Systolic Anterior Motion of the Mitral Chordae Tendineae as a Possible Etiology for a Significant Left Ventricular Outflow Tract Obstruction

Sir,

A 62-year-old obese woman with a history of atypical chest pain and cardiac murmurs was referred to our echocardiography laboratory for further evaluations. She had a history of controlled hypertension of 5 years' duration, for which she was on losartan. In physical examination, a systolic murmur (grade III/VI) was auscultated at the left parasternal border. Electrocardiography showed an intraventricular conduction delay; QRS duration was about 100 ms, left axis deviation, and QS pattern in all precordial and inferior leads with secondary ST-T changes. Despite poor echocardiography windows, transthoracic echocardiography revealed that the base and mid segments of the anteroseptal and inferoseptal walls were hypertrophied with a maximal thickness of 15 mm and a moderate-to-severe subaortic stenosis with a peak pressure gradient of 57 mmHg. Transesophageal echocardiography showed no aortic valve stenosis, no mitral valve prolapse, trivial mitral regurgitation, and a significant systolic anterior motion of the mitral chordae tendineae [Figure 1 and Video 1]. It seems that the systolic anterior motion of the mitral chordae tendineae was the main etiology of the left ventricular tract obstruction.

When we encounter dynamic left ventricular outflow tract, we expect that moderately severe mitral regurgitation would exist, but if the amount of mitral regurgitation is less than expected, mid-cavity obstruction (due to broad base of continuous wave), systolic anterior motion of posterior mitral leaflet, or systolic anterior motion of chordae tendineae as possible cause of dynamic left ventricular obstruction should be considered.[1-5] Accordingly, such as presented case, cardiologists should bear in mind that the systolic anterior motion of the mitral chordae tendineae can result in a significant left ventricular outflow tract obstruction.

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 The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

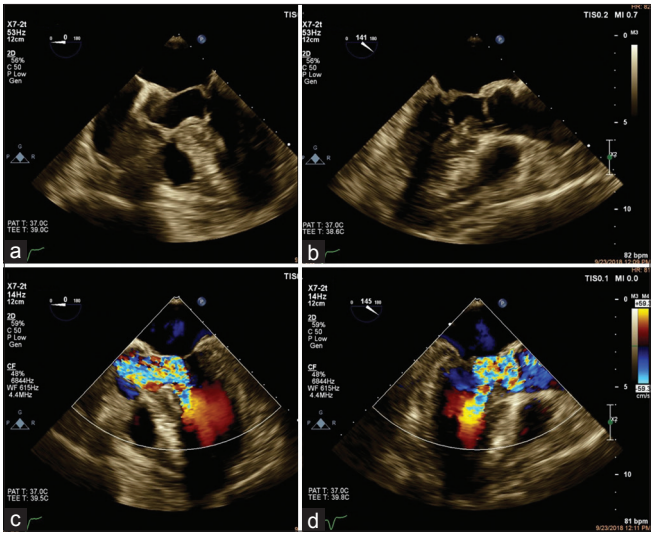

Figure 1: Systolic anterior motion of the mitral chordae tendineae (arrow) in transesophageal echocardiography [(a) mid-esophageal five-chamber view and (b) mid-esophageal long-axis view of the aortic valve], resulting in left ventricular outflow obstruction in color Doppler study [(c) mid-esophageal five-chamber view and (d) mid-esophageal long-axis view of the aortic valve]. LA, left atrium; LV, left ventricle; AO, ascending aorta

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Conflicts of interest

There are no conflicts of interest.

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Incidentally Detected Biventricular Noncompaction Cardiomyopathy

Sir,

A 31-year-old woman with a complaint of atypical chest pain but without dyspnea or palpitation was referred to our echocardiography laboratory for further evaluation. Her medical history was unremarkable, and her electrocardiography showed a normal sinus rhythm without any abnormal findings.

Transthoracic echocardiography revealed preserved systolic functions and normal sizes for both left and right ventricles as well as hypertrabeculation in the left ventricular apex (extending to the mid part of the inferior and lateral walls) and the right ventricular apex, with deep recess formation and flow within, suggestive of biventricular noncompaction cardiomyopathy [Video 1 and Figure 1]. The patient was referred for cardiac magnetic resonance imaging, which confirmed the transthoracic echocardiographic findings [Video 2 and Figure 2].

The left ventricular noncompaction is a rare cardiomyopathy characterized by noncompacted thickness-to-compacted thickness ratio in systole of more than 2, deep recesses, and blood flow in these recesses, and apical and mid-ventricular segments are the most commonly affected places.[1] The left ventricular noncompaction can present with heart failure, arrhythmia, and thromboembolism, and even sudden cardiac death may occur in association with other congenital heart disease.[2] Favorable remodeling and improvement in ventricular systolic function is documented with appropriate medical therapy in patients with reduced systolic function and dilated ventricle.[3]

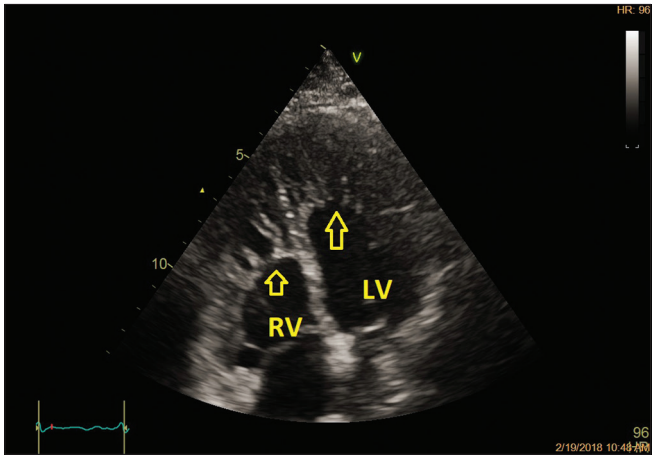

Figure 1: Transthoracic echocardiography in the four-chamber view, demonstrating hypertrabeculation with deep recesses. LV, left ventricle; RV, right ventricle

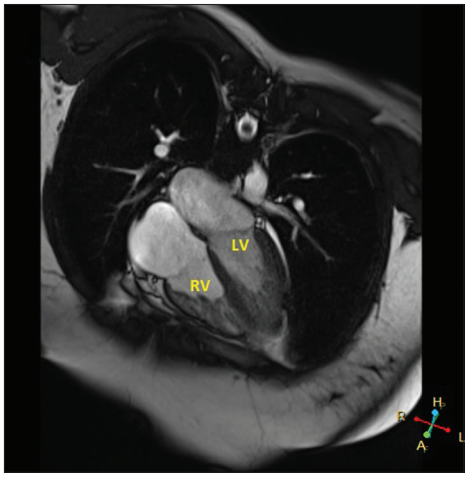

Figure 2: Cardiac magnetic resonance imaging in the transverse view, revealing noncompaction parts in the left and right ventricles. LV, left ventricle; RV, right ventricle