Dynamic Exercise-Induced Right Ventricular Outflow Tract Obstruction in a Patient with Hypertrophic Cardiomyopathy

To the Editor,

A 42-year-old man was referred to our echocardiography ward for stress echocardiography on suspicion of left ventricular outflow tract (LVOT) obstruction. The patient was a known case of hypertrophic cardiomyopathy with a complaint of dyspnea on exertion, compatible with the New York Heart Association functional class III. There was no history of syncope or family history of sudden cardiac death. The electrocardiography showed right bundle branch block and T-wave inversion in I, aVL, and precordial leads. He used 50 mg of metoprolol twice per day. The physical examinations were unremarkable. The transthoracic echocardiography demonstrated hypertrophy in the anteroseptal, anterior, and anterolateral walls, with maximal thickness in the mid anteroseptal wall (21 mm) without systolic anterior mitral leaflet motion. Additionally, the left ventricular ejection fraction was about 65%. There was no LVOT obstruction at rest and after the Valsalva maneuver (peak pressure gradient = 6 mm Hg). A late peaking pressure gradient of 16 mm Hg was detected in the right ventricular outflow tract (RVOT), probably due to dynamic obstruction. There was no muscle bundle in the RVOT. The pulmonary valve had a normal appearance. The right ventricle had normal size and function, and there was no measurable tricuspid regurgitation peak velocity. The left atrial size was normal. The patient exercised for 2 min and then requested the termination of the test because of dyspnea. The heart rate increased from 117 bpm to 178 bpm. The LVOT peak pressure gradient rose to 12 mm Hg, and the RVOT peak pressure gradient increased to 45 mm Hg [Figure 1]. No regional wall motion abnormality appeared.

The presence of RVOT obstruction in hypertrophic cardiomyopathy has been reported previously in isolation or combination with LVOT obstruction. It appears that the therapeutic recommendations are the same as those for LVOT obstruction; nonetheless because of the low prevalence of this condition the evidence for it is not as robust as that for LVOT obstruction.1-2 In this case, we mainly attributed the presence of dyspnea on exertion to left ventricular diastolic dysfunction. In addition, we considered dynamic RVOT obstruction as a rare possible ancillary cause. Therefore, we opted to intensify medical treatment with beta-blockers and follow the patient. The presence of ROVT obstruction in hypertrophic cardiomyopathy that appears in exercise stress echocardiography should be noted during stress echocardiography because it may be a rare possible ancillary explanation for dyspnea in addition to the left ventricular diastolic dysfunction as a main cause of dyspnea.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and Figure 1: Late peaking right outflow tract pressure gradient of 16 mm Hg (a), which increased to 45 mm Hg with limited exercise (b). The right ventricular obstruction in 2D echocardiography with color flow Doppler in the parasternal short-axis view of transthoracic echocardiography (c). RVOT; Right ventricular outflow tract, PA; Pulmonary artery, PG; Peak gradient.
other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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