Abstract

Prosthetic valve thrombosis is defined as any obstruction of prosthesis by non-infective thrombotic material. It has an estimated incidence of 0.03%-4.3% per year and is reported to occur in 0.5%-8% of the left-sided prosthetic valves and in up to 20% of tricuspid prosthesis. We describe a successful treatment of a young man presented with a thrombosed prosthetic aortic valve using a slow intravenous infusion of Tenecteplase without any bolus dose in a newly described dose regimen for aortic valve position and with a complete resolution of the thrombus and regaining the full mobility of both valve leaflets within 48 hours.

Introduction

Prosthetic valve thrombosis (PVT) is considered a serious complication following mechanical valve replacement. It may result in disabling peripheral thromboembolism and life-threatening deterioration in a patient’s hemodynamic status unless dealt with appropriately and promptly. Treatment of PVT includes administration of thrombolytic agents or surgery. Various thrombolytic treatments include streptokinase, urokinase and recombinant tissue plasminogen activators have been reported with variable success rate. However, the data on the use of Tenecteplase (a synthetic tissue plasminogen activator) is limited. We report here a case of 31 years old male patient with aortic PVT that successfully treated with slow intravenous tenecteplase infusion restoring complete valve function.

Case report

A 31 year old male patient underwent aortic valve replacement with St. Jude 23 mm mechanical prosthesis valve for severe aortic valve
regurgitation and was on oral anticoagulant (warfarin), maintaining a therapeutic International normalized ratio (INR) during follow up. He presented 3 years later with progressively worsening dyspnea of New York Heart Association (NYHA) class II of one month duration. He admitted to not taking his oral anticoagulation for two months prior to presentation. On examination, vital signs were stable with an absent ejection click and a grade 4/6 systolic murmur. Transthoracic echocardiogram revealed decreased mobility of one leaflet with a peak and mean aortic valve gradients of 140 and 90 mm Hg, respectively (Figure 1). Left ventricular function was normal. Fluroscopy showed severely restricted mobility of the leaflet. His INR was normal.

After discussing various options, we elected to treat him with slow intravenous infusion of Tenecteplase (Metalyse, Boehringer Ingelheim) 35 mg diluted in 50 ml normal saline over 24 hours and to be repeated according to the patient’s clinical and echocardiographic response. No bolus doses were given. Following thrombolysis, unfractionated heparin infusion and warfarin were commenced until INR is therapeutic. Small dose Aspirin was added to patient therapy. After thrombolysis, the patient subjectively felt relieved within 6 hours and the prosthetic valve click became sharp, and the systolic murmer decreased to grade 1/6. We continued thrombolysis for further 24 hours. After 48 hours his vitals were stable, the click was well audible and his murmer disappeared. Two dimensional echo showed full mobility of aortic valve leaflets, and aortic valve peak and mean gradients were decreased to 22 mmHg and 12 mmHg, respectively (Figure 2). Fluroscopy showed completely mobile prosthetic aortic leaflets. The patient was discharged home with no complication 5 days later.

**Discussion**

PVT is defined as any obstruction of prosthesis by non-infective thrombotic material. It has an estimated incidence of 0.03%-4.3% per year [1] and is reported to occur in 0.5%-8% of the left-sided prosthetic valves and in up to 20% of tricuspid prostheses. The most common cause of PVT is inadequate anticoagulation therapy. [1] Optimal
The management of PVT remains controversial. As per ACC/AHA guidelines 2014, for left-sided PVT, emergency surgery is indicated in patients with NYHA functional class III-IV (class IIa) and fibrinolytic therapy for patients in functional class I-II (class IIb). [2] Lengyel et al considered thrombolysis as the first line of treatment for obstructive PVT, independent of NYHA class and thrombus size, if there are no contraindications. [3] On the other hand, in a recent series of 210 patients reported by Roudaut, surgical treatment was associated with significantly better long-term results in terms of recurrence and mortality and a lower incidence of embolic complications, which reached 15% in the fibrinolysis group (vs. 0.7% in the surgery group). [4]

The fibrinolytic agents used for treatment of PVT are streptokinase, urokinase and recombinant tissue plasminogen activator (alteplase). The newer fibrinolytic agent Tenecteplase is a synthetically engineered variant of alteplase designed to have increased fibrin specificity, greater efficacy, increased resistance to plasminogen activator inhibitor-1(PAL-1) and a longer half life. It has been used extensively in acute myocardial infarction but there are anecdotal reports of its use in treatment of mitral and aortic PVT. Charokopos et al were the first to publish report of Tenecteplase for aortic PVT. [5]

Our patient was symptomatic for 1 month. As Tenecteplase is more fibrin specific, easy to administer and we had previously used it successfully in Mitral valve thrombosis, [6] we felt it was a good choice. In case thrombolysis had failed, re-exploration and redo aortic valve replacement was planned. To our knowledge this is a newly described dose regimen of Tenecteplase in the thrombolysis of a thrombosed prosthetic aortic valve to be given without any intravenous bolus.

PVT can be successfully treated with Tenecteplase. More experience of its use and the rate of its administration might establish its role as the thrombolytic of choice in management of PVT.

Conflict of interest
None.

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References