Pseudo-Paradoxical Jet Flow in a Patient with Midventricular Obstructive Hypertrophic Cardiomyopathy

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INTRODUCTION

Paradoxical jet flow consists of midventricular flow from the apex toward the base during diastole.[1] This flow is typically observed in patients with midventricular obstructive hypertrophic cardiomyopathy and left ventricular (LV) apical aneurysm.[1] Here we describe a patient with midventricular obstructive hypertrophic cardiomyopathy with isovolumic relaxation flow that was caused by a different mechanism.

CASE PRESENTATION

A 60-year-old man with hypertrophic cardiomyopathy was admitted to our department for cardiac examination before surgery for chronic sinusitis.

A physical examination showed that the patient's blood pressure was 135/76 mm Hg and his pulse rate was 65 beats/min. Auscultation of the heart indicated that there was a grade II/VI systolic murmur at the second right sternal border. Blood sampling indicated renal injury and a high plasma level of brain natriuretic peptide (125 pg/mL). Twelve-lead electrocardiography depicted high R waves in the left precordial leads and giant negative T waves in leads I, aVL, and V_3 to V_6 (Figure 1A), suggestive of LV hypertrophy. Chest radiography exhibited a cardiothoracic ratio of 52% (Figure 1B).

Apical four-chamber transthoracic echocardiography revealed diffuse LV hypertrophy (Figure 2, Video 1). Parasternal short- and long-axis views demonstrated marked LV hypertrophy (in the long-axis view, end-diastolic thicknesses of the interventricular septum and posterior wall at the base were 18 and 13 mm, respectively; Figures 3 and 4, Video 2). LV ejection fraction was 73%, and M-mode echocardiography at the aortic valve level displayed midsystolic half closure of the aortic valve (Figure 5). Pulsed-wave Doppler echocardiography at the mitral inflow

VIDEO HIGHLIGHTS

Video 1: Apical four-chamber view showing massive LV hypertrophy with a normal ejection fraction.

Video 2: Parasternal long-axis view showing LV hypertrophy.

Video 3: Apical long-axis view showing midventricular isovolumic relaxation flow.

Video 4: Magnified simultaneous two-dimensional (*left*) and color flow (*right*) movies of apical long-axis view. Apposition of the septum and anteriorly oriented papillary muscle caused midventricular obstruction. Isovolumic relaxation flow was observed at the obstruction.

Video 5: Magnified simultaneous two-dimensional (*left*) and color flow (*right*) movies at the apical region. Small LV cavity and additional obstruction were present during systole (*large arrow*). During IVRT, isovolumic relaxation flow was observed. During early diastole, inflow was observed at the midventricular obstruction earlier than mitral inflow. During diastole, apical flow toward the base was observed (*small arrow*).

Video 6: Cardiac magnetic resonance imaging showing massive LV hypertrophy without LV obstruction or apical aneurysm.

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showed an E wave of 41 cm/sec, an A wave of 65 cm/sec, and deceleration time of the E wave of 328 msec (Figure 6A). Doppler tissue imaging at the septal annulus revealed an e' wave of 2.7 cm/sec and an E/e' ratio of 15.2, suggesting grade 1 LV diastolic dysfunction (Figure 6B).[2]

Color flow imaging demonstrated mild tricuspid regurgitation with an estimated peak pressure gradient of 23 mm Hg. The color flow apical long-axis view suggested midventricular obstruction and isovolumic relaxation flow directed from the apex to the base (Figure 7, Video 3). Magnified images of apical two-dimensional and color flow echocardiography revealed that the apposition of the septum and anteriorly oriented papillary muscle caused midventricular obstruction (Figure 8, Video 4).[3] Furthermore, a small LV cavity and an additional obstruction were present at the more apical region (Figure 9, Video 5). In early diastole, inflow velocity was observed at the midventricular obstruction earlier than mitral inflow. During mid-diastole, there was a small amount of flow at the apex oriented toward the
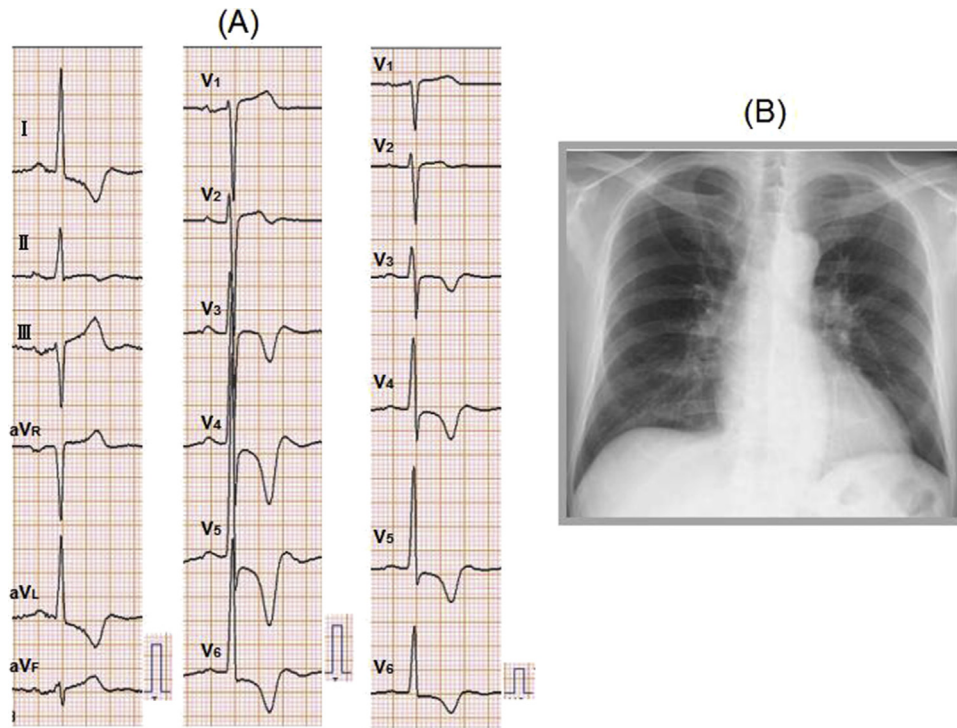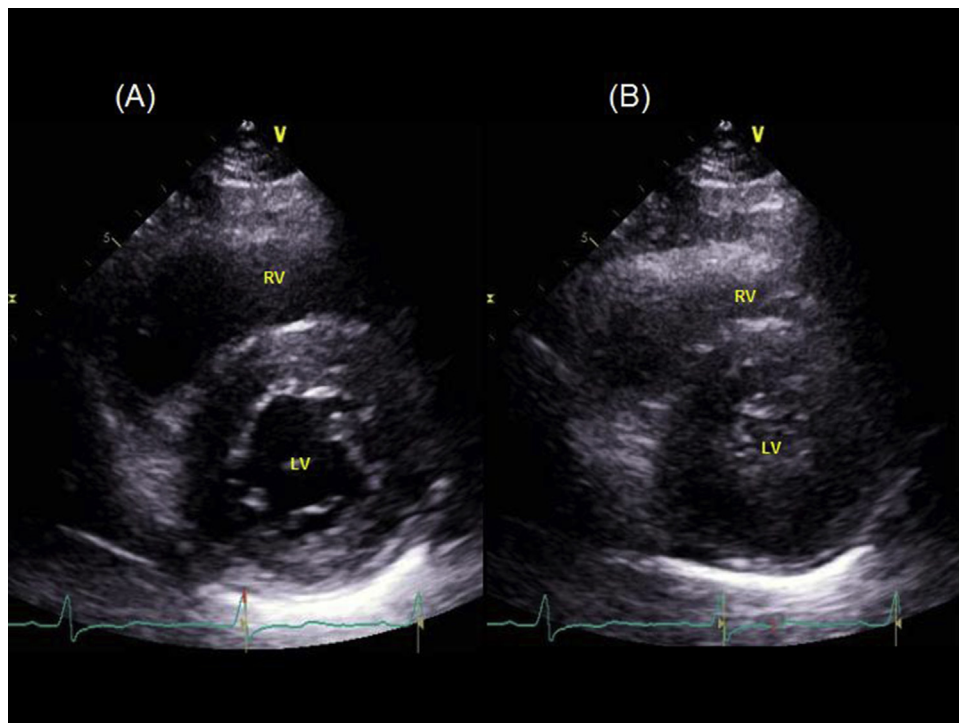
Figure 1 **(A)** Twelve-lead electrocardiogram showing high R waves in the left precordial leads and giant negative T waves in leads I, aVL, and V_3 to V_6. **(B)** Chest radiography showed a cardiothoracic ratio of 52%.
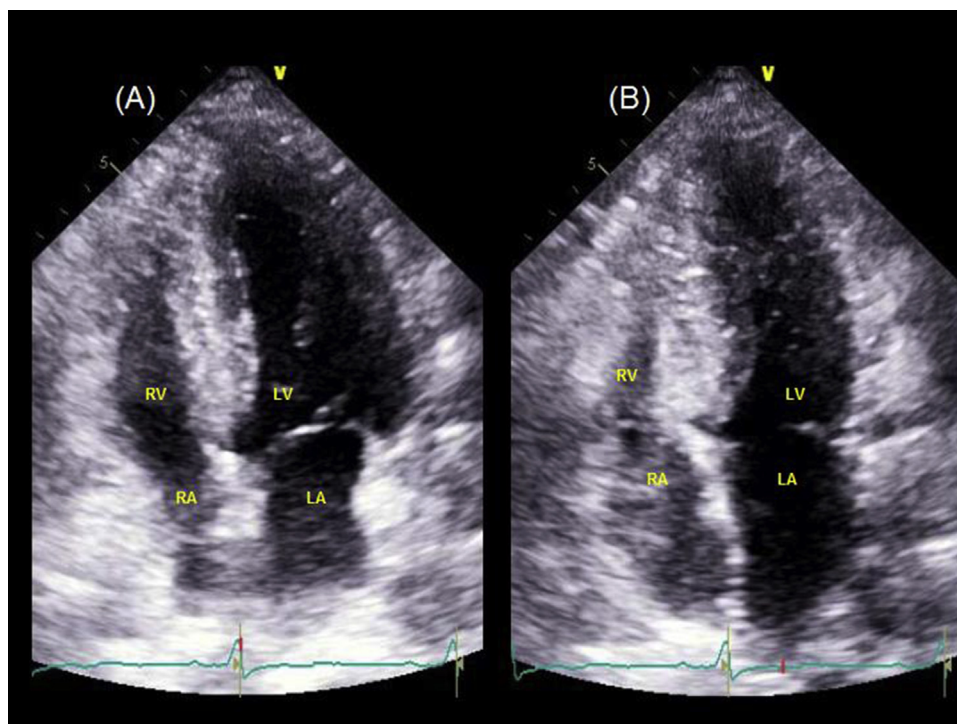

Figure 2 Apical four-chamber views during end-diastole **(A)** and end-systole **(B)**, showing massive LV hypertrophy with normal ejection fraction.

Figure 3 Parasternal short-axis views during end-diastole **(A)** and end-systole **(B)**, which show hypertrophied left ventricle.
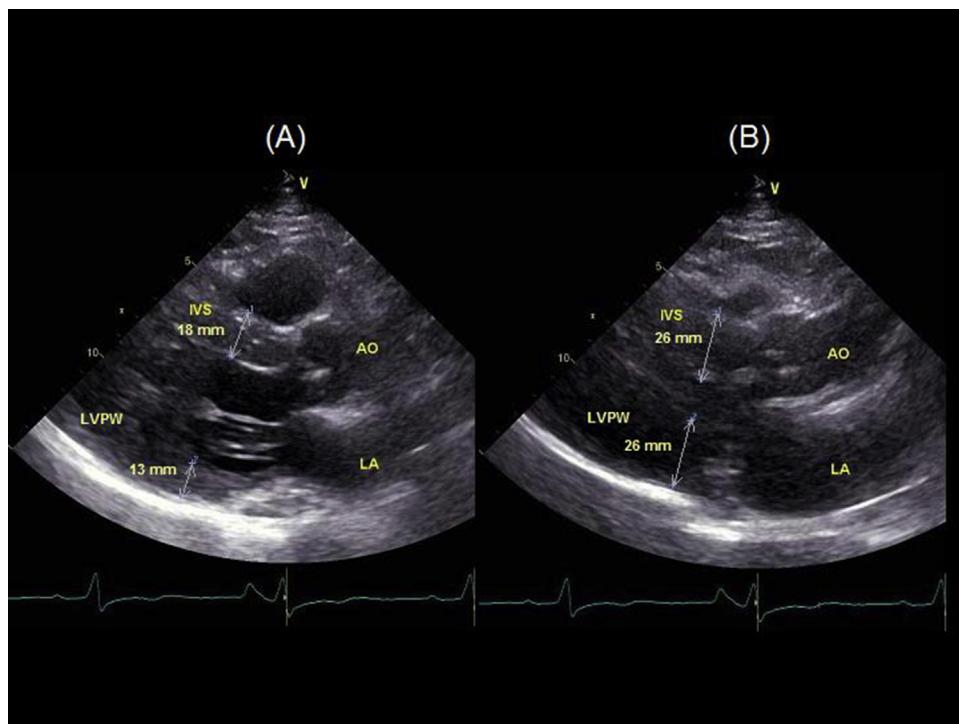
Figure 4 Parasternal long-axis views during end-diastole **(A)** and end-systole **(B)**, which show that the end-diastolic thicknesses of the interventricular septum and posterior wall at the base were 18 and 13 mm, respectively.
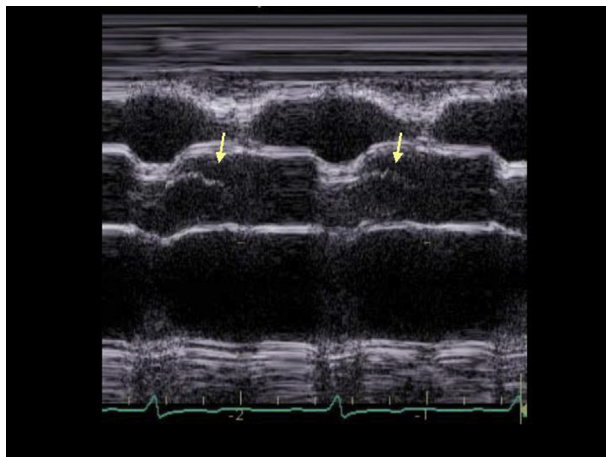
Figure 5 M-mode echocardiogram across the aortic valve demonstrating systolic half closure of aortic valve (*arrows*).
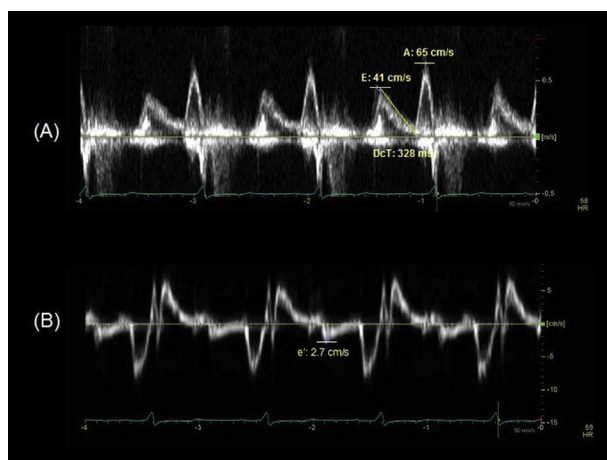
Figure 6 **(A)** Pulsed Doppler echocardiography at the mitral inflow showed an E wave of 41 cm/sec, an A wave of 65 cm/sec, and E-wave deceleration time of 328 msec. **(B)** Doppler tissue echocardiography at the septal annulus showing an e′ wave of 2.7 cm/sec and an E/e′ ratio of 15.2.

base (Figure 9D), although its clinical significance was unclear. Color M-mode echocardiography showed midventricular obstruction with flow, which continued to the end of isovolumic relaxation time (IVRT; Figure 10). Pulsed Doppler echocardiography indicated that the peak velocities of midsystolic flow and isovolumic relaxation flow were 2.3 and 1.8 m/sec, respectively (Figure 11).

Longitudinal strain obtained from speckle-tracking of an apical four-chamber image exposed delayed peak contraction in each segment, after closure of the aortic valve (Figure 12). Peak contraction of global longitudinal strain also occurred after aortic valve closure. Cardiac magnetic resonance imaging showed that there was no LV outflow tract obstruction or apical aneurysm (Figure 13, Video 6). Thus, paradoxical jet flow was due to the coexistence of midchamber obliteration and segmental wall motion abnormalities of the apex.

The patient had undergone coronary angiography 1 year prior, revealing no organic stenosis (Figure 14). The hemodynamic study revealed a mean pulmonary artery wedge pressure of 22 mm Hg, LV systolic/diastolic (end-diastolic) pressures of 159/7 (27) mm Hg, aortic systolic/diastolic (mean) pressures of 161/94 (121) mm Hg, cardiac output of 5.8 L/min, and a cardiac index of 3.1 L/min/m^2.

The patient was treated with β-blockers and successfully underwent nasal surgery.

DISCUSSION

In the present case of hypertrophic cardiomyopathy, M-mode echocardiography displayed midsystolic half closure of the aortic valve,
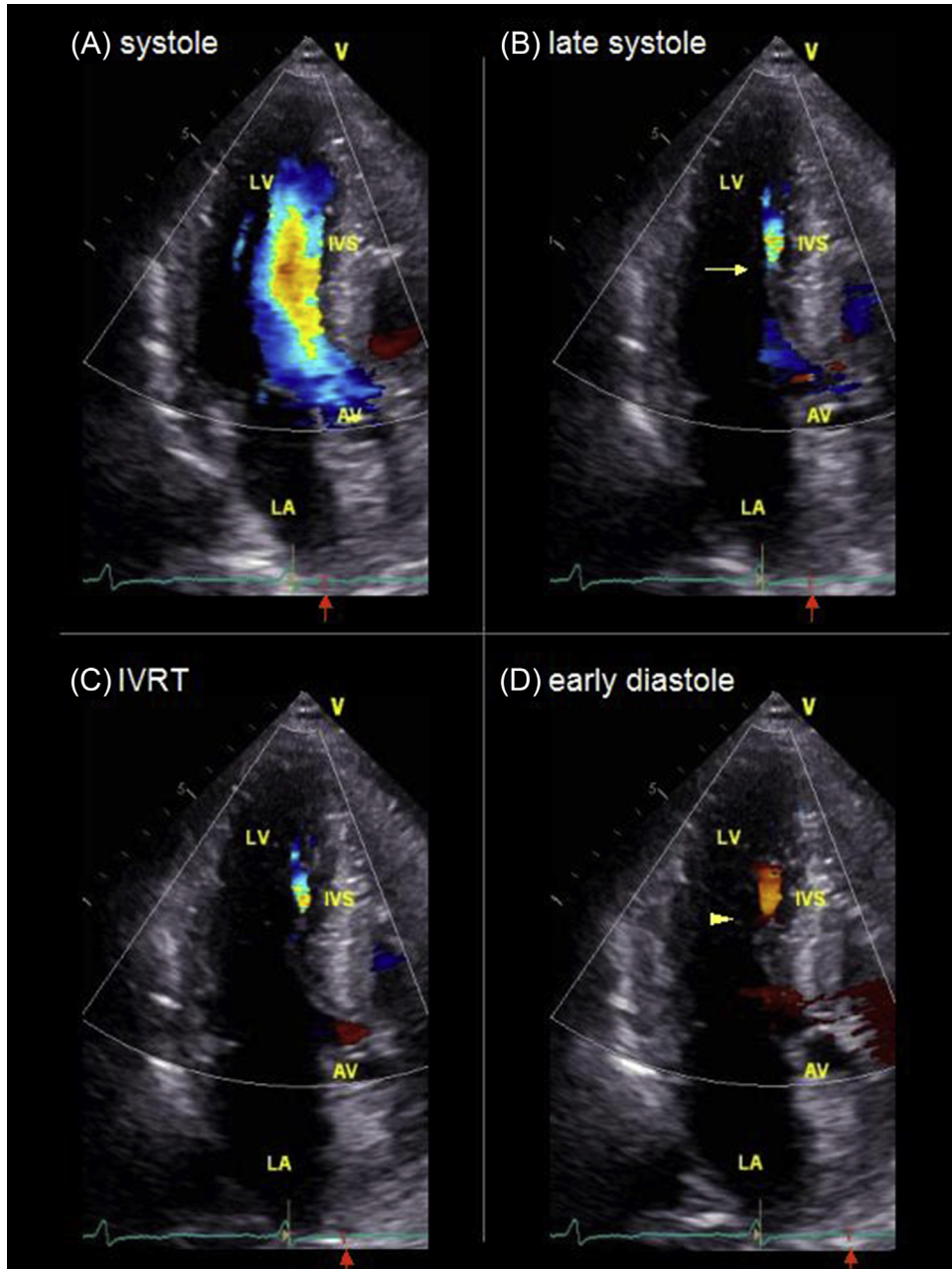
Figure 7 Color flow apical long-axis view during systole **(A)**, late systole **(B)**, IVRT (*arrow* indicates the obstruction site) **(C)**, and early diastole **(D)**. There was midventricular obstruction during midsystole **(B)**. Moreover, isovolumic relaxation flow directed toward the base was present **(C)**. At early diastole, a flow directed to the apex emerged **(D**, *arrowhead***)**.

suggesting LV outflow tract obstruction.[4] Two-dimensional and color flow Doppler imaging revealed a hypertrophied ventricular septum and anteriorly oriented papillary muscle causing midventricular obstruction. Moreover, color M-mode echocardiography showed isovolumic relaxation flow directed from the apex toward the base, mimicking paradoxical jet flow.

Paradoxical jet flow is characterized by midventricular early diastolic flow directed from the apex to the base of the left ventricle. Although its mechanisms are not fully understood, simultaneous measurements of pressure at the LV apex and base have provided vital information.[5] During IVRT, apical pressure is significantly higher than basal pressure because of a delay or an absence of active relaxation at the apex. This pressure gradient is the cause of paradoxical jet flow.

However, in the present case, there were some inconsistent findings of paradoxical jet flow. In patients with paradoxical jet flow, color M-mode echocardiography usually shows high forward or vanished flow due to complete cavity obliteration.[6]
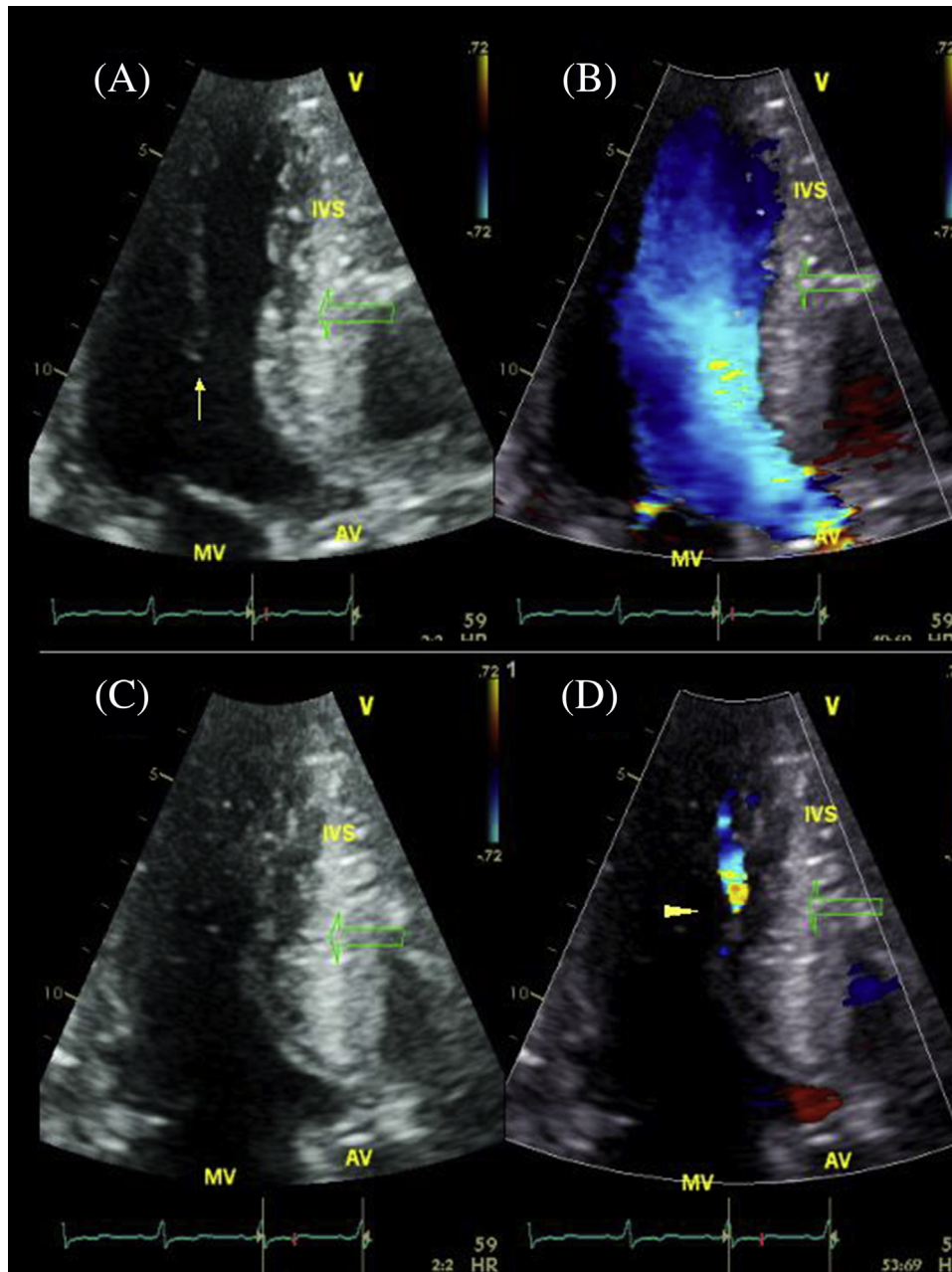
Figure 8 Magnified simultaneous images of apical long-axis views at systole **(A, B)** and IVRT **(C, D)**. Apposition of the septum and anteriorly oriented papillary muscle caused midventricular obstruction (*arrow*). Isovolumic relaxation flow was observed at the obstruction (*arrowhead*).

During diastole, paradoxical jet flow appears from IVRT and continues after the start of early mitral inflow. Thus, color flow imaging may simultaneously demonstrate two opposite flow patterns: paradoxical jet flow and early diastolic mitral inflow.[1] Moreover, paradoxical jet flow may occur because of the coexistence of midchamber obliteration and segmental wall motion abnormalities of the apex.[1] In the present case, systolic flow was low, and isovolumic relaxation flow disappeared before the beginning of early mitral inflow. Moreover, cardiac magnetic resonance imaging showed no abnormality at the apex. Color flow imaging at the apical region suggested the mechanism of systolic low flow at the obstruction. Because the LV cavity was small and there was additional occlusion at the more apical region, flow volume passing through the obstruction might have been extremely small, leading to a high-pressure gradient.

Longitudinal strain on speckle-tracking revealed delayed LV peak contraction in each segment after closure of the aortic
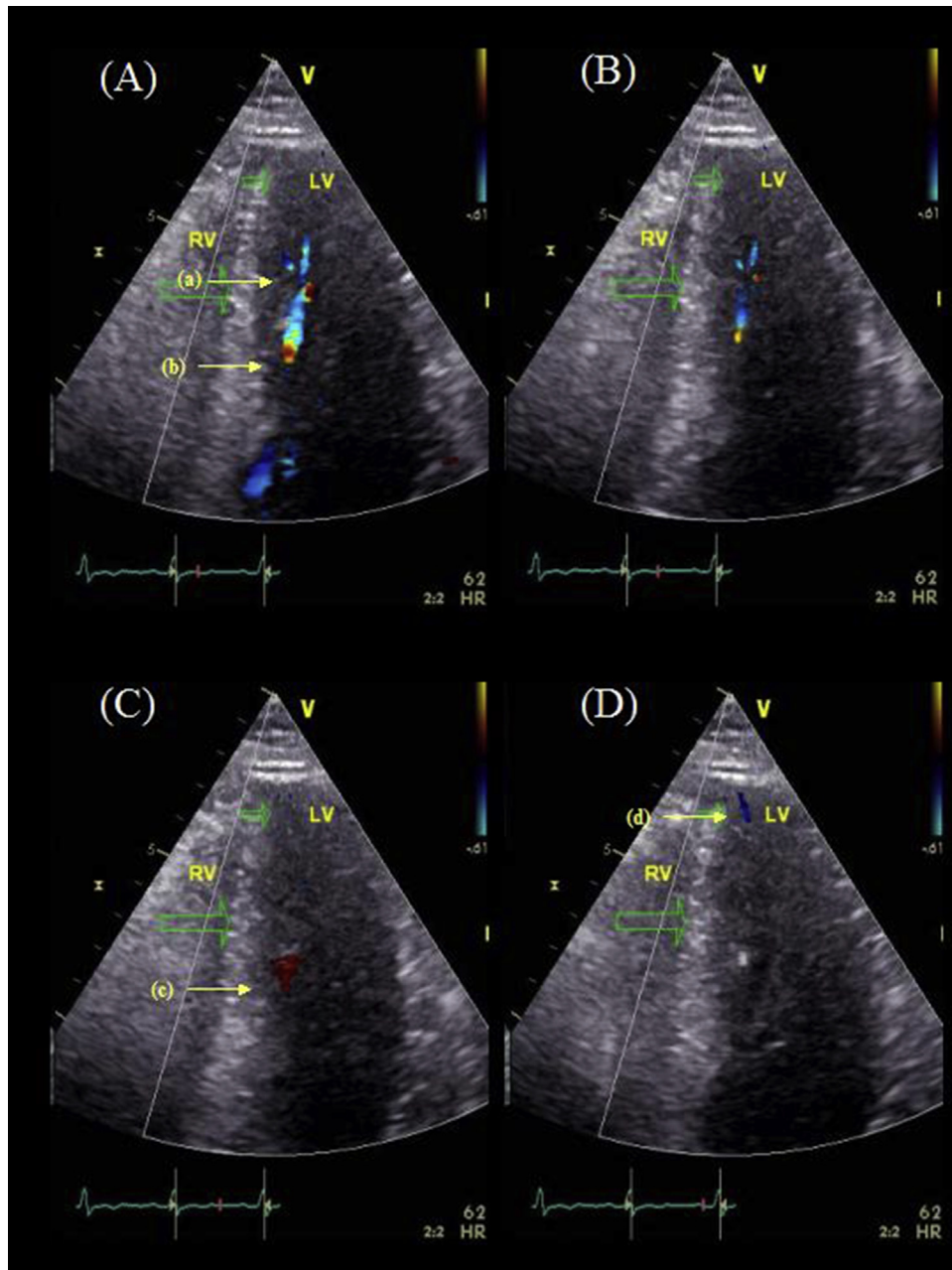
Figure 9 Color flow imaging at the apical region during late systole **(A)**, IVRT **(B)**, early diastole **(C)**, and diastole **(D)**. Small LV cavity and additional obstruction were present during systole **(a, b)**. During IVRT, isovolumic relaxation flow was observed. During early diastole, inflow was observed at the midventricular obstruction earlier than mitral inflow **(c)**. During diastole, apical flow toward the base was observed **(d)**.

valve. This is known as postsystolic shortening (PSS)[7] and may represent continued contraction during IVRT. PSS has been reported as a marker of myocardial ischemia and fibrosis and is frequently observed in hypertrophic cardiomyopathy.[8] In the present case, ischemic heart disease was excluded, and hypertrophic cardiomyopathy might have caused PSS. It is possible that the pushing force from PSS might have produced the isovolumic relaxation flow pattern, which may be referred to as "pseudo-paradoxical flow." Color M-mode echocardiography also demonstrated that midventricular obstruction continued from midsystole to IVRT, when mitral inflow was still absent.

It is questionable whether apical aneurysm may develop in the future. In the present case, diastolic flow was observed at the apex. If this flow increases or is obstructed, apical aneurysm may develop. Careful follow-up may be needed.
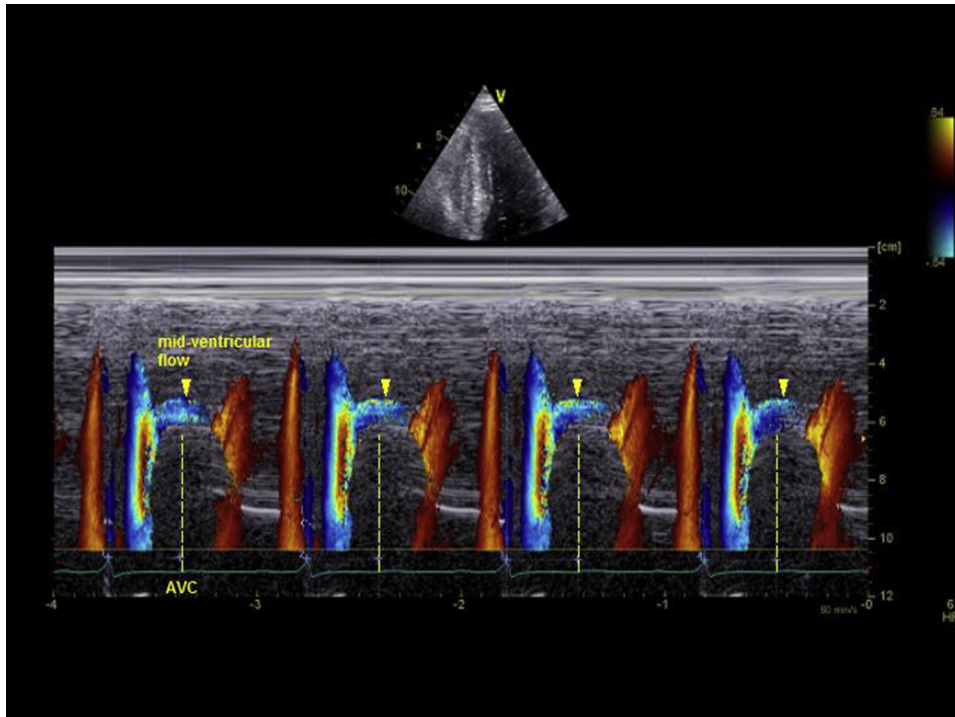
Figure 10 Color M-mode echocardiography showing midsystolic obstruction and midsystolic flow continued until the end of IVRT. Timing of aortic valve closure is indicated by *broken lines*.
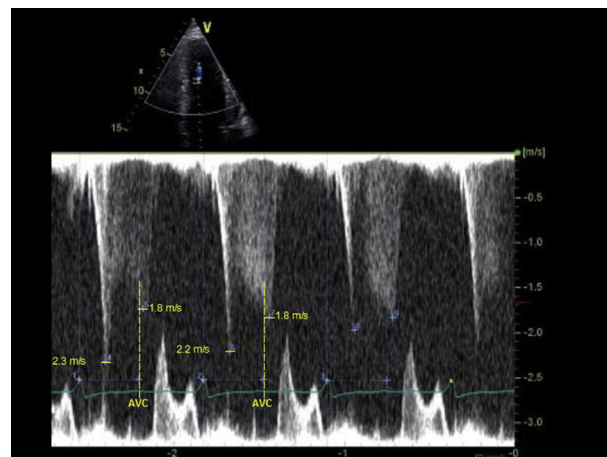

Figure 11 Pulsed Doppler echocardiography indicated that peak velocities of midsystolic flow and isovolumic relaxation flow were 2.3 and 1.8 m/sec, respectively.

CONCLUSION

To our knowledge, this is the first case report of midventricular isovolumic relaxation flow other than paradoxical jet flow. Neither systolic midventricular obstruction nor LV apical aneurysm was present. Longitudinal strain from an apical four-chamber view revealed that PSS caused this mysterious flow during IVRT and may cause isovolumic relaxation flow in some cases. Strain imaging in echocardiography is a simple and reproducible method to detect PSS without significant restriction.
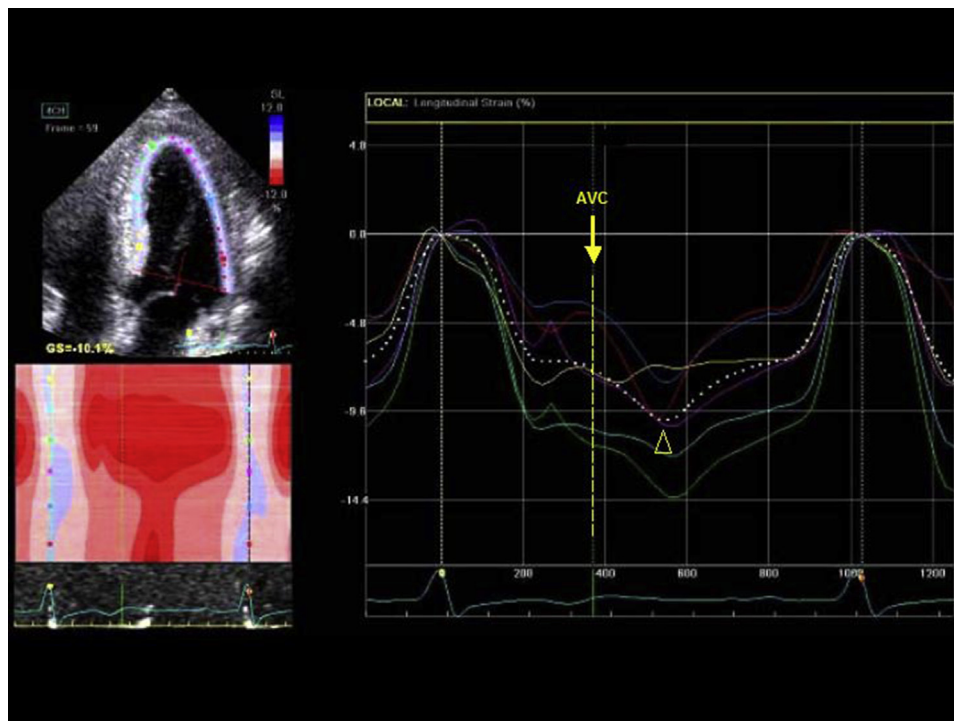
Figure 12 Longitudinal strain obtained from speckle-tracking of an apical four-chamber image showing that peak contraction was reached after the closure of the aortic valve (*dashed line*) in each segment. Peak contraction of global longitudinal strain (*dotted line*) also occurred after aortic valve closure (*arrowhead*).
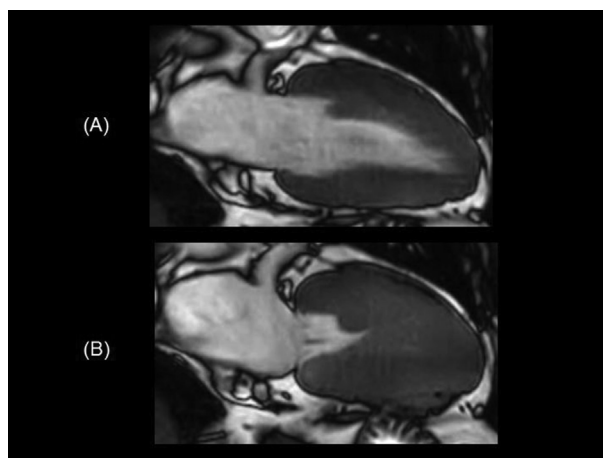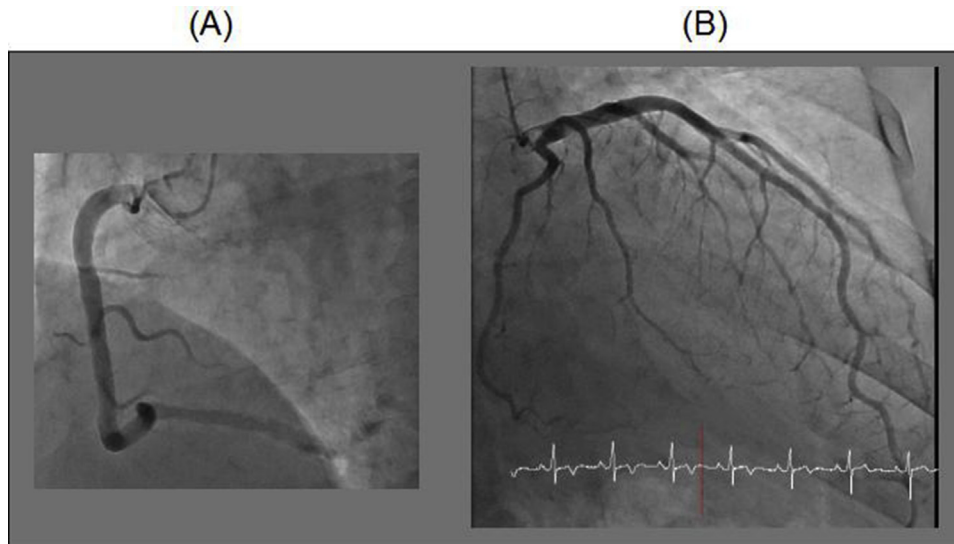

Figure 13 Cardiac magnetic resonance imaging at end-diastole **(A)** and end-systole **(B)** showing massive LV hypertrophy without LV obstruction or apical aneurysm.

Figure 14 Right **(A)** and left **(B)** coronary angiography showing no organic stenosis.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2020.02.003.

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