Case Report

Thrombosis after mitral valvuloplasty: A review of two cases and the role of antithrombotic therapy

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Summary
We describe two cases of thrombosis after mitral valvuloplasty (MV). Antithrombotic therapy after MV in patients with no thromboembolic risk factors is essentially based on treatment with a platelet aggregation inhibitor. This strategy may not be sufficient in some cases and the introduction of oral anticoagulant therapy may be necessary.

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Introduction

Mitral valvuloplasty (MV) is currently the reference treatment for significant mitral regurgitation. The low thromboembolic complication rate associated with this procedure generally avoids the need for anticoagulant therapy in patients in sinus rhythm. However, the optimal antithrombotic strategy remains controversial, especially during the highest risk period, the first 3 postoperative months.

We report two rare cases of thrombosis that occurred after MV in Clermont Ferrand University Hospital and review the guidelines concerning antithrombotic therapy after this procedure.

Case 1

A 69-year-old man with degenerative mitral valve failure underwent MV in June 2008, consisting of partial quadrangular resection of P2 with semi-circumferential annuloplasty over a 34-mm Sorin Sowering® ring. The postoperative result was good with no residual mitral regurgitation. Left ventricular function was almost normal (left ventricular ejection fraction: 55%).

His postoperative course was uneventful apart from a transient episode of complete atrioventricular block. He had sinus rhythm at discharge. Antithrombotic therapy on discharge consisted of aspirin (100 mg/day).
Two months later he suffered a febrile episode during rehabilitation and transesophageal echocardiography (TOE) revealed a mobile 4-mm mass on the lateral aspect of the mitral ring (Fig. 1a and b). Due to the absence of laboratory signs of inflammation, it was decided to monitor the patient closely, with no change of treatment, and to repeat TOE 1 month later.

Despite the absence of any clinical or laboratory signs of infection, extension of the lesions was observed with several mobile images attached to the ring. The two mitral leaflets were also very thickened and reorganized (Fig. 2a and b).

The patient was admitted with suspected infective endocarditis despite negative blood cultures and low C-reactive protein. Intravenous dual antibiotic therapy (vancomycin and gentamicin) was started and heparin was added 4 days later following the discovery of central venous catheter thrombosis.

TOE repeated several days later revealed total resolution of the mobile images with persistence of minor mitral regurgitation. A diagnosis of thrombosis of the annuloplasty ring was made and vitamin K antagonists (VKA) were prescribed.

Three months later, the patient was asymptomatic and transthoracic echocardiography (TTE) confirmed the good result of MV and the absence of any abnormal valvular images.

Case 2

A 50-year-old woman with degenerative mitral valve disease underwent MV in August 2009, consisting of quadrangular resection of P2 and semi-circumferential annuloplasty on a 34-mm Sorin Sowering® ring. Her medical history included chronic alcoholism and active smoking.

No postoperative complication was observed. TTE on discharge showed normal left ventricular wall motion (biplane ejection fraction: 58%), moderate left atrial dilation (28 cm² on planimetry), no residual mitral regurgitation, and sinus rhythm. Antithrombotic therapy on discharge consisted of aspirin (250 mg/day).

In April 2010, she experienced chest pain followed by cardiopulmonary arrest and was resuscitated after five shocks with a semi-automatic defibrillator. She was urgently transferred to the coronary angiography room due to the presence of ST elevation in the inferior leads. Coronary angiography demonstrated a normal coronary network with embolic occlusion of retroventricular branch of right coronary artery which was treated by thrombo-aspiration and balloon angioplasty. The thrombo-aspirated material consisted of a sterile fibrino-thrombotic thrombus.

TOE showed good functioning of the MV with no residual regurgitation and a small (7 mm) mobile thrombus situated on the atrial aspect of the mitral ring, adjacent to the posterior commissure (Fig. 3a and b).
Few studies specifically devoted to MV have been published and most guidelines for antithrombotic therapy are based on those for aortic bioprostheses. Only the European Society of Cardiology (ESC) has proposed specific guidelines [2]: these consist of 3 months of postoperative VKA in all patients and then discontinuation of antithrombotic therapy in the absence of embolic risk factors (ERF). These guidelines include the widespread use of low-dose aspirin (75–100 mg) postoperatively rather than anticoagulants in patients with a low embolic risk, but do not recommend this strategy due to the absence of randomized trials confirming the safety of platelet aggregation inhibitors.


For the American Heart Association/American College of Cardiology and ESC, 3 months of postoperative VKA is justified as this period is associated with the highest thromboembolic risk [4]. This risk can be explained by progressive endothelialization of the implant (prosthesis or ring) and sutures, inflammation, and the high frequency of supraventricular arrhythmias.

The high thromboembolic risk was confirmed in a recent prospective study of 1344 patients undergoing mitral valve surgery (including 897 mitral valve repairs) [1]. The linearized stroke rate after mitral valve repair within the first 30 days and between 30 and 180 days was 1.5/year (±0.4%) and 2.1/year (±0.6%), respectively. The relative risk of stroke compared to a similar population was 31 and 2.1 at 30 days and 6 months, respectively. Stroke rates after a bioprosthesis and mitral valve repair returns to that of the general population after 180 days [1]. In this study, treatment with VKA was only considered when it was prescribed for more than 3 months postoperatively (41% of patients), but no details were provided concerning antithrombotic therapy received during the first 3 months (heparin followed by VKA was generally used). Finally, the stroke risk was similar to that in the general population 6 months after a bioprosthesis or MV.

Despite these guidelines, there is marked disparity concerning anticoagulant management after MV. A survey conducted in 2005 among British surgeons revealed the use of VKA in 64% of cases [5]. This rate was only 47% after an aortic bioprosthesis. In France, many centers, including Clermont Ferrand, no longer use anticoagulant therapy in these indications in low-risk subjects.

In our first case, the patient did not have any ERF and, in line with usual practice in our hospital, therefore only received a platelet aggregation inhibitor (aspirin). The thrombotic complication occurred relatively early (beginning of the 3rd postoperative month) and was discovered incidentally. The second patient had minor ERF: active smoking and moderate left atrial dilation; and also had poor compliance with treatment. The thromboembolic complication occurred later, during the 8th postoperative month.

Such complications are extremely rare. To our knowledge, no case of MV thrombosis has been reported previously in the literature. A literature review revealed two cases of thrombosis of mitral bioprosthesis, presenting with acute pulmonary edema [6,7]. Both patients had a high embolic risk (severe left ventricular dysfunction and history of stroke, and atrial fibrillation and left atrial dilation.

Figure 3 (a) Transesophageal echocardiography showing a left atrial mobile structure and (b) coronary angiography showing cup-shaped occlusion of retroventricular branch of right coronary artery.

The diagnosis was thrombosis of the mitral annuloplasty ring complicated by embolic inferior myocardial infarction. Her subsequent course was favorable with localized wall motion abnormality at inferior wall. Her thrombophilia work-up was negative and the complication was attributed to poor compliance.

Antithrombotic therapy on discharge consisted of aspirin plus VKA.

Discussion

MV is currently the reference treatment for significant mitral regurgitation with excellent long-term results and a low complication rate, especially thromboembolic complications [1]. However, antithrombotic therapy after MV has not been clearly defined. Although anticoagulant therapy is indicated in patients with a high embolic risk (atrial fibrillation, left ventricular dysfunction, coagulopathy, left atrial dilation), this treatment is questionable in young patients at low risk.
respectively). The first patient received only aspirin (325 mg/day) and the thrombosis occurred in the second patient 1 month after stopping VKA.

Our first case illustrates the difficult differential diagnosis between thrombus and vegetation and the role of anticoagulant therapy when clinical and laboratory findings are not in favor of infective endocarditis. In the second case, bacteriological examination of the thrombus confirmed the absence of infection.

To conclude, MV is the current reference treatment for degenerative mitral regurgitation and is associated with a low complication rate, especially thromboembolic events. However, our two cases illustrate that thrombotic complications can occur after MV. Postoperative antithrombotic therapy remains controversial and a large-scale randomized trial is required to define the optimal strategy.

References


