

Mitral Valve Apparatus Preservation and Early Bioprosthetic Thrombosis. A Word of Caution

Paulo Roberto B. Evora, Solange Basseto, Lafaiete Alves Junior, Alfredo J. Rodrigues

Faculdade de Medicina de Ribeirão Preto, Universidade de São Paulo, São Paulo, SP - Brazil

An advantage of bioprosthetic mitral valve replacement in patients with normal sinus rhythm is avoiding the need for long-term anticoagulation. Thrombosis of prosthetic valves usually affects mechanical prostheses. There have been several case reports of bioprosthetic mitral valve thrombosis that led to early valve explantation. At least in theory, the technique of retention of the mitral valve apparatus during mitral replacement may lead to an increased incidence of this unusual complication¹. However, there have been few reported cases involving the preservation of the mitral valve apparatus and prosthesis “mismatch” as a predisposing factor for bioprosthetic valve thrombosis. In addition, it is mandatory to emphasize that there is no evidence in the literature associating subvalvular apparatus preservation and mitral bioprosthesis thrombosis if the proper technique is carried out, without considering the prosthesis “mismatch” association.

In a random number of surgical procedures for correction of heart valve disease a total of 384 patients were selected, 169 (43%) of which underwent procedures involving the mitral valve (repair surgeries; mechanical and biological prostheses). Among these patients we were not able to identify, retrospectively, cases of bioprosthesis thrombosis. Anticoagulation was not advised during the first months after valve replacement, even in patients without risk factors for thromboembolism. However, we did not observe thromboembolic events in such patients, in spite of the fact that we have not adopted this procedure in the last fifteen years, when our follow-up databank was improved with the aid of informatics. Within the group of patients with mitral valve disease, two patients (1.18%) presented thrombosis of bovine pericardial bioprosthesis. In these two instances, a possible “mismatch” prosthesis-patient and preservation of the entire mitral valve apparatus were identified. Both patients were female, aged 34 and 44 years, who showed no adverse events during surgery and had the following immediate postoperative prosthetic gradients: 12 mmHg

(prosthesis M-25, BSA = 1,672 m²) and 6 mmHg (prosthesis M-27, BSA = 1,632 m²), respectively. Neither of the patients was receiving anticoagulants. The case of the youngest patient was more dramatic, because she was admitted to the emergency room with history suggesting paroxysmal acute atrial fibrillation and neurological signs of embolization 18 days after surgery. The transesophageal echocardiogram (TEE) revealed thrombosis affecting the left auricle, atrial wall, and atrial face of the prosthesis. These data were confirmed at the emergency reoperation, when a new mechanical prosthesis was implanted with resection of the leaflets and annular fixation of the papillary muscles.

The prosthesis-patient “mismatch” may be an independent predictor of mortality after mitral valve replacement. Unlike other independent risk factors, this problem can be avoided or its severity may be reduced by using a prospective strategy at the time of operation. For patients considered at risk of serious “mismatch”, every effort should be made to implant prosthesis with a larger area orifice, in order to preserve the continuity between the mitral annulus and the left ventricular wall. Both patients presented gradients consistent with mild to moderate mitral stenosis already taking place in the immediate postoperative period. Based upon previous experience, this observation was underestimated by erroneously considering that the interference of the preserved elements in the bioprosthetic valve is unlikely.

Concerns about the possibility of mitral chordae interference on mechanical prostheses leaflets have been reported frequently, but not when bioprosthesis was used. However, despite having been reported separately, the preservation of both mitral valve leaflets can potentially contribute to possible thrombotic “pseudostenosis” and/or early bioprosthesis failure^{1,2}. Finally, against the participation of the preservation of isolated mitral subvalvar elements in the predisposition to thrombosis, there are isolated reports of bioprosthetic thrombosis in cases of complete valve excision³.

We reported the idea, as “case report” to a highly interactive magazine and the reason for the rejection was the absence of data, which effectively could support the hypothesis of the association of mitral valve preservation apparatus with smaller valve prosthesis with the early occurrence of thrombosis of mitral bioprosthesis.

One reviewer wrote that we were trying to blame preservation of the subvalvular apparatus as the culprit of early tissue valve thrombosis, also suggesting that mismatch played a role in the two reported cases. In his opinion, we wanted to write a paper criticizing a well-established technique, with a lot of good scientific evidence behind, as in this case,

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Mailing address: Paulo Roberto Barbosa Évora •
Rua Rui Barbosa 367/15 - Centro - 14015-120 - Ribeirão Preto, SP - Brazil
E-mail: prbevora@cardiol.br, prbevora@fmrp.usp.br
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preservation of the subvalvular apparatus in patients in their thirties and forties. This is a partial interpretation of the cases' presentation. Indeed, the main idea was to speculate about a possible "synergism" between subvalvular mitral apparatus and a possible "mismatch", since the mitral prosthesis frequently is undersized in consequence of this surgical approach. The reviewer emphasized that from the scarce data presented it might be the case that the reason for thrombus formation inside the left atrium was due to the hemodynamic status of that particular patient: small mitral valve prosthesis as stated by us, with moderate functional stenosis together with the recent onset of atrial fibrillation in a patient that may suffer a prothrombotic state early after surgery, aggravated by the absence of any anticoagulant therapy, as generally recommended in all clinical guidelines. The strong reviewers' criticism was based on the fact that no antithrombotic therapy was given, against current guidelines suggesting one to three months of anticoagulation. Currently, antiplatelet agents have been found to be as effective as anticoagulants early after bioprosthetic valve replacement and would be a good option, taking in account our "social problem" with anticoagulation.

A second reviewer was absolutely right in considering that the hypothesis of a cause and effect relationship between the patient bioprosthesis' "mismatch" and the severe complication of valve thrombosis was not demonstrated by the presented data. While it is highly interesting, such a hypothesis should be supported by many more details about those patients, and discussed along with all other possibilities of valve thrombosis in the postoperative course. The presentation was based on retrospective thoughts and, unfortunately extremely good, unequivocal data necessary to support this argument were missing, but even when speculative or hypothetical, the "word of caution" is valid.

The preservation of the mitral apparatus, which is routinely the author's preference, implies in the possibility of frequently undersized prosthesis. The decision to preserve the entire valve apparatus, in both cases, was a decision based solely on anatomical conditions⁴. As a self-criticism, this was a wrong decision once in reoperations; resection of the valve leaflets and papillary muscle fixation at the mitral valve annulus were possible in the implantation of oversized mechanical prostheses.

This report does not intend to suggest the abandonment of subvalvar or valvar apparatus surgical techniques in cases of valve replacements. Always taking into account the possibility, perhaps caused by the negative experience with the two reported cases, we keep our feelings that this experience would be shared, even in the case of a speculative hypothesis. But the reviewers were sensitive to the idea and wrote important comments to cardiac surgeons and cardiologists, which motivated the presentation of this point of view.

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