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CASE REPORT

Prosthetic mitral valve thrombosis in pregnancy: From thrombolysis to anticoagulation

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KEYWORDS
Valve thrombosis;
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Abstract
Introduction: Pregnant women with mechanical prosthetic heart valves are at increased risk for valve thrombosis. Management decisions for this life-threatening complication are complex. Open-heart surgery has a very high risk of maternal mortality and fetal loss. Bleeding and embolic risks associated with thrombolytic agents, the limited efficacy of thrombolysis in certain subgroups, and a lack of experience in the setting of pregnancy raise important concerns.
Case report: We report a case of mitral prosthetic valve thrombosis in early pregnancy, which was successfully treated with streptokinase. Ten years later, the same patient had an uneventful pregnancy, throughout which acenocoumarol was maintained.
Conclusion: With this case we review the prevention (with oral anticoagulant therapy) and treatment of prosthetic valve thrombosis during pregnancy, which is important for both obstetrician and cardiologist.

PALAVRAS-CHAVE
Trombose de prótese;
Trombólise;
Anticoagulação;
Gravidez

Trombose de prótese mitral na gravidez: da trombólise até à anticoagulação

Resumo
Introdução: Uma doente grávida com uma prótese mitral mecânica tem risco aumentado de trombose de prótese. Esta complicação potencialmente fatal obriga a decisões terapêuticas complexas. A cirurgia cardíaca tem um risco muito elevado de mortalidade materna e fetal.

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Os riscos hemorrágico e embólico associados aos agentes trombolíticos, a eficácia limitada da trombólise em alguns subgrupos de doentes e a falta de experiência existente no contexto de gravidez são uma forte preocupação.
Caso clínico: Os autores descrevem um caso de uma doente com trombose de prótese mitral no primeiro trimestre de gravidez, tratada com sucesso com estreptoquinase. Dez anos mais tarde, a mesma doente tem uma gravidez não complicada sob tratamento com acenocumarol.
Conclusão: Este caso permite uma revisão da prevenção (anticoagulação) e do tratamento de trombose de prótese durante a gravidez.

Introduction

Pregnant women with mechanical prosthetic heart valves are at increased risk for prosthetic valve thrombosis due to both higher levels of coagulation factors and reduced endogenous fibrinolytic capacity. Management decisions for this life-threatening complication are complex. Open-heart surgery has a very high risk of maternal mortality and fetal loss, especially in early pregnancy. Furthermore, bleeding and embolic risks associated with thrombolytic agents, the limited efficacy of thrombolysis in certain subgroups, and a lack of experience in the setting of pregnancy raise important concerns.

Case report

A 25-year-old woman with a mechanical mitral valve prosthesis (St. Jude Medical) and a 14-week pregnancy was admitted to our cardiac intensive care unit due to orthopnea and exertional dyspnea (NYHA functional class III) of 36 hours' duration.

Her past cardiac history began at the age of 10, when she was diagnosed with rheumatic mitral valve disease while being evaluated for fever of unknown origin and signs of heart failure. Following the diagnosis, rheumatic fever prophylaxis was instituted with 1.2 million units of benzathine penicillin intramuscularly every four weeks. Two years later, she was referred to our institution and hospitalized due to decompensated heart failure. Investigation of her heart failure confirmed rheumatic mitral and tricuspid valve disease with severe mitral regurgitation due to anterior leaflet prolapse, severe tricuspid regurgitation, and pulmonary hypertension. She underwent surgical repair of both mitral (Carpentier-Edwards ring) and tricuspid (de Vega technique) valves. Three weeks later, the patient was reoperated due to failure of the mitral valve repair procedure and persistent heart failure. A biological prosthesis was placed in the mitral position and a redo of the tricuspid repair was performed with a ring. Methycillin-resistant *Staphylococcus epidermidis* was isolated through bacteriological examination of the excised mitral valve and ring. The second surgery was complicated by mediastinitis and acute renal failure, and prolonged overall hospitalization (she was discharged nearly three months after the initial surgery). After a symptom-free period of three years, at the age of 15 she developed rapidly progressive clinical manifestations of heart failure and was found to have thickening and calcification of the mitral prosthesis, causing severe stenosis. The biological mitral prosthesis was replaced with a mechanical prosthesis (25-mm St. Jude Medical) and the postoperative course was uneventful. Subsequently, the patient had normal functional capacity, without clinically relevant cardiovascular events, and was simply managed with adjusted-dose warfarin therapy.

The patient's past obstetric history included two unsuccessful pregnancies. The first, at the age of 19, terminated with elective abortion at week 10 due to fetal malformation; the second, at age 22, was complicated with unexplained in-utero embryo death at week 7.

For the current pregnancy, low molecular weight heparin (LMWH) was substituted for warfarin therapy at week 6, and continued up to the time of presentation. The regimen consisted of nadroparin calcium 5700 anti-Xa IU (in our institution, nadroparin was the most commonly used LMWH for bridging anticoagulation) administered subcutaneously every 12 hours. Compliance with LMWH therapy was 100% throughout this period.

The physical exam on presentation showed hyperpnea, intolerance to prolonged (>10 min) supine position, jugular venous distension, regular tachycardia (125 bpm), blood pressure 100/60 mmHg, dull prosthetic closing click, grade 2/6 mid-diastolic apical murmur, and inspiratory crackles heard bilaterally over the lower lung fields. The ECG documented sinus tachycardia and diffuse ST-T abnormalities.

Transthoracic echocardiography (TTE) showed inadequate mobility of one of the prosthetic leaflets. The M-mode recording at the level of the prosthetic leaflets in parasternal long-axis view showed rounded edges. Peak and mean transprosthetic gradients were 48 and 35 mmHg, respectively, and mitral valve area (pressure half-time method) was 0.8 cm^2 (Figure 1). Other echocardiographic findings included mild mitral regurgitation, mild tricuspid regurgitation, and severe pulmonary hypertension (93 mmHg). An attempt to perform a full transesophagoeal echocardiographic evaluation was unsuccessful due to patient discomfort and breathlessness. Nevertheless,