

“Bundle Squeeze Sign” on Coronary Angiography in Hypertrophic Cardiomyopathy: A Case Report

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Case Report

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Abstract

This article reports an unusual diagnostic course in one patient with hypertrophic cardiomyopathy (HCM). The patient was admitted with chest tightness as the main symptom. Electrocardiography and transthoracic echocardiography (TTE) performed after admission showed no obvious signs of hypertrophic cardiomyopathy. During coronary angiography, we identified a bundle-squeezing phenomenon manifested as systolic compressive occlusion of the septal branch derived from the left anterior descending artery; we termed this finding the “Bundle Squeeze Sign”. Guided by the angiographic findings, cardiac magnetic resonance examination was performed, which confirmed the diagnosis of hypertrophic cardiomyopathy. Accordingly, we hold that identification of the Bundle Squeeze Sign on coronary angiography should raise high clinical alert to the possibility of underlying HCM.

Introduction

Hypertrophic cardiomyopathy (HCM) is defined by left ventricular and/or right ventricular hypertrophy with associated diastolic dysfunction. Its clinical presentation is highly heterogeneous, ranging from asymptomatic states to heart failure, arrhythmias, and sudden cardiac death¹. Despite substantial advances in HCM diagnosis and treatment over recent decades, its complex pathophysiology continues to pose significant challenges in clinical practice. Patients with HCM frequently present with concomitant coronary artery anatomical anomalies or microvascular dysfunction, which may lead to myocardial ischemia, left ventricular outflow tract obstruction, and other complications². We recently encountered a patient diagnosed with HCM who exhibited mechanical compression and occlusion of the septal branch of the left anterior descending (LAD) artery during systole on coronary angiography (CAG). This case report analyzes this phenomenon.

Clinical Data

A 57-year-old man was admitted to our hospital on February 17, 2025, with a chief complaint of intermittent chest tightness for more than one month. He had experienced retrosternal discomfort after physical activity since January 16, 2025. Symptoms resolved with rest, occurred once or twice daily, and were exclusively associated with physical exertion. His medical history was unremarkable.

Upon admission, physical examination revealed a heart rate of 70 beats/min and blood pressure of 128/74 mmHg. The apical impulse was normal in position and amplitude, without heaving or thrills. Cardiac dullness was within normal limits. Heart rhythm was regular, with normal heart sounds of appropriate intensity. No pathological murmurs were detected in any valve auscultation areas, and no pericardial friction rub was observed.

A 12-lead resting electrocardiogram (ECG) revealed extreme right axis deviation, clockwise rotational shift, and ST-T changes (Fig. 1). Complete blood count, liver and renal function tests, coagulation profile,

cardiac enzymes, lipid panel, and electrolytes were all within normal limits. Transthoracic echocardiography (TTE) showed: 1) hypertrophy of the interventricular septum (IVS); 2) mild aortic regurgitation (Fig. 2).

Coronary artery disease (CAD) was initially suspected. CAG, performed on February 22, 2025, revealed a balanced coronary circulation. The left main coronary artery (LMCA) was normal. Myocardial bridging was observed in the distal segment of the LAD artery, causing approximately 80% luminal narrowing during systole, with complete recovery during diastole (TIMI grade 3 flow). The first septal branch exhibited compression during systole and relaxation during diastole, a phenomenon not observed in other vessels. The distal right coronary artery showed atherosclerotic plaques with TIMI grade 3 flow.

Provisional diagnoses following CAG included coronary atherosclerosis, coronary myocardial bridging, and suspected HCM (Fig. 3).

Based on these findings, cardiac magnetic resonance (CMR) imaging was performed. Results revealed: 1) hypertrophy of the mid-to-proximal segments of the left ventricular IVS, consistent with HCM; 2) linear subendocardial hypoperfusion in the basal segment of the left ventricle, suggesting myocardial ischemia; 3) focal myocardial fibrosis in the posterior IVS of the left ventricle (Fig. 4).

The final clinical diagnoses were: 1) HCM; 2) Coronary atherosclerosis; 3) Coronary myocardial bridging.

The patient was treated with aspirin (acetylsalicylic acid, ASP) 100 mg orally (po) once daily (qd) for antiplatelet aggregation, atorvastatin 10 mg po qd for lipid-lowering and plaque stabilization, and metoprolol 25 mg po qd to reduce myocardial contractility and heart rate. His condition improved, and he was discharged in stable condition. After discharge, he continued regular oral medications and attended outpatient follow-up regularly. After one year of treatment, the patient's chest tightness has completely resolved without symptom recurrence..

Discussion

HCM is characterized by left ventricular and/or right ventricular hypertrophy with diastolic dysfunction. Its prevalence is estimated to be approximately 1 in 500 in the general population. About 60–70% of patients with HCM develop left ventricular outflow tract obstruction upon provocation^{3,4}. The clinical presentation of HCM varies widely. In this case, the patient presented with intermittent chest tightness. Traditionally, HCM-related angina has mainly been attributed to myocardial bridging-like compression caused by myocardial hypertrophy and coronary microvascular dysfunction⁵. Recently, advances in imaging technology and molecular biology have gradually clarified the pathophysiological mechanisms underlying HCM-related angina. Particularly in refractory cases, angina can result from coronary microvascular dysfunction even without epicardial coronary stenosis. This mechanism can be detected and quantified by advanced imaging techniques such as positron emission tomography (PET), providing a theoretical basis for microcirculation-targeted therapies including the coronary sinus reducer (CSR)⁶.

In this case, ECG and TTE initially revealed no obvious signs of HCM, and physical examination detected no significant murmurs. However, CAG showed systolic compression of the first septal branch arising from the LAD, a phenomenon not observed in other vessels. This finding suggested interventricular septal hypertrophy associated with HCM. CMR imaging, performed based on this clue, confirmed the diagnosis of HCM. The phenomenon of systolic compression and diastolic relaxation of coronary branches in HCM has rarely been reported previously. We propose the term “bundle squeeze sign” to describe this phenomenon. The underlying mechanism may involve abnormal arrangement and contraction of local myocardial bundles, resulting in impaired blood flow in vessels supplying the hypertrophic area, especially the septal branches. This condition may cause subendocardial ischemia and trigger angina. The mechanism is similar to myocardial bridging but affects small branch vessels rather than large epicardial arteries, making it more insidious.

Coronary compression in HCM patients is closely correlated with the degree of myocardial hypertrophy, rate of disease progression, and severity of clinical symptoms⁴. However, systematic studies on this phenomenon remain limited, and the precise pathophysiological mechanism is unclear. Clinically, attention to the “bundle squeeze sign” in arteries supplying hypertrophic myocardium has important diagnostic implications. Prolonged compression of coronary bundles may accelerate myocardial ischemia and cardiac insufficiency. For patients with HCM without evident early ECG or TTE findings, recognition of the “bundle squeeze sign” on CAG can facilitate early detection and intervention, providing the earliest opportunity to alleviate myocardial hypertrophy⁷. Advances in CMR imaging further support the diagnosis of HCM⁸.

In this case, the patient initially showed no clear evidence of HCM. The “bundle squeeze sign” of the septal branch detected on CAG prompted further examination, leading to a definitive HCM diagnosis through CMR. Clinically, for patients with unexplained chest tightness, chest pain, or dyspnea on exertion, close attention should be paid to changes in septal branches and small coronary branches in the apical region during CAG. Recognition of the “bundle squeeze sign” enables early identification of HCM, allowing timely intervention and reducing the risk of misdiagnosis, underdiagnosis, or delayed diagnosis.

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of the Journal of Medical Case Reports.

Competing interests

All authors declare that they have no competing interests.

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Author Contribution

Author Contributions:Yapeng Ma and Xueyan Zhao were responsible for patient management and drafting of the initial manuscript. Bo Li, Hai Zhu and Jing Zeng conducted image analysis and revised the manuscript. Zhang Fu supervised the research implementation.

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Data availability

No data are associated with this article.

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Figures

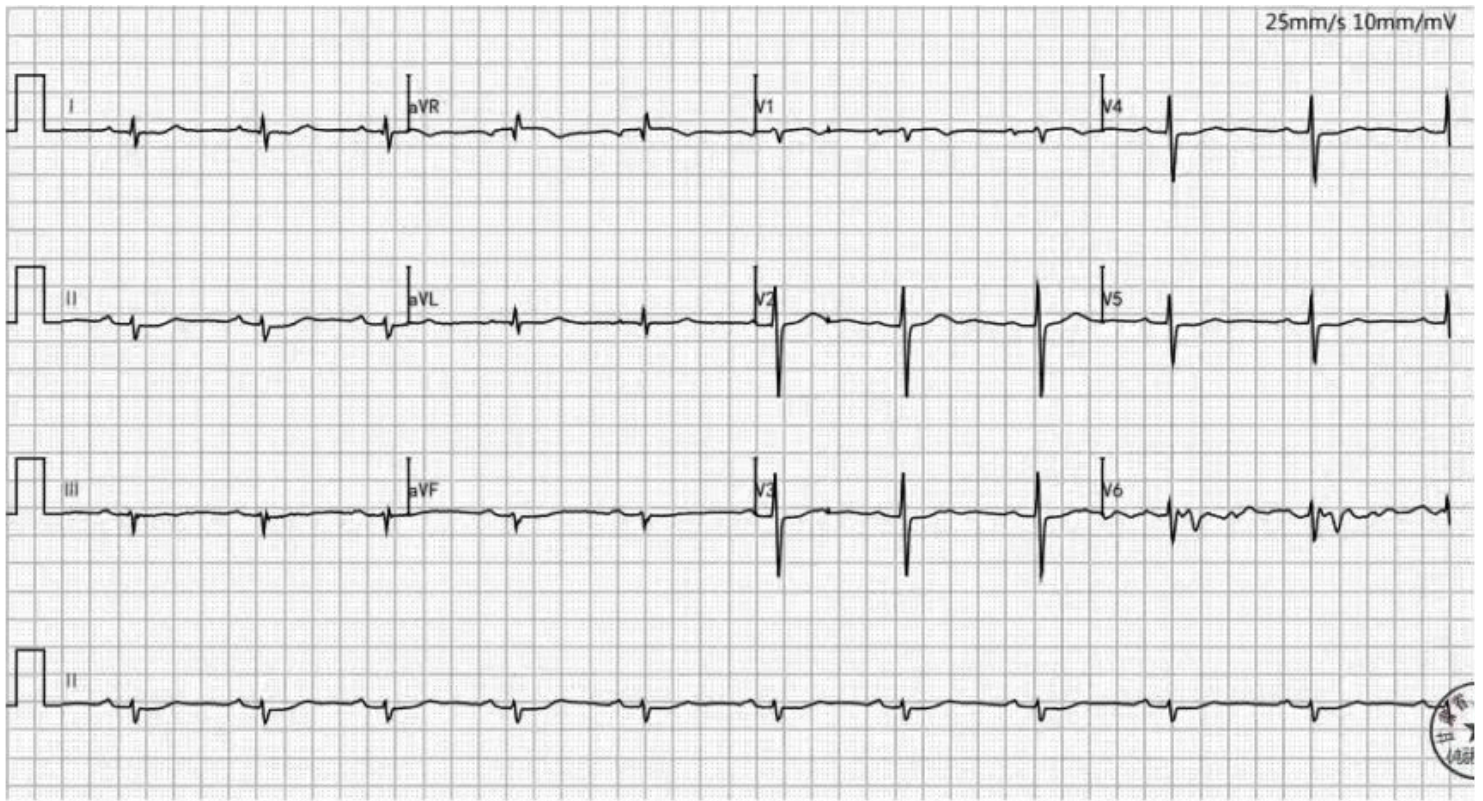


Figure 1

Twelve-lead resting ECG

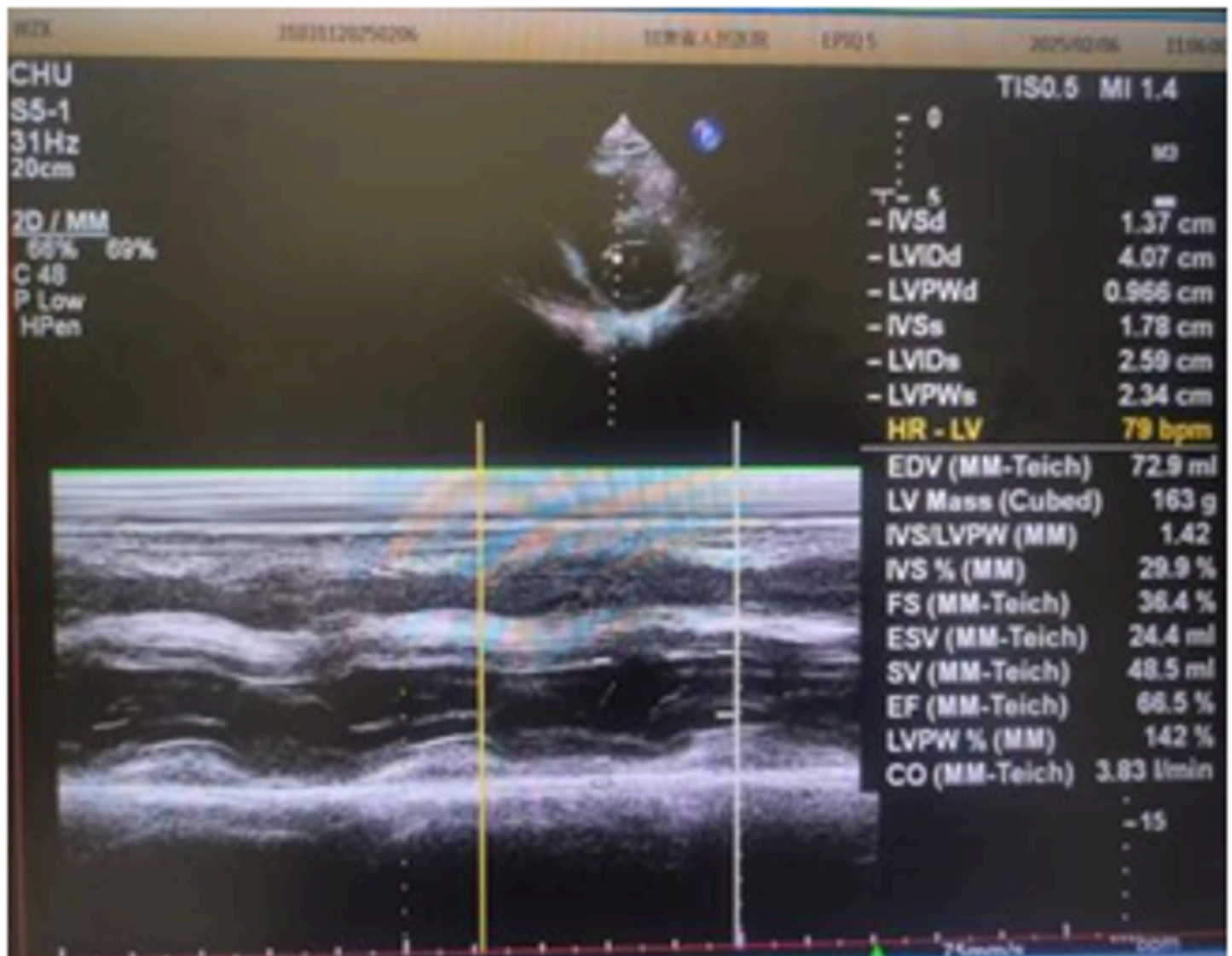


Figure 2

Transthoracic echocardiogram on admission

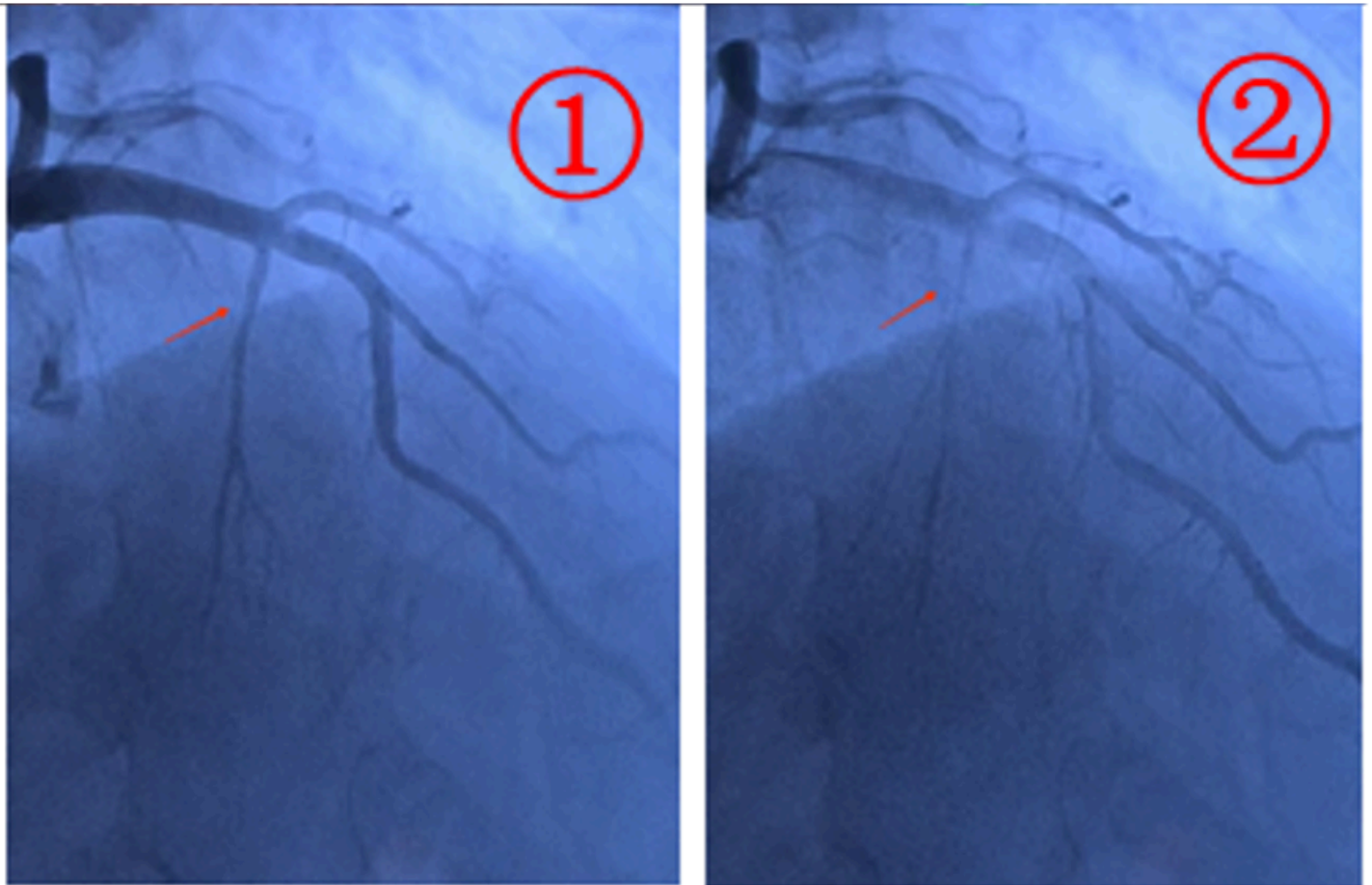


Figure 3

CAG images: (1) The first septal branch of the LAD artery during diastole (arrow); (2) The first septal branch showing compression by adjacent myocardial bundles during systole, demonstrating the “bundle squeeze sign” (arrow)

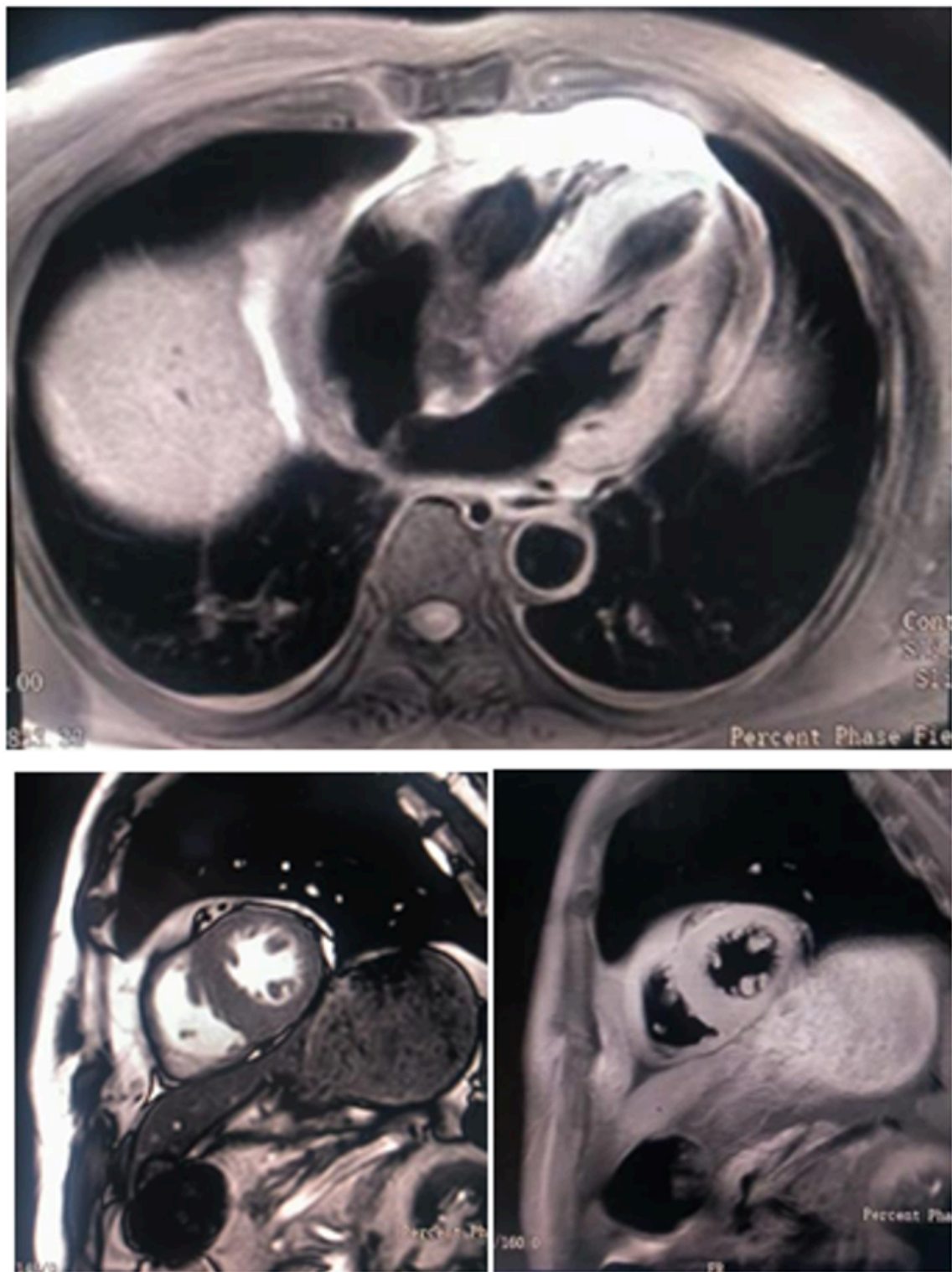


Figure 4

CMR imaging findings

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