Chronic ischaemic mitral regurgitation: exercise testing reveals its dynamic component

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This editorial refers to ‘Exercise-induced changes in mitral regurgitation in patients with prior myocardial infarction and left ventricular dysfunction: relation to mitral deformation and left ventricular function and shape’† by V. Giga et al., on page 1860

Ischaemic mitral regurgitation (IMR) is a complication of coronary heart disease, particularly in the setting of a prior myocardial infarction. Its incidence and clinical importance are largely underestimated partly because physical examination is rather insensitive. IMR occurs despite a structurally normal mitral valve as a consequence of a ventricular disease. It results from distortion of left ventricular (LV) geometry tethering the mitral leaflets and from decreased LV force to close them.1

When present, IMR may exhibit a broad range of severity and conveys a dismal prognosis. The increased mortality risk relates not only to the presence, but also more importantly to the quantified degree of IMR. Several methods can be used to determine the severity of IMR. Semi-quantitative approaches, the colour flow mapping of the regurgitant jet, and the vena contracta width seem to be of limited value, whereas both the Doppler and the PISA methods can quantitate IMR accurately. The effective regurgitant orifice (ERO) area of IMR is the most robust measurement. In the setting of ischaemic heart disease, an ERO ≥ 20 mm² is considered severe and associated with excess mortality.2 However, the evaluation of IMR only under resting conditions might underestimate the full impact of the lesion and its clinical effects. Indeed, IMR is a dynamic lesion and its severity may vary over time. Exercise Doppler echocardiography has recently emerged as a well-suited method to quantitate the dynamic component of IMR. There is a good correlation between ERO measured during exercise by the PISA method and that obtained by the quantitative Doppler volumetric method.3

Exertional dyspnoea is a cardinal symptom in patients with ischaemic LV dysfunction and can occur out of proportion to the LV dysfunction or the degree of IMR at rest. In such a situation, exercise-induced increases in IMR severity might limit the stroke volume adaptation during exercise and contribute in part to the limitation of exercise capacity.4 Intermittent increases in IMR severity during the daily activities of life or during the night could also raise left atrial and pulmonary vascular pressures acutely, generating pulmonary congestion and contributing to worsening dyspnoea or flash pulmonary oedema. The relationship between dynamic IMR and these clinical spectrums has been confirmed recently. Exercise-induced changes in regurgitant volume and in systolic pulmonary pressure are larger in patients who stop their exercise because of dyspnoea as compared with those who stop for fatigue.3 The magnitude of increase in regurgitant volume is also greater in patients hospitalized for pulmonary oedema in the context of chronic systolic LV dysfunction.5 Dynamic changes in IMR provide additional prognostic information over resting evaluation and unmask patients at high risk of poor outcome.6 A large exercise-induced increase in IMR, an increase in ERO ≥ 13 mm², is associated with increased mortality and morbidity—hospital admission for worsening heart failure and major cardiac events.7 Many patients with dynamic IMR die from refractory heart failure. Repetitive transient increase in mitral regurgitation may accentuate the chronic volume overload induced by IMR and contribute to progressive LV dilation and dysfunction, leading in turn to end-stage heart failure. Dynamic IMR is also a determinant of rapid QRS widening and may subsequently lead to permanent electromechanical dyssynchronization, which further deteriorates the LV systolic function.8

Incomplete closure of normal mitral leaflets, the cause of IMR, is due to complex distortion of ventricular geometry and of the mitral apparatus. The systolic mitral valve tenting, leaflet deformation, is the main mechanism of IMR and is due to local LV remodelling (apical and posterior displacement of the papillary muscle) rather than to global LV remodelling.1 During exercise, the magnitude of changes in IMR is widely different from one patient to another and does not correlate with the degree of IMR at rest. Most patients exhibit small increases in the amount of IMR, whereas others have either a large rise or a significant decrease in ERO.9 The results of Giga et al.10 confirm these previous observations in a cohort of 40 patients with chronic revascularized myocardial infarction. Thirty-one (78%) of them displayed an increase in IMR during exercise, whereas IMR decreased in the remaining nine patients (22%). These dynamic changes in IMR strikingly correlated to changes in mitral valve deformation as well as to changes...
in wall motion score index and in end-systolic sphericity index. The strongest correlation was observed with changes in mitral valve configuration. The increase in ERO during exercise resulted from a bulging in systolic tenting area (the area enclosed between the mitral leaflets and the annulus plane), an increase in coaptation distance (apical displacement of the coaptation leaflet tips), and a systolic expansion of the mitral annulus. Importantly, such changes occurred without detectable ischaemia at exercise echo and were independent of exercise-induced changes in haemodynamic parameters (arterial pressure, heart rate). Changes in regional loading conditions and a more spherical LV shape during exercise might contribute to changes in mitral valve configuration.\(^4\)\(^{10}\) The decrease in ERO during exercise was observed mainly in patients with inferior infarction who had recruitable function in the basal segments. Such a reduction is probably related to the reduction of the tethering forces and can be interpreted in the light of an experiment model of reverse remodelling induced by repositioning of the papillary muscles.\(^3\)\(^1\)

Thus, the study of Giga et al.\(^{10}\) provides complementary information about the complexity of the mechanisms associated with dynamic IMR. Practically, exercise echocardiography is indeed a useful tool to unmask what might otherwise be considered a moderate lesion. The questions arising from these studies are (i) should exercise Doppler echocardiography be performed in all patients with IMR and (ii) how would the results of this test affect the clinical decision making. Keeping in mind our previous results,\(^5\)\(^6\)\(^9\) it seems that the dynamic component of IMR might be tested: (i) in patients with chronic ischaemic LV dysfunction presenting with unexplained dyspnea or acute pulmonary oedema without evident contributing factors, (ii) for risk stratification in the individual patient, and (iii) before by-pass grafting in patients with moderate IMR. In the latter situation, the study of Giga et al.\(^{10}\) suggests that exercise-induced reduction in MR as a result of recruitable function in the basal part of the inferior wall would predict that effective revascularization alone may reduce the IMR. If a significant increase in ERO (≥ 13 mm\(^2\)) develops with exercise, a combined treatment, by-pass, and mitral valve surgeries might be proposed. The efficacy of mitral ring annuloplasty as well as the place of more specific sub-valvular approaches need to be tested. When a revascularization procedure is not indicated, biventricular approaches can potentially reduce the severity of IMR at rest and its dynamic component.\(^11\)

References