Patent Ductus Arteriosus Mimicking Hypertrophic Cardiomyopathy

To the Editor,

A 30-year-old woman was referred to our echocardiography ward for further evaluation of her hypertrophic cardiomyopathy. The patient had been diagnosed with asymmetric septal hypertrophy in initial echocardiography in another center before ophthalmic surgery. Her cardiovascular chief complaint was dyspnea on exertion, compatible with functional class II in the New York Heart Association’s classification. Physical examinations revealed clubbing, which was more prominent in the lower extremities, and load the second pulmonic sound. Electrocardiography demonstrated right-axis deviation with a right ventricular (RV) strain pattern. The systemic blood pressure was about 130/80 mm Hg, and O₂ saturation was 91% in the hand and 86% in the foot.

Transthoracic echocardiography (TTE) revealed asymmetric septal hypertrophy (septal thickness = 16 mm, posterior wall thickness = 9 mm), a D-shaped left ventricle (LV) in systole, severe RV hypertrophy (16 mm), mild tricuspid regurgitation, absence of the LV outflow tract, and obstruction of the LV mid-cavity. TTE also illustrated severe pulmonary hypertension (tricuspid regurgitation gradient = 130 mm Hg), a dilated inferior vena cava (22 mm), more than 50% inspiratory collapse, and an estimated systolic pulmonary artery pressure of 140 mm Hg, suggesting that the main pathology might be something else. The other TTE findings were normal LV size and function, mild RV enlargement, moderate-to-severe RV systolic dysfunction, and absence of the valvular or subvalvular pulmonary stenosis. However, the patient did not have a clear echocardiography window in the parasternal short-axis view at the level of the aortic valve and the suprasternal view. The patient’s pulmonary hypertension was further assessed through the transesophageal echocardiography, which demonstrated a large patent foramen ovale (3 mm) with a bidirectional shunt and a patent ductus arteriosus with a bidirectional shunt [Figure 1 and Videos 1-3].

The left and right catheterization revealed systolic, diastolic, and mean pulmonary artery pressures of 180, 60, and 100
mm Hg, respectively, with pulmonary vascular resistance of 27 Wood units and systolic, diastolic, and mean aortic pressures of 160, 60, and 93 mmHg, correspondingly, with systemic vascular resistance of 23 Wood units. The wide pulse pressure of the patient’s pulmonary artery may have been due to her excessive anxiety in the catheterization laboratory, which might also have led to the wide systemic pulse pressure at the time.

The LV and RV systolic pressures were 180 and 160 mm Hg, respectively. Further, O₂ saturation was 60% in the superior vena cava, 60% in the right atrium, 57% in the RV, 70% in the pulmonary artery, 95% in the pulmonary vein, 92% in the LV, 92% in the ascending aorta, and 86% in the descending aorta. The patient was treated with a combination of bosentan and tadalafil.

Although asymmetric septal hypertrophy is a prominent echocardiographic sign of hypertrophic cardiomyopathy, the presence of other findings that are not compatible with hypertrophic cardiomyopathy should hint at the existence of other etiologies, one of which is the Eisenmenger syndrome.

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 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.

Zahra Khajali, Ali Hosseinsabet¹
Department of Cardiology, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, ¹Department of Cardiology, Tehran Heart Center, Tehran University of Medical Sciences, Tehran, I.R. Iran

Address for correspondence: Dr. Ali Hosseinsabet, Tehran Heart Center, Kargar Shomali Street, Tehran, I.R. Iran.
E-mail: Ali_Hosseinsabet@yahoo.com

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