Exercise-Induced Severe Mitral Regurgitation in a Patient with Mild Rheumatic Mitral Valve Disease at Rest: a Case Report

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Abstract

Exertional dyspnea is usual in patients with severe rheumatic mitral stenosis or regurgitation, but this symptom can also occur in patients with seemingly mild mitral valve disease. In these situations, exercise-Doppler echocardiography may be helpful to relate the symptoms to the hemodynamic response to exercise. We report a case of severe dynamic mitral regurgitation occurring during exercise-Doppler echocardiography in a patient with only mild rheumatic mitral stenosis and regurgitation at rest.

Keywords: Rheumatic mitral valve disease; Mitral stenosis; Exercise echocardiography; Dynamic mitral regurgitation

Abbreviations: MVD: Mitral Valve Disease; MR: Mitral Regurgitation; MS: Mitral Stenosis

Case Presentation

A 55 year-old woman with known mild rheumatic MVD was referred for exercise-Doppler echocardiography because of exercise dyspnea out of proportion to the resting echocardiographic assessment. Her past history included: percutaneous mitral balloon valvuloplasty in 2002; paroxystic atrial fibrillation in 2004; ablation of atrial fibrillation and pacemaker (AAI) implantation for sinus dysfunction in 2010. The patient was in New York Heart Association (NYHA) functional class III under treatment of beta-blockers and flecainide.

Two-dimensional (2D) baseline echocardiographic examination was performed at rest and repeated at exercise on a supine table with a Vivid 7 imaging system (GE Healthcare, Oslo, Norway).

The examination at rest showed good result of the mitral valvuloplasty as follows: mitral valve area (MVA) of 1.8 cm² by planimetry (Figure 1); bi-commissural opening of the valve; mean diastolic transmitral gradient (TMG) of 4 mmHg; mild MR with effective regurgitation orifice area (EROA) of 0.12 cm² (Video 1); systolic pulmonary artery pressure (sPAP) of 40 mmHg for an estimated right atrial pressure of 10 mmHg. Left ventricle dimensions and function were normal (telediastolic diameter: 51 mm; telesystolic diameter: 35 mm; ejection fraction: 60%).

Exercise protocol began at a work load of 20W, with an increase of 20W every 2 minutes. Continuous electrocardiographic (ECG) monitoring was used and blood pressure was measured every 2 minutes. Doppler echocardiographic data were obtained at rest and at each step of the exercise test in the same semisupine position.

The exercise lasted 7, 27 minutes and was stopped at 69% of the predicted heart rate at 80 Watt. The heart rate increased from 75 to 100 beats/min; systolic blood pressure increased from 150 to 176 mmHg.

Since the first level of exercise, despite good clinical tolerance, color Doppler identified a marked worsening of MR, with a large central jet, large extension in the left atrium and quantitative parameters of severe MR (EROA 0.40 cm²) (Figure 2 and Video 2) associated to a simultaneous visual increase of TR severity at color-Doppler and early elevation of sPAP (estimated sPAP 65 mmHg). Exercise stopped because of aggravation of dyspnea and fatigue. During the whole test MR remained severe with a mean diastolic TMG increasing from 4 to 8 mmHg (Figure 3) and a sPAP at peak of 90 mmHg (Figure 4). There were no new segmental wall motion abnormalities and no electrocardiographic evidence of ischemia. Left ventricular end-systolic and end-diastolic volumes did not changed from values at baseline. In the recovery period the MR and the sPAP progressively reached the baseline characteristics.

**Figure 1:** Rheumatic mitral valve stenosis: parasternal grand axe view and parasternal small axe view.

**Video 1:** Mitral regurgitation at baseline.
**Discussion**

We report an unusual case of dynamic MR in a patient with mild rheumatic mitral valve disease at rest.

Exertional dyspnea is usual in patients with severe MS or MR, but this symptom can also occur in patients with seemingly mild mitral valve disease when evaluated at rest [1,2].

In this case, exercise echocardiography showed severe dynamic MR in the absence of acute myocardial ischemia, left ventricular dysfunction or valve prolapse. We noticed in our patient a small increase in heart rate (25 beat/min) under betablockers and pacemaker stimulation. We did not observe any changes in the left ventricular volumes from baseline to the peak of the exercise (EDV 100 to 103 ml; ESV 38 to 38 ml). Although rarely reported, Tischler et al. [3,4] described similar cases of worsening MR during exercise in fourteen symptomatic patients with exertional dyspnea and mild rheumatic mitral stenosis and regurgitation at rest. They observed also a marked increase on sPAP and mean TMG during dynamic exercise in these patients. In their paper, the mechanism of worsening of MR remains unclear but they suggested the possibility that a smaller heart rate increase and the unchanged left ventricular geometry could have lead to a morphological deformation of the valve that, in some way, influenced its function. In our case, another potential mechanism that could influence the role of left ventricular geometry for the increasing of MR is the septal dyssynchrony induced by the presence of PM.

**Conclusion**

The present case underlines the role of exercise echocardiography in interpreting symptoms in patients with rheumatic valve disease, either MS or mixed MV disease. In addition to the conventional hemodynamic parameters that are assessed during exercise, mean mitral gradient and pulmonary pressure, it is important to look at the dynamic variation of MR during exercise echocardiography that can occur in a minority of patients.

**References**


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