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CASE REPORT

A rare case of two mechanisms of prosthetic valve dysfunction in the same patient☆

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Abstract Prosthetic valve dysfunction is a significant clinical event. Determining its etiological mechanism and severity can be difficult. The authors present the case of a 50-year-old man, with two mechanical valve prostheses in aortic and mitral positions, hospitalized for decompensated heart failure. He had a long history of rheumatic multivalvular disease and had undergone three heart surgeries.

On admission, investigation led to a diagnosis of severe dysfunction of both mechanical prostheses with different etiologies and mechanisms: pannus formation in the prosthetic aortic valve and intermittent dysfunction of the mitral prosthesis due to interference of a ruptured chorda tendinea in closure of the disks. The patient was reoperated, leading to significant improvement in functional class.

PALAVRAS-CHAVE
Disfunção de prótese valvular mecânica;
Pannus;
Disfunção protésica intermitente

Um caso raro de dois mecanismos de disfunção protésica no mesmo doente

Resumo Em doentes portadores de próteses valvulares mecânicas, a ocorrência de disfunção protésica constitui um evento clínico relevante. Ocasionalmente, a demonstração do seu mecanismo etiológico e gravidade podem revelar-se difíceis.

Apresenta-se o caso clínico de um doente de 50 anos, portador de duas próteses valvulares mecânicas nas posições aórtica e mitral, internado por descompensação de insuficiência cardíaca. Nos antecedentes apresentava uma história longa de doença plurivalvular reumática com três cirurgias cardíacas prévias.

No internamento a investigação conduziu ao diagnóstico de disfunção grave de ambas as próteses mecânicas, sendo a etiologia e o mecanismo diferente em cada caso: recidiva de

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pannus no caso da prótese aórtica e bloqueio intermitente do encerramento dos discos por interposição de uma corda rota do aparelho subvalvular, no caso da prótese mitral. O doente foi reoperado, tendo havido melhoria significativa da classe funcional.

Introduction

Progressive refinements in the design and hemodynamic profile of valve prostheses have led to reductions in prosthetic dysfunction.[1] However, all valve prostheses continue to be associated with complications[2] and dysfunction of varying severity, and this should be borne in mind when assessing the risk/benefit ratio in individual patients referred for valve replacement.[3]

Obstructive mechanical prosthetic valve dysfunction can be due to valve thrombosis, pannus formation or a combination of the two.[4]

Thrombosis is usually associated with subtherapeutic levels of anticoagulation,[5] develops rapidly and is potentially fatal.[6] However, cases have been reported of a more insidious course, and differential diagnosis with pannus formation is of the utmost importance since the only therapeutic option in cases of pannus causing significant hemodynamic compromise is surgery (prosthesis replacement or pannus resection in selected cases),[7] while in cases of prosthesis thrombosis thrombolytic therapy can be considered.[8,9]

Pannus results from the intraprosthetic development of fibrovascular and/or granulation tissue, which can cause significant obstruction.[2,10] Diagnosis is hindered not only by its habitually slow and insidious formation, but particularly by the fact that routine diagnostic exams such as echocardiography can document high transprosthetic gradients but do not provide adequate visualization of pannus ingrowth.[11]

Prosthetic valves are also liable to pathological regurgitation; in mechanical prostheses, this is usually the result of endocarditis or of technical problems during implantation.

The case presented here describes a patient with recurrent mechanical aortic prosthetic valve obstruction due to pannus formation, together with intermittent dysfunction of the mitral mechanical prosthesis due to interference of a ruptured chorda tendinea of the residual native subvalvular apparatus in closure of the disks.

Case report

A 50-year-old man, with a history of rheumatic fever at age 22 and three previous heart valve surgeries, was admitted to a cardiology ward in March 2010 for decompensated heart failure (HF).

In 1991, following a first episode of atrial fibrillation (AF), he was diagnosed with rheumatic valvular disease, with regurgitation in both the aortic and mitral valves, together with moderate left ventricular (LV) dilatation and dysfunction. At that time, he underwent his first cardiac surgery, the aortic valve being replaced by a 23-mm Duromedics mechanical prosthesis and the mitral valve being repaired with a Carpentier-Edwards ring. Echocardiography on the seventh postoperative day showed a peak gradient through the aortic prosthesis of 23 mmHg and no apparent regurgitation. The mitral valve presented only minor regurgitation and normal area by planimetry. LV dilatation (LV end-diastolic diameter [LVEDD] of 69 mm) and moderate LV systolic dysfunction persisted. After two years during which he remained asymptomatic with generally therapeutic levels of anticoagulation, he began suffering HF symptoms, which rapidly worsened to NYHA functional class III. Echocardiography revealed significantly increased transprosthetic aortic gradients (peak and mean of 90 mmHg and 57 mmHg, respectively). Function of the repaired mitral valve remained good, with minor regurgitation. LV dilatation persisted (LVEDD and LV end-systolic diameter of 67 mm and 53 mm, respectively), with moderate systolic dysfunction. Fluoroscopic assessment confirmed prosthetic dysfunction, showing incomplete closure of the prosthetic discs.

The patient was reoperated to replace the aortic valve, the diagnosis of obstruction due to pannus formation being confirmed by direct intraoperative inspection. Following removal of the prosthetic valve and resection of the pannus, a 23-mm St. Jude aortic prosthesis was implanted. Echocardiography on the fifth postoperative day showed a peak gradient through the aortic valve of 29 mmHg.

This second surgery resulted in significant functional recovery, and the patient remained asymptomatic for 10 years. HF symptoms reappeared in 2003 and progressively worsened up to 2007. At that time, echocardiography revealed worsening mitral valve disease (stenosis and valve orifice area of 1.1 cm^2 by planimetry, with severe eccentric regurgitation due to marked hypomobility of the posterior leaflet) and severe tricuspid regurgitation, with estimated pulmonary artery systolic pressure (PASP) of 45 mmHg. The aortic prosthesis presented peak and mean gradients of 41 mmHg and 27 mmHg, respectively. Worsening LV dysfunction was also observed, with ejection fraction of 35%.

A third cardiac surgery was performed in May 2007, which confirmed the echocardiographic findings regarding the mitral valve. Intraoperative inspection of the aortic prosthesis revealed no significant macroscopic changes. The mitral valve anterior leaflet was removed and the posterior leaflet rolled up, together with the chordae tendineae, and sutured to the posterior region of the annulus, thus preserving the mitral subvalvular apparatus. A St. Jude mechanical prosthesis was implanted in mitral position and tricuspid annuloplasty was performed using a 34-mm Carpentier-Edwards ring.