

The conundrum of mitral valve etiology and the association with clinical outcomes

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In the paper by Rzucidło-Resil et al¹ published in this issue of *Kardiologia Polska (Kardiol Pol)*, the authors analyzed the perioperative results of 337 consecutive patients with severe mitral regurgitation (MR), who underwent a surgery in their institution during an unspecified period. The authors found no difference in early clinical outcomes after surgery among different etiologies of mitral valve (MV) disease. The results were influenced only by the type of procedure and perioperative comorbidities. Unfortunately, the small sample size and the low percentage of patients with secondary MR (28%) call for some caution in the interpretation of the results. It is of paramount importance to place all pieces of the game on the table to have a global understanding of this association.

Cardiac surgery most often requires extracorporeal circulation and full heparinization, hypothermia and cardioplegia induce several changes in the homeostasis of the patient that impact perioperative outcomes.² Therefore, the sicker the patient is preoperatively, the more pronounced is the influence of the deleterious effects of surgery. The 2 most used surgical risk scores, the Society of Thoracic Surgeons score and European System for Cardiac Operative Risk Evaluation (EuroSCORE) II, were developed based on the evaluation of thousands of patients and are currently used in most studies evaluating the risk of cardiac surgery.^{3,4} Although the etiology of the MV disease is not included in the structure of these scores, there are several considerations that deserve discussion.

Firstly, patients' demographic characteristics vary among the different etiologies, even between the several categories of primary and secondary MR. Degenerative pathology is the most

frequent etiology of primary MR in Western countries and can assume several presentations, from myxomatous disease, where Barlow disease is the far extreme of the spectrum, to fibroelastic deficiency, which is in the antipodes of Barlow disease.⁵ Patients with fibroelastic deficiency are generally older, more symptomatic, and have more comorbidities, greater degrees of left ventricular (LV) dysfunction, and other valve diseases (tricuspid or aortic), which translates into higher risk scores.⁶

On the other hand, massive mitral annular calcification, which can be included in the degenerative category, is being increasingly observed, because it appears to have a straight correlation with age, and life expectancy is rising worldwide. This condition poses special technical considerations, since it may preclude MV repair, and even MV replacement can be cumbersome. Passing sutures in a heavily calcified mitral annulus is demanding and may be associated with atrioventricular sulcus disruption or relevant periprosthetic leakage after surgery. Hence, mitral annular calcification is usually associated with higher mortality and morbidity.⁷

New cases of acute rheumatic fever have almost disappeared from most high-income countries, where rheumatic heart disease is now a remnant of the past and is usually seen in middle-aged or elderly patients, in whom it does not represent a problem significantly different from that of other etiologies of MV disease. By contrast, rheumatic heart disease remains an important health burden in low- and middle-income countries, where rheumatic MV disease is usually observed in younger patients with completely different risk factors.⁸

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Secondary (previously termed “functional”) MV disease has been under close scrutiny in recent times, with antagonist results coming from the 2 randomized control trials: COAPT (Cardiovascular Outcomes Assessment of the MitraClip Percutaneous Therapy for Heart Failure Patients With Functional Mitral Regurgitation) and MITRA-FR (Multicentre Study of Percutaneous Mitral Valve Repair MitraClip Device in Patients With Severe Secondary Mitral Regurgitation).^{9,10} The term “secondary” or “functional” is used because MV dysfunction does not result from structural abnormalities of the mitral apparatus. Rather, it is a ventricular problem, resulting in apical and lateral displacement of the papillary muscles with consequent leaflet tethering, finally leading to MR. However, even amongst the 2 most frequent forms of secondary MR, ischemic MR and cardiomyopathy, there are substantial differences that may translate into different perioperative and clinical outcomes.

Ischemic MR occurs as a consequence of LV remodeling after myocardial infarction. The presence of viable ischemic myocardium and mild degrees of LV dysfunction portends better outcomes, and MV surgery is indicated only when there is severe MR, since it appears that intervening in moderate MR does not modify the course of the disease.¹¹ Additionally, MV repair does not appear to be better than replacement in this context.¹²

On the other hand, secondary MR related with dilated cardiomyopathy represents a surrogate of dismal prognosis in end-stage heart failure. These patients have severe LV dilation and very poor LV function, and correction of the MR is usually associated with clinical improvement but not with improved survival. Adjunctive measures, such as LV resynchronization and medical therapy optimization, may be as important as surgery in this setting.¹³

Secondly, MV repair is heavily influenced by MV etiology, and in the majority of reports, it carries a lower surgical risk than replacement.¹⁴ The goal of repair is to return to functional and anatomical normality, and the durability of repair is a key issue. Looking just at immediate results in these conditions is too short. It is widely recognized that better results are achieved in degenerative disease, especially with isolated posterior leaflet prolapse, where operative mortality should be close to zero.¹⁵ By contrast, MV repair in secondary MR is associated with increased recurrence of regurgitation, while the results in rheumatic disease are in between these 2 etiologies, although several works have lately reported good long-term results.¹⁶

In conclusion, clinical results during and after MV surgery are mostly influenced by the surgical technique (MV repair is generally better) and preoperative characteristics of patients, but different etiologies are associated with different

types of comorbidities that can indirectly influence surgical outcomes (LV dysfunction, previous myocardial infarction, older age, etc). Here, we have to disagree with the conclusions of Rzucidło-Resil et al.¹ The authors are, naturally, commended for their work, which raises the discussion, but we recommend that other studies be conducted to confirm or dispute the results and conclusions achieved by this group.

ARTICLE INFORMATION

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