Case report

Case Series on Prosthetic Valve Thromboses Treated with Intravenous Thrombolysis at a Tertiary Teaching Hospital in Nairobi – Kenya

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How to cite this article: Odhiambo JO, Abdallah SS, Ngunga LM. Case series on prosthetic valve thromboses treated with intravenous thrombolysis at a tertiary teaching hospital in Nairobi – Kenya. *JOKAP*. 2025; **7**(1): 29 – 32. DOI: https://dx.doi.org/10.4314/jkap.v7i1.5

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Abstract

Valvular heart disease is a significant global health issue, particularly in developing countries where Rheumatic Heart Disease (RHD) is the leading cause. Valve replacement surgeries have increased, with mechanical prostheses favored for their durability. However, thromboembolism remains a critical complication post-surgery, with prosthetic valve thrombosis rates of 0.03% for bioprosthetic valves and between 0.5% to 8% for mechanical valves in the aortic and mitral position.

This report details two cases of heart failure following valve replacement due to rheumatic heart disease. The first case involves a 42-year-old female who developed prosthetic valve thrombosis after discontinuing warfarin for menorrhagia post-mechanical mitral valve replacement. The second case features a 37-year-old male who experienced valve thrombosis after undergoing mechanical aortic valve replacement and subsequently became dehydrated from acute gastroenteritis while on warfarin. Both patients were managed with anticoagulation and fibrinolysis, highlighting the importance of careful monitoring and management in post-operative care to prevent thromboembolic complications.

Key words: Valvular heart disease, Intravenous thrombolysis

Introduction

Valvular heart disease is a prevalent condition that significantly contributes to the cardiovascular disease burden worldwide, especially in developing countries (1). Rheumatic Heart Disease (RHD) is the most common aetiology in developing countries, while degenerative valve disease is more prevalent in developed countries such as the United States (2). RHD tends to occur among younger individuals aged 5-15 years, while degenerative valve diseases affect the older population (3).

In the past several decades, many patients have undergone valve replacement, with mechanical prostheses being preferred over biological valves due to their longevity. However, complications following valve replacements can either be valve-related or non-valve-related, with thromboembolism, bleeding, and prosthetic valve endocarditis being common (4). The incidence of prosthetic valve thrombosis is reported to be 0.03% in bioprosthetic valves and 0.5% to 8% in mechanical valves in the aortic and mitral positions, respectively (5).

Prosthetic valve thrombosis results from various factors, such as the thrombogenicity of the prosthesis, abnormal trans-prosthetic blood flow, atrial fibrillation, and sub-optimal anticoagulation (6). The clinical presentation of prosthetic valve thromboses varies among patients and can manifest as heart failure or peripheral embolism features (6). The commonest precipitants for prosthetic thrombosis are inadequate anticoagulation and poor patient compliance (7).

This report presents two cases of patients who presented with heart failure post-valve replacement for rheumatic heart disease. The patients were managed with anticoagulation and fibrinolysis.

Case reports

Case 1

A 42-year-old female who had a mitral valve replacement for rheumatic heart disease in 2016

and was taking torsemide 10mg once daily, bisoprolol 2.5mg OD, and warfarin 4mg twice daily, was admitted to the hospital due to progressively worsening dyspnoea over a period of one week. She experienced exertional dyspnea - NYHA III, cough with brown sputum, and moderate post-tussive chest pain. She reported occasional heavy menses and evaluated at the gynaecology clinic with a pelvic ultrasound that turned out normal. She denied hemoptysis, hematemesis or melena stool. There were no constitutional symptoms. However, the dose of the warfarin was reduced in view of the menorrhagia.

On examination, she appeared sick and in mild respiratory distress, hypoxic requiring oxygen supplementation at 2L/min via nasal prongs. There was no pallor or lower limb edema. Her jugular venous pressure was normal, and her precordium was normoactive with S1 + S2 plus a grade III systolic murmur heard best over the apical area, accentuated on expiration and radiating to the axilla. Bilateral lower lung zone inspiratory crepitations were present.

Heart failure secondary to mitral prosthetic valve failure was suspected, which was confirmed by 2D-Echocardiogram that showed markedly increased mitral valve gradients of 36/23mmHf, compared to 16/4mmHg three months prior. 12-lead electrocardiogram, showed widespread T wave inversion with no acute ST segment changes, and chest X-ray, revealed pulmonary edema.

Laboratory evaluation showed mild neutrophilic leukocytosis, microcytic hypochromic red blood cells with normal haemoglobin, an INR of 1.29, prothrombin time of 16.1, normal renal function, an elevated CRP at 188mg/L, and an NT pro-BNP at 735pg/ml.

Initially, the patient was started on treatment doses of enoxaparin in addition to the warfarin and antibiotics for suspected pneumonia. She also underwent brochoscopy for suspected pulmonary infection. This was normal and the blood cultures were negative. A repeat transthoracic echocardiogram was performed that showed suspicion of prosthetic valve thrombosis, thereafter a transesophegal echocardiogam was performed and it confirmed a thrombus in the MV and was thrombolysed with alteplase 100mg infusion over 2 hours on day 5 of admission.

Post-thrombolysis, a transesophageal echocardiogram was performed, which showed a normal functioning mechanical mitral valve with normalized gradients of 8/4mmHg and normal LV systolic function.

Case 2

A 37-year-old male diagnosed with rheumatic heart disease in 2000, underwent aortic valve replacement with a mechanical prosthesis in 2004 and has been on warfarin since then, on follow-up at another center. He had never required any admissions until he presented to the hospital with progressive dyspnea (NYHA class III), chest pains, fevers, and chills for one day. He reported that he could not hear the valve click after the symptoms began. A few days before the presentation, he had sufferred a diarrhoeal illness that was managed as food poisoning. Notably, he had been on warfarin 7.5mg alternating with 5mg once daily, and his INR was at 2.1 during one month before the onset of the symptoms.

Upon evaluation, he was febrile with a temperature of 39 degrees centigrade and had bilateral lower limb edema. The blood pressure was 80/54mmHg and he was saturating at 90% room air, requiring 1L/Min of oxygen supplementation via nasal prongs.

The cardiovascular examination was remarkable for cold extremities, palpable peripheral pulses, the precordium was normoactive with the apex beat at the sixth intercostal space lateral to the midclavicular line. There were, no heaves, and no palpable heart sounds. There was, grade IV systolic ejection murmur over the right upper sternal border with radiation to the carotids, and grade II diastolic murmur best heard at the left lower sternal border with the patient leaning forward.

Laboratory evaluation showed normal white cell counts, INR of 1.54, raised APTT at 35.7, normal acid-base balance from an arterial blood gas analysis done. The bilirubin level was 28 micromol/L with mild indirect hyperbilirubinemia. The troponin I was elevated at 409ng/L, elevated CRP at 33mg/dl, and PCT at 0.18ng/ml.

An impression of prosthetic valve dysfunction was made with a possibility of valve thrombosis to rule out prosthetic valve infective endocarditis given the presence of constitutional symptoms. Blood cultures were obtained, but no antibiotics were started.

An Echo report from a peripheral facility showed severe aortic regurgitation and high gradient across the AV mechanical prosthesis with a mean gradient of 59mmHg, a maximum gradient of 87mmHg,

(peak velocity of 4.5m/s and PHT - 141ms). The INR was 1.6. Warfarin was stopped, and the patient was started on enoxaparin treatment doses.

He underwent a transesophageal echocardiogram, which showed severe prosthetic valve regurgitation and an immobile prosthetic aortic valve. A mass concerning for thrombus was seen on one of the leaflets. LV systolic function was preserved with EF 55-60%. The patient was thrombolysed with alteplase 100mg infusion over 2 hours.

Post-thrombolysis, the BP improved to 118/75mmHg, patient came off oxygen, and he felt better. By the end of the lysis he reported hearing the valve click. A repeat echocardiogram showed a reduction in the mean gradient to 28mmHg, a maximum gradient of 49mmHg (peak velocity of 2.9m/s, and only mild AR). The prosthetic valve leaflets showed improved mobility, and the mass was no longer visualized.

Discussion

These two cases illustrate use of thrombolytic agents – alteplase infusion 100mg over 2 hours to treat acute valve thrombosis. The dose of alteplase used to treat valve thrombosis remains unclear but doses of 50mg to 100mg over 2 hours have been used (8,9). In other case reports, changes in the mean gradient across the prosthetic valve, changes in the motion of valve leaflets, and improvement in clinical symptoms of the patients, have been used as measures of success in fibrinolytic therapy for prosthetic valve thrombosis (10,11). However, there are no meta-analysis or randomized controlled trials to objectively delineate these measures. Other agents such as streptokinase, urokinase and tenecteplase have been used in other cases (12-14).

The predisposing factor in the first case was the alteration of the dose of warfarin while in the second case, there was reduced warfarin bioavailability of the warfarin in the background of an already borderline INR.

Thrombolysis, while an effective treatment for dissolving blood clots, is not without its risks. Bleeding is the most significant adverse effect associated with thrombolysis. Major bleeding events occur in approximately 5% to 7% of patients undergoing this treatment (15). Among these bleeding complications, intracranial haemorrhage is particularly concerning, occurring in about 0.5% to 1% of patients. In addition to bleeding, other potential adverse effects include allergic reactions, reperfusion arrhythmias, and hypotension. Fortunately, in the case of these two patients, no thrombolysis-related adverse effects were observed.

These cases underscore the importance of high clinical suspicion and early performance of an echocardiogram. Thrombolysis is a highly effective treatment for resolving valve thrombosis and restoring proper valve function.

Conclusion

Suspected valve thrombosis requires prompt treatment with thrombolysis which will be lifesaving. Alteplase was used successfully without any significant adverse events.

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