

Mild to Moderate Mitral Regurgitation in Patients with Atrial Fibrillation: Are we Drying Ice? What can be Done?

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Primary Mitral Regurgitation (MR) covers all etiologies in which intrinsic injuries disturb one or numerous constituents of the mitral valve apparatus. The abridged incidence of rheumatic fever and augmented lifespan in industrialized countries have progressively changed the distribution of etiologies, with degenerative MR now being the most common [1-3]. Echocardiography is the principal investigation and must include an assessment of severity, mechanisms, reparability, and consequences [4]. In chronic primary MR, the pathology of ≥ 1 of the components of the valve (leaflets, chordae tendineae, papillary muscles, annulus) causes valve incompetence with systolic regurgitation of blood from the left ventricle (LV) to the left atrium. The stages of primary MR are: at risk of (mild) MR, progressive (moderate) MR, asymptomatic severe MR, and symptomatic severe MR [5]. According to 2012 European Guidelines on the management of valvular heart disease [6] the indications for surgery in severe chronic primary MR in the presence of Atrial Fibrillation (AF), surgery must be well thought-out in asymptomatic patients with preserved LV function and new onset AF or pulmonary hypertension (systolic pulmonary pressure at rest >50 mmHg). The 2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease [5] suggests that mitral valve reparation is rational for asymptomatic patients with chronic severe non-rheumatic primary MR and preserved LV function in whom there is a great likelihood of a positive and durable repair with new onset of AF or resting pulmonary hypertension (pulmonary artery systolic arterial pressure >50 mm Hg). But what to do when the patients develop paroxysmal AF and have mild or moderate MR? The procedures to correct MR have no place yet. The ideal approach for the treatment of AF is rhythm control, but this is sometimes very hard to accomplish [7]. The Left atrial pressure (stenosis) and volume (regurgitation) load are the main drivers of atrial enlargement and structural atrial remodelling in these patients. Valvular heart disease can be associated with an increased thrombo-embolic risk, which probably also adds to the stroke risk in AF patients. Similar to heart failure, valvular disease, and AF interact with and sustain each other through volume and pressure overload, tachycardiomyopathy, and neurohumoral factors [7]. In fact, while AF implies an incremental risk for thrombo-embolism in patients with mitral valve stenosis, there is no clear evidence that other valvular diseases, including mitral regurgitation or aortic valve disease, need to be considered when choosing an anticoagulant or indeed to estimate stroke risk in AF [7]. New oral anticoagulants (NOACs), including the direct thrombin inhibitor dabigatran and the factor Xa inhibitors apixaban, edoxaban, and rivaroxaban, are suitable alternatives to vitamin K antagonists (VKAs) for stroke prevention in AF, according to the CHA₂DS₂-VASc score=Congestive Heart failure, hypertension, Age ≥ 75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65-74, and Sex (female). Their use in clinical practice is increasing rapidly. All NOACs have a predictable effect (onset and offset) without the need for regular anticoagulation monitoring [7].

AF frequently accompanies valvular heart disease. Left Atrium (LA) distension is an initial expression of progressive mitral valve illness, and the existence of paroxysmal or permanent AF is a recognized indication for prompt percutaneous or surgical mitral intervention [8]. AF is also normally seen in advanced stages of aortic valve disease when LV dilatation and high end-diastolic pressure exert secondary effects on LA function. Management of AF follows conventional recommendations in the setting of valvular heart disease, although a rate control strategy is typically adopted because of the low likelihood of sustaining sinus rhythm in the long term. Principal concerns surround the high risk of thrombo-embolism in subjects with valvular heart disease, and a low threshold for

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anticoagulation is recommended⁷. According to 2016 ESC AF guidelines: VKAs therapy (INR 2.0-3.0 or higher) is recommended for stroke prevention in AF patients with moderate-to-severe mitral stenosis or mechanical heart valves (Class I, Level B). The optimal antithrombotic therapy in the first months after biological valve replacement (including after catheter-based valve replacement) is not known. VKAs remain the mainstay during the initial post-operative period; NOACs probably deliver the same protection. In patients without AF, many centres use platelet inhibitors only [7]. VKAs are currently the only treatment with established safety in AF patients with rheumatic mitral valve disease and/or a mechanical heart valve prosthesis [7].

Recently, we reported that during 18 months of follow-up, AF recurrence was higher in the moderate MR patients who underwent PVI than in the other groups (no MR and mild MR) because of the major mechanical problem in those mitral valves, generating an augmented pressure and a greater stress into the LA, perpetuating the vicious AF cycle, and worsening the recurrence rate [8]. Due to the largest AF recurrence rates among patients with mild to moderate MR in comparison with patients without MR, should we think about to perform the surgical valve repair and surgical AF ablation procedure rather than simply perform the PVI? Should the catheter-based interventions to correct MR percutaneously (MitraClip procedure) take place in such cases?

Conflicts of interest

nothing to disclose.

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