

Surgical thrombectomy without valve removal for the treatment of acute mechanical valve dysfunction due to thrombosis

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Abstract

Background: Prosthetic valve thrombosis is one of the most serious long-term complications after heart valve replacement, thrombosis can cause the obstruction of prosthetic valve leaflet, which leads to acute valve dysfunction. Reports on specific surgical methods currently for surgical treatment of prosthetic valve thrombosis are rare.

Case presentation: A 61-year-old elderly woman came to our hospital's ICU due to thrombosis on the artificial mitral valve, causing the stuck of valve leaflets, acute heart failure, and heart function class IV. After the diagnosis was confirmed, emergency surgery was given. During the operation, we did not remove the original artificial valve, but only removed the thrombus on the valve leaflets, and then the valve function returned to normal. The patient recovered well and has been in good health since follow-up.

Conclusion: In this case, we used in situ thrombectomy to treat the artificial valve dysfunction caused by thrombosis, shortening the operation time and extracorporeal circulation time, reducing the probability of postoperative adverse events to a certain extent, and also reducing the patient Economic burden. Further investigations are required to confirm the clinical effectiveness and safety of this operation method.

Keywords

Thrombosis; Prosthetic valve dysfunction; Surgical treatment.

Case Presentation

The patient was a 61-year-old-55 kg woman with a history of the "Mitral Valve Replacement (St Jude Prosthetic valve) + Coronary Artery Bypass Graft (LIMA-LAD)" in our hospital 4 years ago. She did not regularly take warfarin and Other anticoagulant drugs; "Thrombus Removal of both lower Extremities" was performed in our hospital 4 months ago. Due to sudden chest tightness and dyspnea 5 days ago, she was

treated by a local hospital. Then, her condition worsened and she was transferred to our cardiac intensive care unit.

Investigation

On arrival, the patient was fully conscious but with the assist of a ventilator in breathing. Her respiratory rate was 100 cycles/min, with coarse crepitations heard over all lung zones. Significant systolic and diastolic murmurs of 3/6 level can be heard in the precordial area. Blood tests showed international normalized ratio (INR) 7.52, Prothrombin time (PT) 88.00s, Activated Partial Thromboplastin time (APTT) 36.70s, D-dimer: 0.96mg/L (DDU); Fibrin (pro) degradation product: 6.43 mg/L. pro-BNP: 14008.0 pg/ml, Troponin I was 0.476 ug/l, creatinine 84 mmol/l, ALT 595(n=0-40 U/L) and CRP 67. Full blood count showed Hb 97 g/l, WBC 9.50×10^9 /l. ECG: inferior myocardial infarction-like changes; ST-T segment changes; Q-T interval prolongation. A transthoracic echocardiogram was urgently performed, which demonstrated that double room enlargement, left atrium about 44 mm, right atrium about 42 x 53 mm; prosthetic mitral valve leaflets are poorly opened and closed; the prosthetic mitral valve is moderately stented and moderately closed Insufficiency; severe tricuspid regurgitation; pulmonary hypertension (moderate 62 mmhg). We consider the patient's mitral valve prosthetic valve jam caused by thrombosis and arrange emergency surgery for it. Considering the patient's coagulation status, 4 units of vitamin K were given intramuscularly before surgery.

Treatment and outcome

After successful general anesthesia, a TEE probe was inserted. A secondary median sternotomy was performed and division of adhesion was carefully to prevent the heart from bleeding and rupture. The ascending aorta was blocked, and myocardial protective fluid was infused through the aortic root, and the heart stopped beating satisfactorily, Cardiopulmonary bypass was established successfully. Open the right atrium and the interatrial septum, the St Jude mitral valve was exposed. Extensive thrombi and granulation tissue were revealed overlying the valve and interfering with the movement of the leaflets (Figure 1). There was no evidence of infection. The tricuspid valve annulus is enlarged to accommodate four fingers. For the consideration of economic factors and at the same time to shorten the time of cardiopulmonary bypass, we adopted thrombectomy instead of valve replacement. Without removing the mechanical valve, we entered the left atrium through the interatrial septum and the left ventricle through the aortic valve after cutting the aortic root respectively, so that the thrombus around the artificial mitral valve can be remove completely. After all the thrombus were removed (Figure 2), the Tricuspid valvuloplasty was performed with Bairensi Tricuspid Angioplasty Ring. The patient was then taken off bypass with a ventilator and inotropic support and transferred to the cardiac intensive care unit. There were no complications and the patient was discharged home on day 20. The patient recovered smoothly after the operation, assisted by mechanical ventilation for 2 days, stayed in the intensive care unit for 3 days, and was discharged home on day 35. The pathological examination report for valve vegetation suggests: thrombosis (Figure 3).

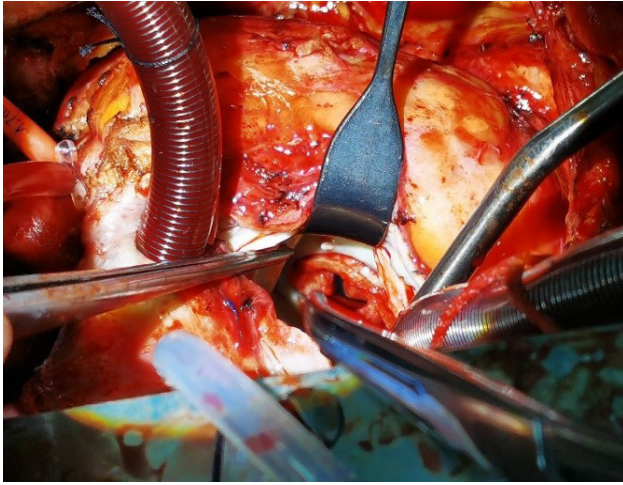


Figure 1: Through the interatrial septum, we can see thrombus growing around the prosthetic valve leaflets, the valve orifice area is very small, only about 5 mm.



Figure 2: Thrombus removed.

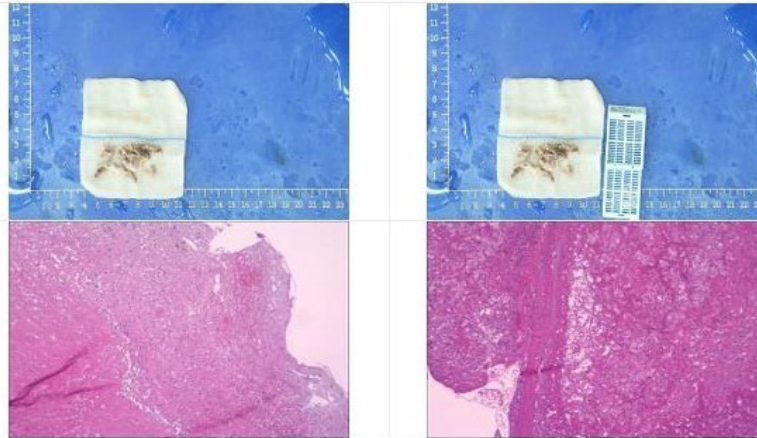


Figure 3: The pathological examination report for valve vegetation suggests: thrombosis.

Discussion

Prosthetic valve thrombosis is one of the major cause of primary valve dysfunction, which can be life-threatening [1]. According to reports, the incidence of PVT for bioprosthetic valves is 0.03%, for mechanical valves in the position of the mitral and aorta valve are 0.5%-8%, and as high as 20% in mechanical tricuspid valves [2]. Prosthetic valve thrombus can cause reduced leaflet motion or impaired leaflet coaptation, leaflet thickening, reduced or increased effective prosthesis orifice area (leading to either stenosis or insufficiency as the primary valve defect, respectively), increased transvalvular gradient or transvalvular regurgitation, with or without development of valve-related symptoms [3]. The most common cause of prosthetic valve thrombosis is inadequate anticoagulant therapy. Even with the use of VKA, the risk of thromboembolism is 1%-2% per year, but the risk is considerably higher without or inadequate treatment with warfarin[4]. The cause of this case is obvious for the patient did not take warfarin for anticoagulation treatment after her first valve replacement.

PV thrombosis is a pathological entity characterized by thrombus formation on the prosthetic struc-

tures, with subsequent PV dysfunction with or without thromboembolism. This disease needs to be differentiated from the following conditions: fibrotic pannus ingrowth; PV degeneration; and PV endocarditis with vegetation formation [5]. However, in most cases, these situations usually occur at the same time. Patients may be asymptomatic or may present with different symptom, such as dyspnea, decreased exercise capacity, palpitation, chest pain, vertigo, cerebrovascular accident, embolic events, cardiogenic shock or even sudden death [2,6]. At the same time, stenotic or regurgitant murmurs may be heard during auscultation.

For diagnosis, Transthoracic echocardiography (TTE) is usually the first way to detect PVT, which is usually used for evaluation of severity of obstruction. Sometimes PVT may be missed during initial TTE examination [7]. On the contrary, thrombus can be clearly visualized by Transesophageal echocardiography (TEE), because of the proximity of the esophagus to the heart and absence of interference with lungs and ribs [8]. TEE has an indispensable value to assess thrombus size, mobility, and location, which may help in treatment decisions, such as thrombolysis, anticoagulation, and surgery.

The current recommended treatment options for PVT include thrombolysis and surgical treatment. According to the 2020 AHA/ACC guidelines, for patient with a thrombosed left-sided mechanical heart valve who present with symptoms of valve obstruction, urgent initial treatment with either slow-infusion, low dose fibrinolytic therapy or emergency surgery is recommended [9]. The decision should be individualized after review by the heart valve team, several factors support surgery: Low surgical risk, Contraindication to fibrinolysis, Recurrent valve thrombosis, NYHA class IV, Large clot(>0.8 cm²), Other valve disease, Possible pannus, and LA thrombus [9].

In this case, the diagnosis was acute prosthetic valve obstruction by thrombus. The patient had a thromboembolism in the arteries of the lower extremities in the previous few months, which should arouse our vigilance. Unfortunately, no doctors related to cardiac surgery noticed her condition at that time. When the patient finally entered the ICU, acute respiratory failure had already occurred, with NYHA class IV, and coagulation disorders. After the discussion of heart valve team about this patient, we urgently performed the operation on the patient. Given the condition of this patient, to shorten the operation time, we removed the thrombus without removing the prosthetic valve, instead of re-do mitral valve replacement with longer time and higher risk. In the end, the operation went smoothly and there were no complications until the patient was charged home.

Conclusion

Acute prosthetic dysfunction caused by thromboembolism is highly fatal and need to be diagnosed and intervened in time. Manage therapy needs to be individualized, when dealing with this situation, we should make the most reasonable decision based on the specific situation of the patient. As so far, the specific methods of PVT surgical treatment are rarely reported in the literature. We hope that through this successful management, we can provide experience for others' treatment. Regardless of theoretical cause, it is important to recognize that even without clear echocardiographic evidence of valve thrombosis, patients should be taken to the operating room expediently if the overall clinical picture warrants it.

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