

Significant left ventricular outflow tract obstruction observed during postexercise verticalization in a symptomatic patient with hypertrophic cardiomyopathy

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In patients with hypertrophic cardiomyopathy (HCM), identification of left ventricular outflow tract (LVOT) obstruction is important in the management of symptoms and assessment of the risk of sudden cardiac death. In symptomatic patients with HCM without a significant LVOT obstruction at rest, and if bedside maneuvers fail to induce a LVOT pressure gradient of 50 mm Hg or higher, exercise stress echocardiography is recommended to assess LVOT obstruction during exercise (Class I, Level B).¹ Until now, there have been 3 types of protocols used for stress echocardiography to assess LVOT pressure gradient in patients with HCM: fully physiological (an upright position during exercise and recovery), nonphysiological (a supine position at both stages), and semi-physiological (an upright position during exercise and a supine position at recovery).² We suggest using, in some cases, the fourth type, namely, reverse semi-physiological (a supine or semi-supine position during exercise and an upright position during recovery).

A 65-year-old obese woman (body mass index, 34 kg/m²) was admitted to our department because of type 2 myocardial infarction secondary to the episode of paroxysmal atrial fibrillation with fast ventricular rhythm. She had a history of recurrent syncope, arterial hypertension, hyperlipidemia, type 2 diabetes, obesity, and smoking, as well as a family history of sudden cardiac death.

On admission, the patient was in poor clinical condition (sweating, weakness), with a resting

irregular heart rate of 175 bpm and a blood pressure of 110/80 mm Hg. Baseline laboratory tests indicated normal red and white blood cell count, electrolyte, and creatinine levels, as well as elevated lipid and cardiac troponin T levels (maximum value, 1242 ng/ml; upper limit of normal, 14 ng/ml). Atrial fibrillation was converted to sinus rhythm after an intravenous administration of metoprolol and amiodaron. Coronary angiography revealed no abnormalities in coronary arteries. The patient was treated with a β -blocker, angiotensin-converting enzyme inhibitor, statin, and vitamin K antagonist. On electrocardiography during sinus rhythm, left ventricular hypertrophy was present. Resting echocardiography revealed significant left ventricular hypertrophy (interventricular septum, 21 mm; posterior wall, 14 mm), without LVOT obstruction at rest and after the Valsalva maneuver (FIGURE 1A and 1B), left ventricular ejection fraction of 65%, diastolic dysfunction (grade I), slight left atrial enlargement, mild mitral and tricuspid insufficiency, and normal right ventricular systolic function. No pathology on chest X-ray was present. During in-hospital cardiac rehabilitation, an episode of syncope occurred, following which the patient was referred for exercise stress echocardiography to assess LVOT obstruction during exercise.

Exercise stress echocardiography was performed on a semi-supine cycle ergometer, using the ramp protocol (25 wats/2 minutes). The exercise was terminated after 5 minutes and 57 seconds because of excessive fatigue (8 points on the 10-point Borg scale) without

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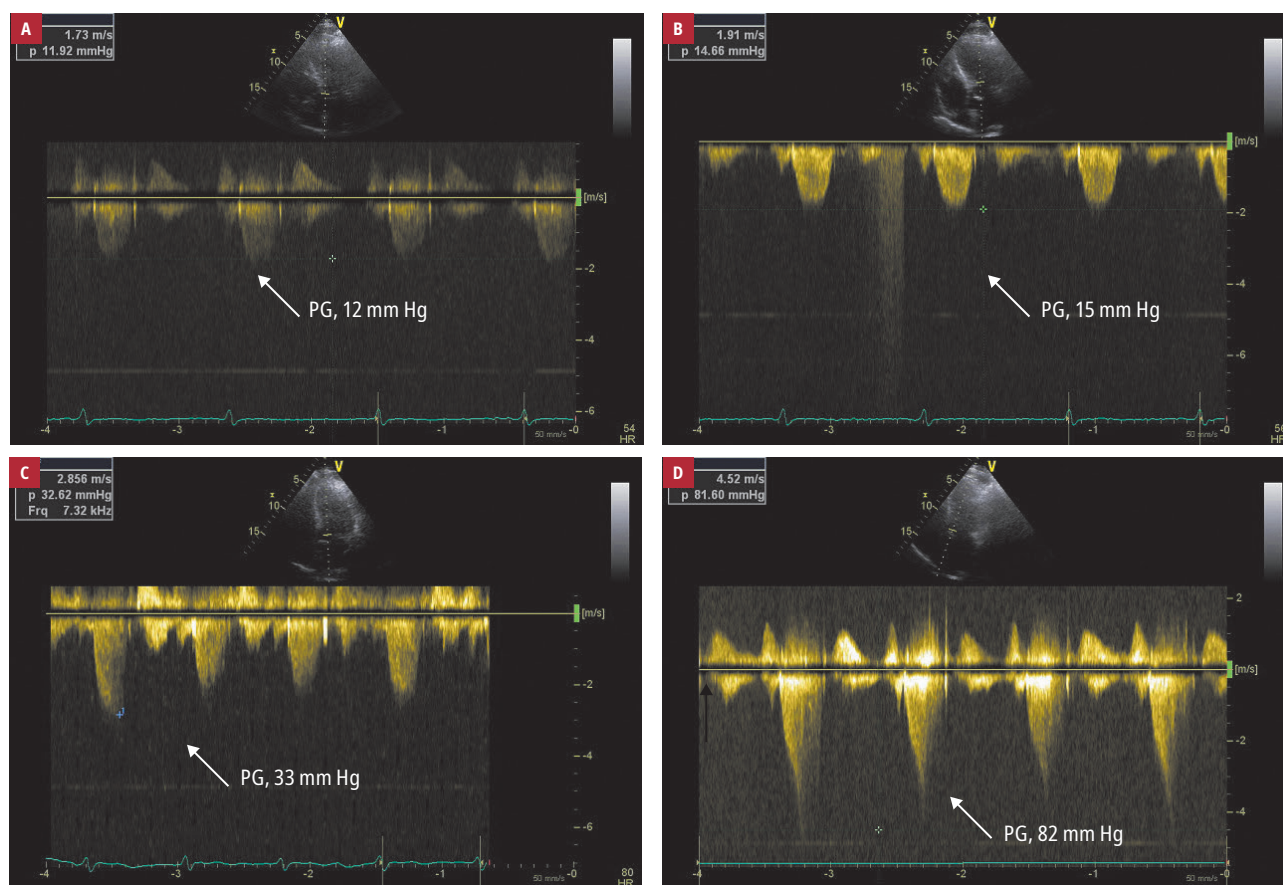


FIGURE 1 Continuous-wave Doppler ultrasound showing left ventricular outflow tract maximum pressure gradient (arrows) at rest (A), during the Valsalva maneuver (B), at peak exercise (C), and during verticalization (D)

arrhythmia and ischemia. The maximum load achieved was 69 wats (80% predicted), the heart rate was 84 bpm (54% predicted), and systolic blood pressure was 140 mm Hg (an increase from 115 mm Hg).

Hyperkinetic left ventricular response to exercise was present without regional wall motion abnormalities. There was no hemodynamically significant LVOT obstruction at peak exercise and after exercise cessation (FIGURE 1C). After verticalization, in the third minute of recovery, symptoms of presyncope with a rapid drop in systolic blood pressure (105 mm Hg) occurred, and LVOT obstruction with the maximum pressure gradient of 82 mm Hg was recorded (FIGURE 1D).

The “verticalization test” during the recovery phase may reveal a significant LVOT gradient in symptomatic patients with HCM without LVOT obstruction at peak exercise.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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