

RECURRENT OBSTRUCTIVE MECHANICAL VALVE THROMBOSIS IN MITRAL POSITION

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We report a case of 37-year-old woman with mechanical prosthesis in mitral position with a history of recurrent prosthetic valve thrombosis (PVT). The patient was evaluated for hypercoagulable state and treated with streptokinase, tissue plasminogen activator and re-replacement of a bioprosthesis.

Key words: Prosthetic valve thrombosis, recurrent thrombosis.

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Valve obstruction is a life threatening complication of mechanical prosthesis. This case is of particular interest for the recurrent episodes and being the first reported patient with four times obstructive and one time non-obstructive prosthetic valve thrombosis (PVT) during 22 months.

CASE REPORT

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A 37-year-old woman was operated on 9 years ago for closed mitral commissurotomy for rheumatic mitral stenosis in another center. She had six children after her first operation.

She was operated on for mitral valve replacement (MVR) with a # 29 St Jude Medical prosthesis on May 26, 1996. Before the operation, the ECG revealed normal sinus rhythm and the echocardiographic examination revealed the presence of mitral restenosis with left atrial enlargement (50 mm) and pulmonary hypertension (systemic pressure of 60 mm Hg). In the early postoperative period, enoxaparine was given subcutaneously and converted to warfarin (international normalized ratio [INR] 2.5 to 3.5) and aspirin 300 mg/day was also started on the third postoperative day. Postoperative course was

uneventful. Transthoracic echocardiography (TTE) confirmed the normal prosthetic valve (PV) before discharge.

Six months after MVR, PVT occurred. Echocardiography confirmed an immobile leaflet of the PV with 36 mm Hg transvalvular gradient due to an obstructive thrombosis and a gross left atrial wall thrombus including left atrial appendage. Within 6 hours of admission, a course of streptokinase was given as IV infusion over 30 minutes at a dose of 1,500,000 IU and continued for 24 hours at 100,000 IU/h followed by heparinization. After thrombolytic therapy, normal PV function was restored. The transesophageal echocardiographic (TEE) examination revealed the complete lysis of either PV or left atrial thrombosis without any nidus of thrombus.

Four months later, second episode of thrombosis was observed and TEE revealed a huge obstructive thrombus (25 mm) that was adherent to the annulus of the PV and obstructed the orifice with 39 mm Hg transvalvular gradient. The thrombolytic therapy protocol consisted of recombinant tissue-type plasminogen activator (rt-PA) (100 mg in 90 minutes) followed by heparinization. Control echocardiography no longer showed significant obstruction and the transvalvular gradient decreased from 39 mm Hg to 6 mm Hg.

Two weeks later, a non-obstructive thrombus (14 mm) was detected and another course of thrombolytic therapy was given using rt-PA (100 mg in 90 minutes).

Five weeks later, the fourth episode of re-thrombosis occurred. The prosthetic valve orifice was found to be 1.05 cm². She was immediately treated with intravenous rt-PA (100 mg over 90 minutes) followed by heparinization. After thrombolytic therapy, vaginal bleeding lasting one week occurred due to anticoagulation.

Nine months later, the patient had another PVT with sudden pulmonary edema and heart failure. Prompt diagnosis of obstructive PVT with echocardiography and emergency surgery was undertaken within 2 hours. At the operation, a huge thrombus arising from the entire bileaflet mitral prosthesis on the atrial

surface was seen to occlude the orifice completely. Thrombectomy was performed and mechanical valve was replaced with a # 27 Hancock bioprosthesis.

Follow-up echocardiography no longer showed significant thrombosis either at the mitral bioprosthesis or at the left atrium after the re-replacement. The patient, with aspirin therapy 300 mg/day, has been well at follow-up two years after her discharge.

DISCUSSION

The incidence of mitral PVT is low, ranging from 0.1% to 5.7% per patient-year; however the recurrence rate of mitral PVT is considerably high as 15-30% (1, 2).

The risk of PVT is mainly dependent on the type of either bioprosthesis or mechanical prosthesis. The risk also varies according to valve design and material.

Generally, patients who receive inadequate anticoagulation, particularly with PV in the mitral position, have an increased risk for thrombus or pannus formation. INR values of the patient during thrombotic attacks were 1.38, 1.20, 2.41 (non-obstructive PVT), 2.25, 2.04, respectively. The patient was found to be inadequately anticoagulated particularly at the first and second thrombotic episodes. Subtherapeutic anticoagulation was the key factor for thrombotic complications. The socioeconomic status of the patient led to an ineffective anticoagulation regimen and a low INR level, which was recommended to be kept at a level of 2.5 to 3.5. Education of the patient as well as the primary care physician is required to reduce life threatening thromboembolic complications, particularly in patients who return to their hometown for follow-up care.

Patient-related coagulability may significantly influence postoperative thromboembolic complications and the risk of PVT (3). We determined the clinical predictive factors of PVT recurrence in the patient based on rheologic and immunohistological assessment. The patient was evaluated for hypercoagulable state; titers of anticardiolipin antibodies (ACLA; both IgG and IgM types),

antiphosphatidlyserine antibody (APSA) were found to be high. Lupus antibody (LA) antigens were strongly positive. Lipoprotein (a) [Lp (a)] and fibrinogen levels were found to be within normal limits (Table 1).

Several mechanisms can develop a hypercoagulable state in patients with mechanical PV. Artificial heart valves contribute to the generation of systemic hypercoagulable state (4). There are not many clinical studies related to hypercoagulable states and development of recurrent thrombotic valve obstruction in the literature. Gençbay et al. (5) published a study on frequency of abnormal levels of ACLA-IgG, ACLA-IgM, APSA, LA, Lp (a) and the prevalence of hypercoagulable states in 15 patients with recurrent thrombotic valve obstruction by comparing 15 matched patients with mechanic PV without thrombotic complication. They found that at least one of the factors was abnormal in 93% of the patients and the difference between PVT group and control group in antiphospholipid antibodies (APLA) and the other hypercoagulable state factors was statistically significant. The prevalence of APLA was found to be 31% in the patients with acquired heart valve deformities in another study (6). The combination of hypercoagulable state and ineffective anticoagulation prophylaxis increased tendency of recurrent PVT in our

case. Although surgical intervention has long been the standard therapy for patients with PVT, our case experience suggests that thrombolytic therapy may be considered as an effective alternative to surgical intervention by avoiding re-operation-related risks. Our patient was treated with streptokinase and rt-PA (three times). During thrombolytic therapy, there was no systemic embolism. We concluded that the patients with recurrent PVT should be evaluated for a hypercoagulable state. In case of recurrence of thrombosis, re-replacement with a biological substitute should be considered.

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Table 1. The factors associated with hypercoagulable state in the patient.

ACLA-IgG	30	(<15 GPLU / ml)
ACLA-IgM	13	(<12.5 MPLU / ml)
APSA-IgG	24	(<12 RLU / ml)
LA	strongly positive	
Lp (a)	24	(<30 ml / dl)
Fibrinogen	3.1	(2 - 4 gr / L)

ACLA: anticardiolipin antibody,
 APSA: antiphosphatidlyserine antibody,
 LA: lupus antibody,
 Lp (a): lipoprotein (a).