Case Report

Prosthetic Valve Endocarditis Complicating Acute Obstruction of a Mechanical Mitral Valve Successfully Treated with Antibiotics and **Thrombolytic Therapy**

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Abstract: The incidence of either mechanical heart valve thrombosis or prosthetic valve endocarditis is rare but associated with high mortality and the traditional treatment has been emergent reoperation. A 21-year-old man with an obstructed Carbomedics prosthetic mitral valve complicated by prosthetic valve endocarditis was reported. He was successfully treated with thrombolytic agents and antibiotics. Further studies are warranted to elucidate the incidence of mechanical valve obstruction associated with prosthetic valve endocarditis and to compare the efficacy between medical and surgical therapy.

Key Words: Mechanical heart valve obstruction: Prosthetic valve endocarditis.

Introduction

Mechanical heart valve thrombosis with obstruction is relatively rare. It has an annual incidence from 0.1 to 0.5%, but is associated with a high mortality of 60 to 80%. In another aspect, prosthetic valve endocarditis is also an infrequent (incidence of 1 to 4%) but serious complication of cardiac valvular replacement and the mortality remains 42 to 77% despite intensive antibiotic therapy.² In either condition, the traditional treatment has been emergent reoperation, but conservative remedies that include thrombolysis and antibiotics have also been successful as reported in recent studies.^{3,4}

A number of risk factors for the development of

prosthetic valve endocarditis have been described, such as recipient of multiple valves, aortic prosthesis, mechanical than bioprosthesis, male sex, prolonged cardiopulmonary bypass time, antecedent native valve endocarditis and black race.⁵ In this report, we suggest that prosthetic malfunction may also predispose to the development of endocarditis, which has not been stated in previous literature.

We describe a case of successful thrombolysis of an obstructed Carbomedics prosthetic mitral valve in a young man with rheumatic heart disease in whom prosthetic valve endocarditis developed later and was subsequently eradicated by antibiotics.

Case Report

A 21-year-old man with a history of rheumatic fever as a child complained of chest pain and exer-

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tional dyspnea 1 and 1/2 years before admission. Echocardiography at that time showed severe mitral and moderate tricuspid regurgitation and the patient underwent mitral valve replacement with a Carbomedics prosthesis. There was no surgical complication. The patient was apparently well over the ensuing year, except that frequent occurrence of sore throat was complained. Three days before admission, he had progressive dyspnea and palpitation. The patient was hospitalized for evaluation and on admission his prothrombin time was 13.6 seconds, whereas control was 12.5 seconds. Before admission he took alternatively 2.5 and 5 mg warfarin daily, but he had been irregularly followed up due to poor compliance.

On examination the patient was conscious, tachypneic and afebrile, with mildly pitting edema of the lower extremities and remarkable jugular venous engorgement. Blood pressure was 110/84 mmHg and the pulse was 100 beats/minute. There was coarse breathing sound with moist rales over both lung fields. Prosthetic valve clicks were muffled regularly with no murmurs. The liver and spleen were impalpable and no mass was detected. The ranges of motion of the extremities were normal and the peripheral pulses were intact. Neurological examination was unrevealing. The complete blood count, blood chemistry profile and the urine test were normal. The chest radiograph showed cardiomegaly and pulmonary edema. An electrocardiogram revealed sinus tachycardia and left ventricular hypertrophy.

Five days following admission to the ward, the patient experienced fever, cough and sore throat and Janeway skin lesions were observed on the right foot five days following thrombolytic therapy. Throat swab culture showed Klebsiella pneumoniae and symptoms of tonsillitis were relieved with the use of cefazolin and gentamycin in 3 days.

Transthoracic echocardiography upon admission and transesophageal echocardiography (Fig. 1, top) after 5 days revealed a markedly diminished excursion of the leaflets of the mitral prosthesis. Doppler examination showed a peak velocity across the mitral prosthesis of 2.7 m/sec, with an estimated pulmonary arterial pressure of 46 mmHg, a mitral pres-

sure half time of 376 msec and a mitral valve area of 0.6 cm². There were also minimal to mild aortic and tricuspid insufficiency. However, no thrombus or vegetation was found. Under the impression of mitral prosthetic malfunction with severe mitral stenosis and congestive heart failure with pulmonary edema. probably due to poor anticoagulation and clotting on the prosthesis, the patient was treated with heparin and transferred to the intensive care unit for treatment with tissue-type plasminogen activator (t-PA). He received 100 mg t-PA intravenously as a continuous infusion over two hours. Immediate improvement of the patient's symptoms followed, respiration became less labored, heart rate decreased, diuresis appeared and prosthetic valve clicks were clearly audible. Echocardiography after 2 days (Fig. 1, bottom) in the



Fig. 1. Transesophageal echocardiograms showing mitral prosthesis in diastole before (top) and after (bottom) thrombolysis. LA = left atrium; LAA = left auricle; LV = left ventricle; MP = mitral prosthesis.

intensive care unit showed improved extent of tilting of leaflets. There were decreases in the peak velocity across the prosthesis (from 2.7 to 1.5 m/sec), the estimated pulmonary arterial pressure (from 46 to 34 mmHg) and the pressure half time (from 376 to 97 msec), respectively. A mitral valve area of 2 cm² was measured. Intravenous heparin and oral warfarin therapy were continued and the patient was transferred to an ordinary ward for further management.

Three days later, his body temperature elevated again, with chills and general malaise and the white blood cell count increased. A transesophageal echocardiography (Fig. 2, top) performed 4 days later revealed a highly mobile small vegetation of 6 mm at-

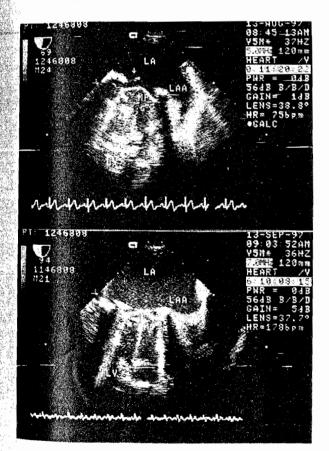


Fig. 2. Transesophageal echocardiograms showing the vegetation (arrow) arising from the prosthetic valve annulus in systole before (top) and after (bottom) treatment with penicillin. LA = left atrium; LAA = left auricle; LV = left ventricle; MP = mitral prosthesis.

tached to the annulus of the mitral prosthetic valve and a small amount of paravalvular regurgitation. However, movement of the mitral prosthesis was still good, with a mitral valve area of 2.2 cm². Blood cultures showed no growth. A diagnosis of prosthetic valve endocarditis was made based on the criteria for diagnosing infective endocarditis by Lukes and Durack⁶ and penicillin G at 12 million units daily was administered. The patient's fever disappeared after 7 days. Tonsillectomy was advised for his chronic tonsillitis and the patient received the procedure. Another transesophageal echocardiography (Fig. 2, bottom) performed after four weeks of therapy revealed no vegetation. The patient was discharged on a regimen of oral anticoagulant and still remains well.

Discussion

Since either condition of mechanical heart valve thrombosis and prosthetic valve endocarditis is rare but has a high mortality rate, ^{1,2} our patient may represent an even rarer case that deserves to be reported.

The cause of prosthetic valve obstruction involves formation of thrombus or pannus or both. Inadequate anticoagulation is a factor in 52 to 94% of patients with obstruction. Pannus is infrequent (6%) and patients with obstruction in the setting of therapeutic anticoagulation are probably found in this subset. In our patient, the cause of prosthetic valve obstruction was due to inadequate anticoagulation and hence likely thrombotic obstruction.

Echocardiography, both transthoracic and transesophageal, is a safe and convenient tool to detect abnormalities of prosthetic valves and the effect of thrombolytic therapy in patients with prosthetic valve obstruction. In our patient, the prosthetic valve movements, peak pressure gradients and mitral valve areas before and after thrombolytic therapy were clearly demonstrated and demarcated, respectively. This further suggests that echocardiography may be effective in the diagnosis and continued evaluation of thrombolysis and valve mobility in obstructed prosthetic valve patients.

Recent studies suggest that thrombolytic therapy is a safe and effective first-line approach to obstructed prosthetic valves.³ It has been reported that three quarters of patients gained improvement with thrombolysis while the risk of systemic embolization during thrombolysis was relatively small (15%) and such emboli were rarely of clinical importance.³ Our case further shows that recombinant t-PA can be used safely and effectively in patients with prosthetic valve obstruction, resulting in an immediate restoration of valve function and improvement of symptoms with no observed complication. Certainly, if thrombolytic therapy fails, operation should be considered.

Our patient was diagnosed with prosthetic valve endocarditis on clinical manifestations. The negative blood cultures in this case were probably due to antecedent antibiotic therapy for his chronic tonsillitis. It has been shown that only 63% blood cultures were positive in infective endocarditis patients with small vegetation of 5 mm⁸, as similar to our case. Moreover, this patient had positive echocardiography with oscillating intracardiac mass on valve and new valvular regurgitation, predisposing heart condition, fever and Janeway lesions. The above findings fulfilled the recent Duke criteria⁶ for diagnosing infective endocarditis. As prosthetic valve endocarditis appeared later after successful thrombolysis of prosthetic valve obstruction in our patient, we speculate that this prosthetic malfunction may predispose to the development of endocarditis, with the further possibility that the latter may otherwise lead to the development of prosthetic malfunction. Furthermore, since prosthetic valve endocarditis carries a very high mortality and needs protracted treatment, often involving operations, we suggest that antibiotics may be administered upon suspicion of it, or even prophylactically in patients with prosthetic valve obstruction.

In conclusion, we have successfully treated a case of mechanical mitral valve obstruction complicated by prosthetic valve endocarditis with the use of antibiotics and thrombolytic regimens. As recommended by Hurrell *et al.*⁹ patients with prosthetic

valve obstruction but who remain asymptomatic or have minimal symptoms (NYHA Class I or II) may initially be managed by intravenous heparin and aggressive warfarin therapy. If no response occurs or if symptoms progress (Class III), thrombolytic therapy may be used. If thrombolytic therapy is unsuccessful after a reasonable trial or if symptoms progress (Class IV), then reoperation may be performed. Further studies are warranted to elucidate the incidence of mechanical valve obstruction associated with prosthetic valve endocarditis and to compare the efficacy between medical and surgical therapy.

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機械人工瓣膜阻塞合併心內膜炎之病例報告

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摘要:機械人工瓣膜之阻塞或心內膜炎為罕見但致命的病灶,一般都需要緊急開刀治療。本文報導一個二十一歲男性被發現有機械人工瓣膜阻塞及心內膜炎,成功地以血拴溶解劑及抗生素治療。我們以內科治療機械人工瓣膜阻塞及心內膜炎,其發生率及與外科治療效果之比較值得進一步探討。

關鍵詞: 機械人工瓣膜阻塞; 心內膜炎。