THROMBOSIS IN A MITRAL BIOLOGICAL PROSTHESIS ASSOCIATED WITH SEVERE VALVE DYSFUNCTION DURING ORAL ANTICOAGULATION. CASE REPORT.

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ABSTRACT

The occurrence of a thrombus in a biological prosthesis is rare, but it presents a potentially fatal outcome when there is significant obstruction of the valve area. We describe the case of a 60-year-old patient who presented severe cardiac decompensation 10 months after implantation of biological mitral and aortic prostheses. The diagnostic transesophageal echocardiogram (TEE) showed a pedicled thrombus adhered to the left ventricular (LV) papillary muscle and important thickening of the mitral biological prosthesis (MBP) leaflets by a laminated thrombus with significant limitation of its opening causing severe mitral stenosis. Anticoagulant therapy started with low molecular weight heparin (LMWH) with resolution of the condition. Patient maintains regular monitoring with satisfactory evolution in the last four years.

KEYWORDS: THROMBOSIS / COMPLICATIONS; HEART VALVE PROSTHESIS; MITRAL BIOPROSTHESIS

INTRODUCTION

The incidence of thrombosis in mechanical valve prostheses varies around 0.4/100 patients/year, with the occurrence in the mitral position about 5 times greater than in the aortic position[1]. On the other hand, the formation of a thrombus in a biological prosthesis, despite having frequent potentially fatal outcomes, seems to be underdiagnosed or inadequately diagnosed as valve degeneration. The incidence ranges from 0.1/100 valves/year to 6% in the mitral position, in some reports[2].

Specific risk factors for the occurrence of thrombosis in biological prostheses are unknown, but states of hypercoagulability may predispose to the formation of thrombi[3] associated with conditions such as enlarged left atrium, atrial fibrillation, ventricular dysfunction and a previous history of thromboembolic events[4]. Considering these factors, the Brazilian Valvulopathies Directive recommends anticoagulation with warfarin in the first six months after placing a biological prosthesis in the mitral position even for patients in sinus rhythm (IIb NE B) [5].

The purpose of this report is to describe a case where there was a thrombus in a mitral biological prosthesis, with major obstruction of the valve area, associated with cardiac decompensation, even when using oral anticoagulation.

CASE REPORT

G.M.S., a 60-year-old female was evaluated in a Cardiology outpatient clinic complaining of dyspnea on minimal exertion, orthopnea and paroxysmal nocturnal dyspnea for about six days. She had been admitted to another service with a slight improvement in her condition, being discharged with instructions for TEE and early return to an outpatient clinic.

Patient presented a history of percutaneous mitral valvuloplasty 13 years ago due to rheumatic mitral valve disease and positive serology for Chagas disease with intestinal involvement already demonstrated. 13 months ago, after severe pain in the left lower limb, acute arterial occlusion was diagnosed and the surgical approach was

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performed to remove the thrombus. Two days after discharge, the patient returned to the hospital with severe chest pain, dyspnea on minimal exertion, sweating and tachycardia, consequently being diagnosed with atrial fibrillation (AF) with high ventricular response and an important systolic murmur in the mitral area. She underwent a transthoracic echocardiogram (TTE) that showed a left atrium (LA) of 53mm, an ejection fraction of 37%, severe mitral stenosis and severe aortic insufficiency. Cardiac catheterization did not show significant lesions in coronary arteries. 10 months ago, she underwent cardiac surgery to replace the mitral and aortic valves with biological prostheses, associated with the closure of the left atrial appendage and surgical isolation of pulmonary veins. Despite the initial success in the occurrence of sinus rhythm, she resorted to atrial fibrillation on the second postoperative day. She was discharged using digoxin, metoprolol, amiodarone, spironolactone, warfarin, acetylsalicylic acid and pantoprazole. She maintained outpatient follow-up with difficulty in controlling the prothrombin time international normalized ratio (PT/INR). Echocardiogram performed six months ago, four months after the surgical intervention, revealed a 51mm left atrium, 65% ejection fraction with normal functioning of mitral and aortic prostheses.

Upon admission, 10 months after valve replacement surgery, she presented decompensated heart failure with complaints of palpitations and precordial pain associated with dyspnea on minimal efforts. Electrocardiogram (ECG) showed AF rhythm with HR: 104bpm. TEE showed EF: 56%, LA: 48mm with mass adhered to LV papillary muscle, pedicled thrombus measuring 16X9mm (Figures 1A and B), mitral valve gradient 29 (peak) and 18 (medium), PBM with important limitation of the opening of the leaflets, with laminated thrombus attached (figure 2), LA with spontaneous contrast (+++/+4) (Figure 3) and sessile thrombus occupying 2/3 of its area (figure 4), normal functioning aortic biological prosthesis. The patient was admitted and submitted to full anticoagulation with enoxaparin.

Considering the previous embolic events and the current condition, even when using warfarin, the hematology team requested an evaluation, which attributed competition factors to coumarins, such as the use of furosemide and self-medication with laxatives, with justification for therapeutic failure in the control of PT / INR.

The patient was discharged nine days after admission with clinical improvement and strict guidance on the control of warfarin use. Patient maintains outpatient follow-up with regular and strict control of PT/INR, without the need for hospitalization. TTE performed months after hospital discharge showed aorta: 33mm LA: 39mm EF: 44% absence of image suggesting thrombus in LA, mitral valve gradient 11mmHg (medium) and 26mmHg (peak) valve area: 1.8mm.

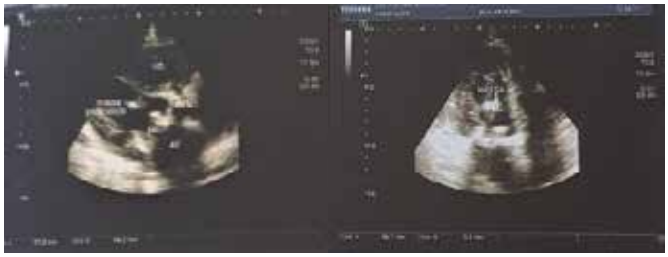

Figure 1 A and B: mass attached to the end papillary muscles compatible with pedicled thrombus measuring 16X9mm

Figure 2: Mitral biological prosthesis with significant limitation of the opening of the leaflets (laminated thrombus).

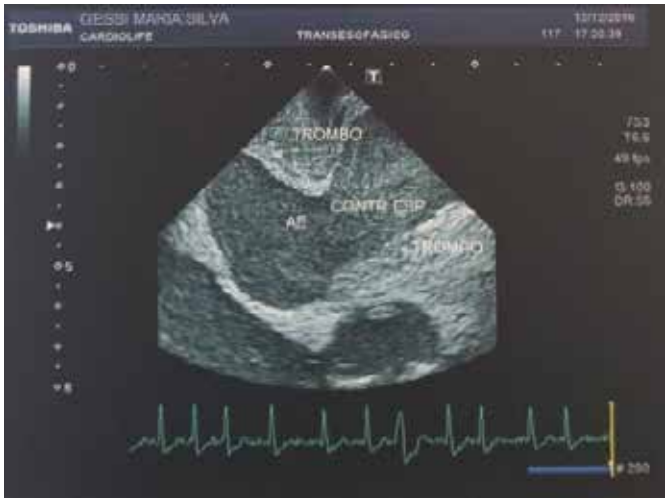

Figure 3: LA presents 48mm with the presence of spontaneous contrast and thrombus.

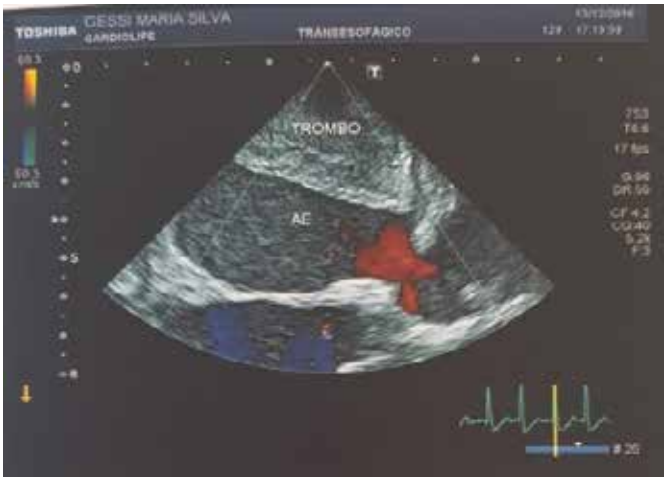

Figure 4: Thrombus with a sessile aspect, occupying 2/3 of the left atrial cavity.

DISCUSSION

Rheumatic heart disease, despite the significant reduction in incidence and mortality in Brazil, remains highly prevalent (1 to 7 cases/1000) when compared to the occurrence in developed countries[6]. The repercussions of changes in mitral valve disease in the myocardial structure contribute to the development of atrial cardiomyopathy with hypocontractility and impaired atrial endothelial function that could potentially contribute to the formation of clots, regardless of the detection of atrial fibrillation[7].

The occurrence of thrombus in our patient was in the tenth month after the placement of the mitral biological prosthesis, about two months before that described in a report by Pislaru, from the Mayo Clinic[3] that suggests thrombi between 13 to 24 months after implantation, as well as a description of more frequent occurrence in patients with subtherapeutic anticoagulation.

Treatment with the use of LMWH is suggested as the first choice in the treatment of thrombosis in biological valve prosthesis, associated or not with initial oral anticoagulation[8]. It was the therapeutic option at our service, despite the size of the thrombus and the important impairment of valve function due to the low risk compared to fibrinolysis or reoperation[4] and the patient's good evolution, in association with the therapies instituted to control cardiac decompensation.

The benefit of warfarin in the prevention of embolic events is already well defined, however, the reach of the therapeutic dose verified through the PT/INR even in selected populations and with strict monitoring is 66.4% [9]. Many factors can contribute to suboptimal control of INR including inadequate adherence to warfarin therapy, drug interactions, inadequate or erratic intake of foods containing vitamin K and genetic differences between patients[10]. Warfarin was maintained in the patient (CHA2DS2:3) during hospitalization and after hospital discharge and the better adequacy of INR in subsequent evaluations was attributed to reorientation regarding the importance of proper medication use, self-medication and strict outpatient monitoring.

CONCLUSION

The evaluation of the patient with cardiac decompensation and a history of valve replacement, even with the placement of a biological prosthesis, should take into account the hypothesis of thrombosis in the prosthesis. TEE is the method of choice for clarifying the etiology of unfavorable developments. The use of LMWH even in the presence of large and pedicled thrombi that result in significant limitation of valve mobility is a safe therapeutic option in patients without hemodynamic instability. Strict control and perseverance in guiding the patient on the proper use of warfarin can change outcomes, even with the limitations we face in our health system.

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