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Case Report

Accessory and solitary main papillary muscle hypertrophy resulting in dynamic mid–left ventricular obstruction: Contribution of multimodality imaging in highlighting of dynamic and structural abnormalities

Iskander Slama (MD)[a,*], Saoussen Antit (MD)[a], Elhem Boussabah (MD)[a], Habib Ben Hajel (MD)[b], Moez Thameur (MD)[a], Lilia Zakhama (MD)[a], Soraya Benyoussef (MD)[a]

[a] Cardiology Department, Security Forces Hospital, La Marsa, Tunisia
[b] Radiology Department, Berges du Lac Clinic, La Marsa, Tunisia

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ABSTRACT

Solitary main and/or accessory papillary muscle (PM) hypertrophy may be an uncommon type of hypertrophic cardiomyopathy that does not meet all the usual criteria. The dynamic intraventricular obstruction related to this PM hypertrophy can be deleterious with an important clinical impact on patients. The mechanisms of such obstruction attracted a lot of attention in order to propose an appropriate treatment. We report a case of a 36-year-old man presenting with a chief complaint of progressively worsening exertional dyspnea. He had demonstrated labile systolic murmur for more than 3 years. Rest echocardiography revealed coexistence of a solitary main PM hypertrophy and additional accessory PM with no left ventricular outflow tract or mid ventricle obstructions. The patient underwent exercise stress echocardiography unmasking severe mid-ventricle obstruction (peak systolic gradient at exercise of 100 mmHg). There was no obvious parietal hypertrophy elsewhere. Cardiac magnetic resonance imaging provided us with anatomical arguments that could explain the dynamic process of obstruction. In fact, multimodality imaging has a determinant role in the screening of spatial configuration and structural abnormalities of PMs in order to avoid the misinterpreting of some atypical presentations of hypertrophic cardiomyopathy.

<**Learning objective:** Solitary main and/or accessory papillary muscle (PM) hypertrophy is an uncommon type of hypertrophic cardiomyopathy: hypertrophy concerns only the PM and spares the other parietal left ventricle segments. Multimodality imaging has a crucial role in the characterization of PM morphology and their incrimination in the pathological process of dynamic obstruction. Surgical treatment depends on the concerned PM, its extent to the mitral leaflet or the septum, or both, and the severity of hypertrophy.>

Introduction

In hypertrophic obstructive cardiomyopathy (HOCM), dynamic left ventricular outflow tract (LVOT) obstruction is commonly inherent to asymmetrical septal hypertrophy with dynamic systolic anterior motion (SAM) of mitral leaflets [1]. Mid-ventricular obstruction has also been described in some forms of HOCM and is frequently occasioned by the anomalous insertion of a main papillary muscle (PM) directly into the anterior mitral leaflet resulting in narrow apposition between the PM and anterior septal muscle [2]. Solitary PM hypertrophy (main or accessory) could be a particular form of hypertrophic cardiomyopathy: hypertrophy concerns only the PM and spares the other parietal left ventricle (LV) segments [3]. This entity has recently drawn attention owing to the LVOT or mid-ventricular obstructions it might cause.

* Corresponding author at: Cardiology Department, Security Forces Hospital, La Marsa, 2070, Tunisia.
E-mail address: slamaiskander@live.fr (I. Slama).

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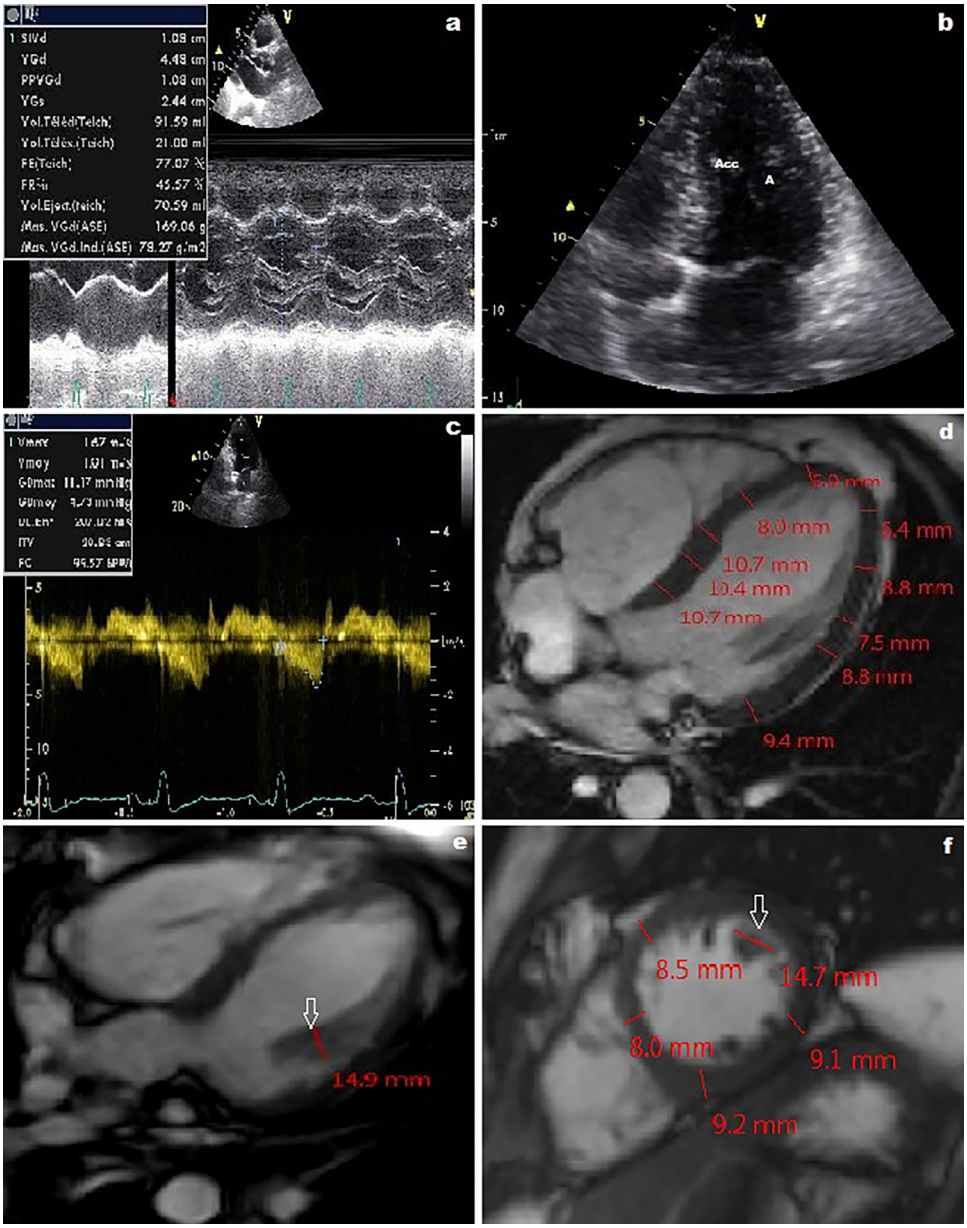

Fig. 1. Rest transthoracic 2D echocardiography. (a) M mode showing no obvious left ventricle (LV) parietal hypertrophy with a LV mass index = 79.3 g/m^2, echo dense muscular strip shadowing in LV cavity corresponding to the anterolateral (A) papillary muscle (PM). (b) Apical four-chamber view revealing an accessory (Acc) PM attached to the septum and a hypertrophied A PM. (c) Continuous wave Doppler showing no significant dynamic intra-ventricular obstruction. (d) Cine sequences of cardiac magnetic resonance imaging (CINE CMR), end-diastole four-chamber view demonstrating unremarkable LV wall thickness. (e) CINE CMR, end-diastole four-chamber view showing a hypertrophied anterolateral PM to 14.9 mm (white arrow). (f) CINE CMR, end-diastole mid-ventricular short-axis view demonstrating unremarkable LV wall thickness (8–10 mm) with hypertrophied anterolateral PM (white arrow).

Case report

We report a case of a 36-year-old man who presented with a chief complaint of progressively worsening dyspnea. He had demonstrated labile mid-systolic ejection murmur along the left sternal border for more than 3 years. Complete electrocardiogram disclosed a sinus rhythm with normal QT interval, discreet U wave in V2–V3, and no evidence of ventricular hypertrophy. Rest transthoracic echocardiography (TTE) revealed solitary anterolateral PM hypertrophy (a muscular strip making 14 mm of thickness), an accessory PM (bulbous in morphology and attached to the septum) and no obvious parietal hypertrophy elsewhere (Fig. 1a,b, see Supplementary Video S1 in the online version at DOI: 10.1016/j.jccase.2018.05.009). There were no significant LVOT or mid LV obstructions (Fig. 1c, see Supplementary Video S2 in the online version at DOI: 10.1016/j.jccase.2018.05.009). The LV end-diastolic and end-systolic volumes were in normal ranges with preserved LV ejection fraction and LV end-diastolic pressure was normal. Neither aortic stenosis nor SAM was observed. No mitral valve structural abnormalities were found except trivial mitral regurgitation. The patient underwent exercise stress echocardiography unmasking severe mid-wall obstruction revealed by an important aliasing and a phenomenal flow convergence zone on color Doppler with late-peaking dagger-shaped appearance on continuous Doppler (peak systolic gradient at exercise of 100 mmHg) (Fig. 2a–c,e–f, see Supplementary Videos S3–S5 in the online version at DOI: 10.1016/j.jccase.2018.05.009). Mapping of the LVOT with pulse Doppler showed slightly accelerated

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