

Negative results - Valves

Fatal early acute thrombosis of mechanical mitral prosthesis

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Abstract

Objective: To report a rare case of fatal early acute thrombosis of mechanical mitral prosthesis. **Methods:** A 62-year-old lady, who underwent closed mitral valvotomy for rheumatic heart disease 19 years ago, presented with Canadian Cardiovascular Society Class II angina and New York Heart Association Class III dyspnoea. Investigations including echocardiogram and cardiac catheterisation revealed mixed mitral valve disease. She had severe pulmonary artery hypertension with pulmonary artery wedge pressure of 35 mm of mercury. Significant stenosis in the midportion of dominant right coronary artery was also demonstrated. She underwent mitral valve replacement using mechanical tilting disc prosthesis, DeVega tricuspid annuloplasty and saphenous vein bypass graft to the distal right coronary artery. After having made satisfactory early postoperative progress, she suffered an asystolic cardiac arrest about 26 h postoperatively. **Results:** The patient succumbed despite all resuscitative measures. Autopsy revealed an adherent thrombus occluding the atrial surface of the minor orifice of mechanical prosthesis, which prevented the valve from functioning. **Conclusion:** To the best of our knowledge, this is the first case of prosthetic valve thrombosis (PVT) occurring on the first postoperative day in a patient with a mechanical valve prosthesis, without any circulatory assist device.

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Keywords: Heart valve; Mechanical; Prosthesis; Thrombosis; Valve disease

1. Introduction

Prosthetic valve thrombosis (PVT) is a rare but life threatening complication of mechanical heart valve prosthesis, the incidence varying from 0.5 to 6% [1]. Diagnosis is difficult and almost 50% of obstructed valves are diagnosed at autopsy [2]. There are only a few cases of this nature reported in literature.

2. Case report

A 62-year-old lady had undergone closed mitral valvotomy 19 years ago. She represented with Canadian Cardiovascular Society Class II angina and New York Heart Association Class III dyspnoea. Clinical examination found her to be in atrial fibrillation, having mixed mitral valve disease and signs of tricuspid regurgitation with hepatic enlargement and cardiac cachexia.

Transthoracic echocardiography revealed moderate mitral stenosis with regurgitation and severe tricuspid regurgitation. The left ventricular function was preserved, the cardiac chambers were dilated and there was no mural thrombus. Cardiac catheterisation demonstrated mixed mitral valve disease. Pulmonary artery and pulmonary artery wedge pressures were 60/30 and 35 mm of mercury,

respectively. She had significant stenosis in the midportion of the right coronary artery.

Using moderate hypothermic cardiopulmonary bypass and intermittent antegrade cold blood cardioplegia, she underwent transeptal mitral valve replacement. A size 27 mm Ultracor (AorTech Europe Ltd, Lanarkshire, Scotland, UK) mechanical tilting disc prosthesis was inserted using 2/0 prolene continuous suture technique, with minor orifice oriented posteriorly. The posterior leaflet and subvalvular apparatus were preserved. No thrombus was seen on the left atrial wall or the atrial appendage. The left atrial appendage was excluded surgically. DeVega tricuspid annuloplasty and saphenous vein graft anastomosis to the distal right coronary artery were also performed. Aprotinin (Trasylol-Bayer PLC, Newbury, Berks, UK) was used as per Hammersmith protocol.

She came off cardiopulmonary bypass in atrial fibrillation and on modest inotropic support with a cardiac index of 3.7 l min⁻¹ m² and pulmonary artery wedge pressure of 14 mm of mercury. She was extubated 12 h later, on first postoperative day and maintained normal blood gases. The INR, APTT ratio and platelet count were satisfactory with values of 1.9, 1.63 of control and 242,000 cm², respectively. Approximately 26 h after the operation, she suffered an asystolic cardiac arrest. She had been progressing satisfactorily, 15 min before arrest the cardiac index was 2.5 l min⁻¹ m², systemic vascular resistance at 845 dynes s⁻¹ cm² and pulmonary artery wedge pressure of 26 mm of mercury. Resuscitation was carried out, including re-exploration of

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Fig. 1. Artistic impression of the explanted valve at autopsy. Thrombus is shown occluding the valvular mechanism.

the chest. No features of cardiac tamponade were found and she succumbed, despite all efforts.

Autopsy revealed a patent and intact vein graft to the right coronary artery.

The atrial surface of the minor orifice of mitral prosthesis was occluded by an adherent thrombus (Fig. 1), which also interfered with the mobility of the disc of the valve. The left atrium itself was free of thrombus. The subvalvular apparatus did not seem to affect the excursion of valve leaflet.

3. Comment

Mechanical valvular prostheses have the advantage of longevity, but carry a risk of thrombosis, dependent on valve design, materials and host-related factors. While pannus is common to both biological and mechanical valves, acute thrombosis is mostly a complication of mechanical valves. Prosthetic valve thrombosis (PVT) is a rare but devastating complication. The reported incidence of PVT varies widely from 0.5 to 6% per patient year in the aortic and mitral valve position depending on the adequacy of anticoagulation and valve design [1]. Massive thrombotic obstruction is rare with bileaflet design [3].

Guglielmi and colleagues [4] demonstrated that the relative risk of thrombosis was 12 times higher for the tricuspid prosthesis and seven times higher for the mitral prosthesis. In their multivariate analysis they showed a 67% risk reduction with larger prosthesis (> 27 mm), a 69% risk reduction with sorin tilting disc prosthesis and an 83% risk reduction with bileaflet prosthesis.

Renzulli and colleagues [5] demonstrated a 5-year actuarial incidence of obstruction to be 6.08%. Significant risk factors in their study included tilting disc prosthesis, prosthesis without pyrocarbon coating, large prosthesis, tilting disc prosthesis with small orifice oriented posteriorly, atrial fibrillation, enlarged left atrium, time from implant greater than 4 years and age between 40 to 50 years.

Shachar and colleagues [6] found obstruction of mitral prosthesis by bulky mural thrombus originating at the septal wall of the left atrium. They suggested that the process of postoperative mural thrombosis of the septal and posterior

walls of the left atrium might result from trauma to these structures during operative procedure.

Other factors known to precipitate a thrombotic process are coagulation abnormalities like protein C, protein S and antithrombin III deficiency [7], left ventricular dysfunction or low cardiac output state [8]. The importance of subvalvular apparatus and the preservation of annuloventricular continuity in mitral valve replacement is well documented. However, complications such as chordal entrapment of bioprosthesis [9] has been reported which can initiate a thrombotic process.

The earliest reported incidence of PVT is 4 days post-operatively [10]. A case of bioprosthetic PVT is reported on the first postoperative day but in this case a circulatory assist device was used and thrombosis was related to low transvalvular flow [8].

In our patient it is difficult to conclude the aetiology of PVT. She had risk factors of atrial fibrillation and tilting disc mechanical valve in mitral position. Since coagulation abnormalities were not suspected preoperatively, investigations like protein C, protein S and antithrombin III deficiency were not done.

4. Conclusion

The incidence of PVT has decreased in recent years as a result of improved design and haemodynamic profile of valvular prosthesis. Thrombosis, however, remains a devastating complication. This case highlights the need for awareness among clinicians for the possibility of valve thrombosis in the early postoperative period.

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