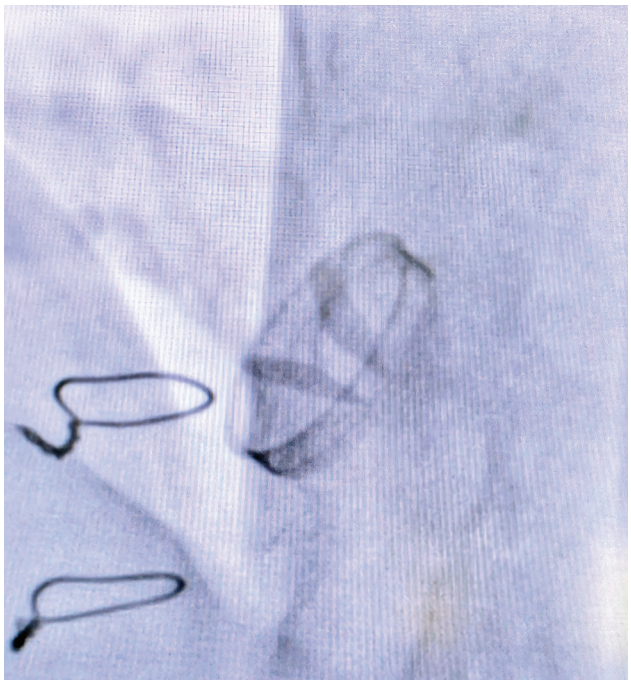

Figure 3. A fluoroscopic image displaying preserved leaflet motion in systole.

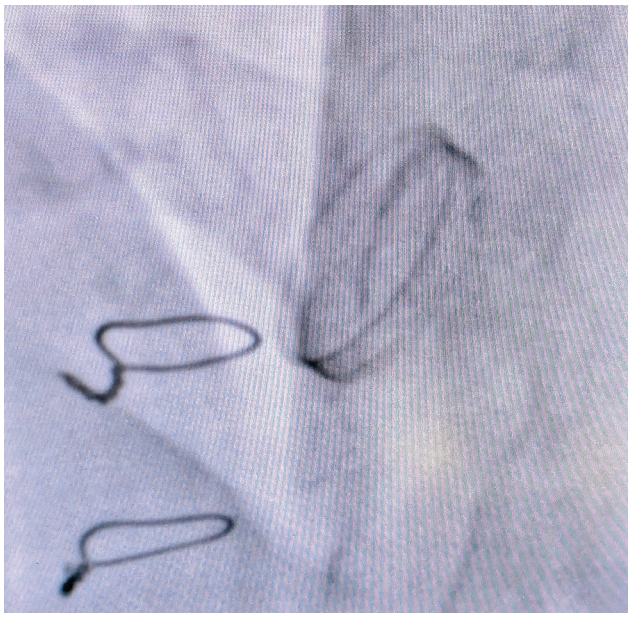

Figure 4. A fluoroscopic image displaying preserved leaflet closure in diastole.

*Intraoperative macroscopic findings included, from the aortic perspective, a mechanical valve prosthesis free of thrombosis and pannus **(Fig. 5)** and normal mobility of the leaflets. After removal of the valve, a circumferential shaft of connective tissue (pannus) with cartilaginous density was found underneath it (on the side of the outflow tract of the left ventricle), forming a kind of neo-subvalvular membrane within the suture ring of the valve prosthesis **(Fig. 6)**.*

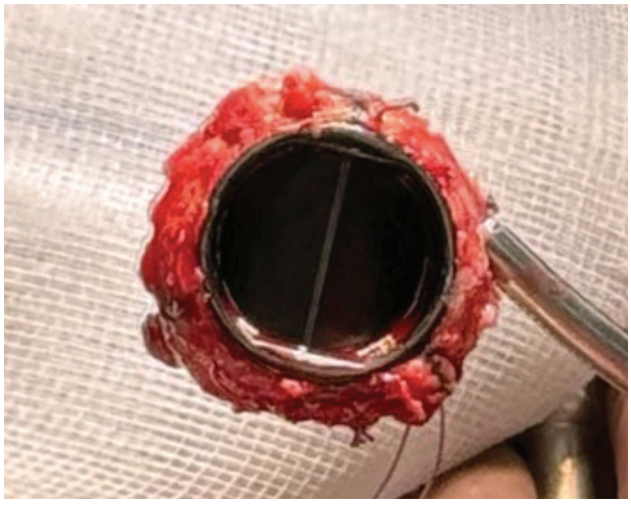

Figure 5. Intraoperative image of the extracted mechanical prosthesis from aortic perspective, showing no visible thrombi, obstructing the blood flow through the prosthesis.

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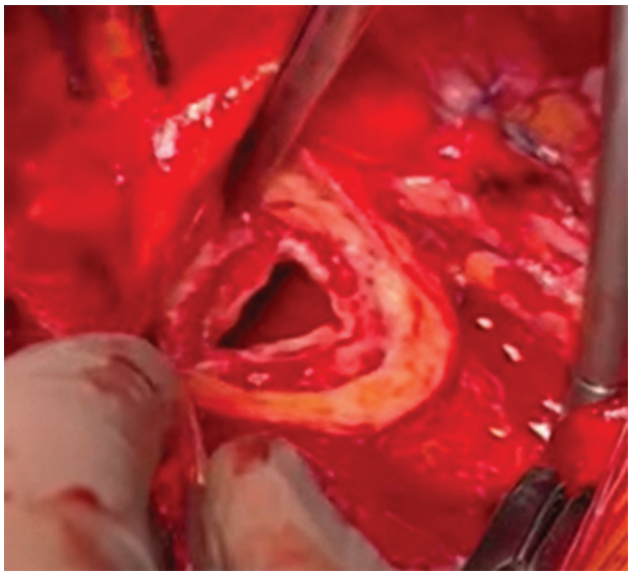

Figure 6. Intraoperative image of pannus formation in the LVOT, just proximal to the prosthesis, obstructing the blood flow.

A Wheat operation was performed, and a new mechanical valve prosthesis, SJM Regent Size 25 (bileaflet mechanical heart valve), was implanted. The postoperative period was uneventful, and the patient was discharged on the seventh postoperative day.

Discussion

Dysfunction of a mechanical prosthetic valve refers to its inability to perform its function—to ensure unidirectional blood flow without obstructing the passage of blood through it. The causes of dysfunction are generally two: incomplete opening of the prosthesis—because of thrombosis or pannus limiting the mobility of the plates or hinges of the prosthesis—or the return of blood in the opposite direction to the normal one through the valve prosthesis (intraprosthetic regurgitation) or past the valve prosthesis (periprosthetic regurgitation).

The obstruction of blood flow through the mechanical valve prosthesis—prosthetic valve stenosis—can be due to thrombosis, growth of granulation tissue (pannus), a combination of thrombosis and pannus, incorrect orientation of the plates of the prosthesis, excessively long ends of the nodes with which it is fixed, and some endogenous factors.[4]

The imaging modalities used to detect prosthetic valve stenosis are as follows: transthoracic echocardiography, transesophageal echocardiography, cinefluoroscopy, and computed tomography. Transthoracic echocardiography is a screening method for monitoring the structure and function of prosthetic valves. The algorithm for diagnosing prosthetic aortic stenosis includes measuring peak velocity (Vmax) across the prosthesis. Acceleration time (AT) and the so-called dimensionless index (DVI) should be measured at peak velocity greater than 3 m/s. Prosthetic aortic stenosis can be diagnosed with high probability at Vmax>3 m/s, DVI<0.25, and AT>100 ms.[5]

Transesophageal echocardiography provides additional information on the mobility of the leaflets of the valve prosthesis, the presence of periprosthetic regurgitation, and structures impeding the movement of the leaflets—thrombotic masses, vegetations, and pannus.

Cinefluoroscopy allows the mobility of the leaflets of mechanical valve prostheses to be observed and the opening angle to be compared with the factory one, according to information from the manufacturer of the specific model of mechanical valve prosthesis.

Establishing the exact cause of the prosthetic stenosis is of crucial importance for determining the correct therapeutic approach—conservative treatment (intensification of anticoagulation, fibrinolysis) or surgical treatment (most often valve re-replacement)—as well as for the timing of the procedure (emergency or urgent in case of prosthetic thrombosis, urgent or planned in case of pannus growth).[6]

Computed tomography, and in particular multidetector CT technology (MDCT), has the ability to differentiate prosthetic valve thrombosis from pannus. When the density of the suspected periprosthetic mass exceeds 145 Hounsfield units (HU), the diagnosis of pannus can be made with high sensitivity and specificity. In such cases, patients should be referred directly for reoperation. When the density of the mass is less than 90 HU, the probability of successful fibrinolysis is high, and this should be the treatment of choice.[7]

The differential diagnosis between the two most common causes of prosthetic valve stenosis—thrombosis and pannus—is made taking into account clinical features and imaging findings[2] **(Table 1)**.

Conclusions

This case highlights a rare but significant cause of mechanical aortic prosthetic valve dysfunction in a young adult—subvalvular obstruction due to pannus formation mimicking a de novo subaortic membrane. The diagnostic challenge lies in differentiating pannus from prosthetic thrombosis, as their therapeutic approaches diverge dramatically. A comprehensive evaluation combining clinical history, echocardiographic data, fluoroscopy, and, when available, computed tomography is essential in guiding appropriate management. The gradual onset of symptoms, long interval since the initial surgery, preserved leaflet mobility, and absence of thrombotic masses favored the diagnosis of pannus in this patient, ultimately confirmed intraoperatively. Surgical reintervention, although complex, led to a favorable outcome. This case underscores the importance of vigilant long-term follow-up in patients with mechanical valve prostheses and illustrates a structured diagnostic and therapeutic algorithm for prosthetic valve stenosis to improve clinical decision-making and outcomes.

Table 1. Pathological, clinical, and imaging characteristics of prosthetic valve thrombosis versus fibrotic pannus ingrowth

	Prosthetic thrombosis	Granulation tissue ingrowth (pannus)
Clinical features	Shorter time from valve replacement to prosthetic	Longer time from valve replacement to prosthetic dysfunction (months to years)
	More often the onset of symptoms is sudden and distinct	More often the onset of symptoms is gradual and even imperceptible
	More often associated with suboptimal anticoagulation	Less often associated with suboptimal anticoagulation
Imaging findings	Larger volume and area of masses on the prosthesis detected by CT or TOE	Smaller volume and area of masses on the prosthesis detected by CT or TOE
	More often the masses are located on the atrial side of mitral prostheses and the aortic side of aortic prostheses	More often the masses are located on the ventricular side of both aortic and mitral valve prostheses
	More pronounced limitations in the mobility of the leaflets	Less or no restriction in leaflet mobility

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Additional information

Ethical statement

- The authors declared that no clinical trials were used in the present study.
- The authors declared that no experiments on humans or human tissues were performed for the present study.
- Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.
- The authors declared that no experiments on animals were performed for the present study.
- The authors declared that no commercially available immortalized human and animal cell lines were used in the present study.

Conflict of interest

The authors have declared that no competing interests exist.

Artificial Intelligence (AI) use

The authors accept full responsibility for the content of the manuscript, including the disclosure of any use of AI. No AI tools were used in the preparation of this manuscript.

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Data availability

All of the data that support the findings of this study are available in the main text.