

An Unusual Association of Coronary Cameral Fistula and Mid-Cavitary Hypertrophic Cardiomyopathy

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ABSTRACT

The presence of both myocardial hypertrophy and coronary-to-cameral fistula has been reported previously. However, the exact mechanisms are not clear. Herein, we reported a patient with mid-cavitary hypertrophic cardiomyopathy and concomitant connections between coronary arteries and left ventricular cavity.

Keywords: Hypertrophic cardiomyopathy, Coronary fistulae

To the Editor,

A 67-year-old woman with a history of hypertension and hyperlipidemia was admitted to the emergency room with anginal chest pain lasting more than 30 minutes. The physical examination was normal, and the electrocardiogram showed sinus rhythm (72 bpm) and changes compatible with left ventricular (LV) hypertrophy (Figure 1A). Transthoracic echocardiography demonstrated normal systolic functions of the LV with mid-cavitary hypertrophy (max. LV wall thickness of 16 mm) consistent with hypertrophic cardiomyopathy (HCM), with no LV outflow tract gradient and no systolic anterior motion of the mitral valve leaflet. Imaging with color flow doppler demonstrated blood flow from the epicardial surface into the LV cavity through the apical segments of the myocardium during diastole (Figure 1B, Video 1). The two-dimensional (2D) speckle-tracking echocardiography showed a relatively reduced longitudinal systolic function at the LV apex (-14.2%) (Figure 1C). Cardiac biomarker levels were within normal reference limits. During coronary angiography, there was no obstructive atherosclerotic lesion in the epicardial coronary arteries, and several connections were present in between the epicardial coronary arteries and left ventricular chamber (Figures 1D and 1E, Video 2). Beta-blocker therapy was started as anti-ischemic therapy, and the patient was discharged uneventfully. HCM Risk-SCD revealed a low sudden cardiac death risk. The patient was asymptomatic at the 6-month follow-up visit. The association of HCM with coronary cameral fistula was only reported in 39 patients until now. This rare finding implies that genetic changes in HCM may interfere with the abnormal embryological dedifferentiation of those microvessels draining into the LV [1-3]. This letter to the editor also highlights a rare association between mid-cavitary HCM with coronary cameral fistula.

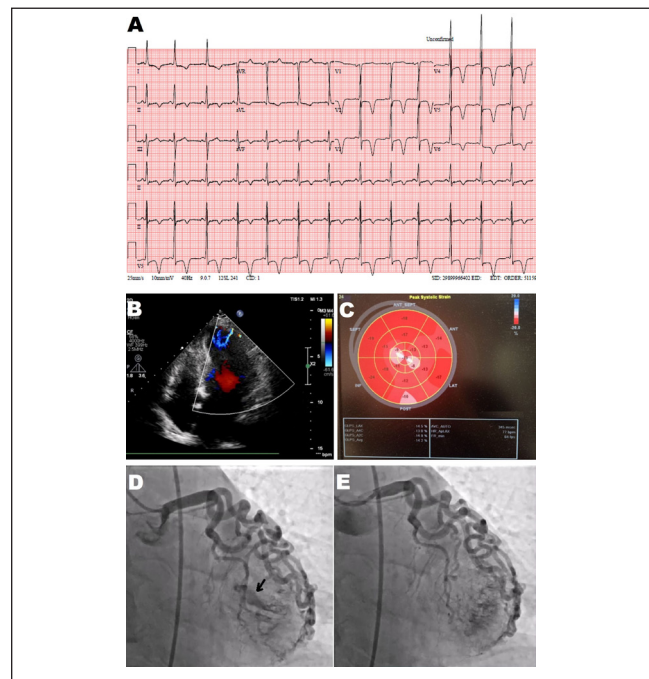


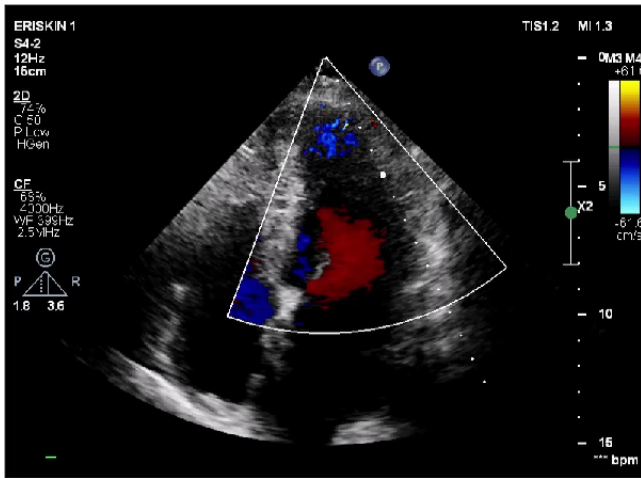
Figure 1. Electrocardiography showing sinus rhythm with voltage criteria and T wave inversion at V2–6, DI–aVL, DII–III–aVF compatible with left ventricular hypertrophy (A). Transthoracic echocardiography demonstrating transmyocardial color flow from the epicardial surface to the endocardial border of apical LV segments (B) and 2D–speckle tracking echocardiography showed a reduced global longitudinal strain at the LV apex (C). Coronary angiography revealed no atherosclerotic lesion and multiple direct communications between the LV cavity and the epicardial coronary arteries (E).

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Supplementary Video 1. Echocardiography showing mid-cavitary hypertrophy and color flow Doppler demonstrating blood flow from the subepicardial to the subendocardial area of the apical segments of the myocardium.



Supplementary Video 2. Coronary angiography showing several connections in between the epicardial coronary arteries and left ventricular chamber.

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