Systolic Flow Reversal in a Case of Mid-Ventricular Obstructive Hypertrophic Cardiomyopathy

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Abstract

A 69-year-old man presented to our hospital with chest pain. Two-dimensional transthoracic echocardiography showed hypertrophy of the left ventricle, mid-ventricular obstruction and an apical aneurysm. Color-flow imaging at the obstruction site on the apical four-chamber view demonstrated systolic flow reversal in addition to a paradoxical jet flow. The systolic flow reversal may have been caused by a decreased apical contractility and pressure during systole.

Key words: systolic flow reversal, mid-ventricular obstruction, Doppler echocardiography

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Introduction

Apical aneurysms may be present in patients with mid-ventricular obstructive hypertrophic cardiomyopathy (MVOCM) (1) and are frequently associated with an unusual type of intraventricular isovolumetric relaxation flow called a paradoxical jet flow (2). We herein report a rare case of MVOCM associated with additional systolic flow reversal from the base of the ventricle (LV) to the apex.

Case Report

A 69-year-old man presented to our hospital after experiencing chest pain and a transient loss of consciousness while driving a taxi. His blood pressure was 140/95 mmHg and his pulse rate was 87 beats/min. Auscultation showed a Levine 2/6 systolic murmur at the right upper sternal border, while a 12-lead electrocardiogram (ECG) showed a sinus rhythm with complete right bundle branch block and negative T waves. In addition, a chest radiograph disclosed a cardiothoracic ratio of 0.52, and two-dimensional transthoracic echocardiography revealed hypertrophy of the left LV (interventricular septum: 14 mm thick, and LV posterior wall: 13 mm thick) as well as mid-ventricular obstruction and an apical aneurysm (Fig. 1). Continuous-wave Doppler echocardiography of the site of obstruction (Fig. 2) and color-flow imaging in the apical four-chamber view (Fig. 3) demonstrated systolic mid-ventricular obstruction and a paradoxical jet flow typical of MVOCM. Furthermore, there was a systolic flow reversal from the LV base to the apex between the systolic and paradoxical jet flow (Fig. 2, 3), and cardiac MRI imaging showed massive LV hypertrophy and mid-ventricular obstruction (Fig. 4, right panel, arrows).

The patient underwent cardiac catheterization. Although no significant coronary artery stenosis was observed, coronary spasms were induced by the intracoronary injection of acetylcholine. The patient’s hemodynamic data showed a mean pulmonary capillary wedge pressure of 12 mmHg, LV end-diastolic pressure of 25 mmHg, cardiac output of 5.5 L/min and cardiac index of 2.6 L/min/m². There were no significant peak systolic pressure gradients in the LV apex, LV base or aorta (Fig. 5).

The patient was subsequently treated with a calcium channel blocker and angiotensin II receptor blocker and discharged from our hospital.

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Figure 1. Apical four-chamber view in diastole (left panel) and systole (right panel) shows an apical aneurysm (An.) and mid-ventricular obstruction (arrows).

Figure 2. Continuous Doppler echocardiography at the site of left ventricular obstruction shows mid-systolic flow reversal (a) and a paradoxical jet flow (b).
Figure 3. Color-flow imaging in the apical four-chamber view in systole (left panel), late-systole (middle panel) and isovolumetric relaxation (right panel) during the cardiac cycle. Systolic flow reversal is observed between the systolic forward flow and paradoxical jet flow.

Figure 4. Cardiac MRI imaging shows massive LV hypertrophy with mid-ventricular obstruction (arrows).

Discussion

MVOCM may be accompanied by apical aneurysm formation. Although the precise pathogenesis of apical aneurysms is unknown, these lesions may be caused by high apical pressure due to mid-ventricular obstruction (3).

Another proposed mechanism underlying aneurysm formation is myocardial ischemia. The oxygen supply may decrease as a result of an impaired coronary flow reserve, decreased coronary perfusion pressure, squeezing of the coronary arteries and coronary spasms (4, 5). The oxygen demand is also increased by high apical pressure and wall stress (6).

Moreover, enlargement of the apex and thinning of the wall caused by aneurysm formation may increase wall stress, which subsequently induces positive feedback, thus leading to a vicious cycle (Fig. 6).

This case may be the second reported case demonstrating additional transient mid-systolic flow reversal between the
systolic flow and a paradoxical jet flow. We previously encountered a case of atrial fibrillation in a patient with MVOCM and systolic flow reversal, suggesting that atrial fibrillation contributes to the development of this unusual flow (7). However, the present patient had a sinus rhythm. We therefore consider that the systolic flow reversal may have been caused by a decrease in the systolic apical pressure equal to the LV basal pressure as a result of hypokinesis of the apex (Fig. 6, left lower).

In conclusion, we herein reported a rare case of systolic flow reversal in a patient with MVOCM exhibiting an apical aneurysm.
The authors state that they have no Conflict of Interest (COI).

References


