Journal of Cardiology Cases 11 (2015) 69–72

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Left ventricular outflow tract obstruction with abnormal papillary muscles

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ARTICLE INFO

Article history:
Received 25 August 2014
Received in revised form 19 October 2014
Accepted 22 October 2014

Keywords:
Left ventricular outflow tract obstruction
Abnormal papillary muscle
Cardiac magnetic resonance imaging

ABSTRACT

A 65-year-old man with a history of hypertension was admitted to our hospital with fainting and syncope. He had experienced recurrent syncope since 20 years of age. On admission, systolic heart murmur was audible at the apex of the heart. Echocardiography revealed anteriorly displaced papillary muscles (PMs), elongation of the anterior mitral valve leaflet (AML), and systolic anterior motion (SAM) of the AML. Color Doppler imaging showed accelerated flow with a pressure gradient (PG) of 56 mmHg at the left ventricular outflow tract (LVOT). Cardiac magnetic resonance imaging revealed mild asymmetric septal hypertrophy and multiple accessory PMs. Cine images clearly demonstrated SAM and LVOT obstruction due to anteriorly displaced PMs. Based on these findings, the patient was diagnosed as having hypertrophic cardiomyopathy and LVOT obstruction due to abnormal PMs. Oral administration of bisoprolol (2.5 mg/day) was initiated, because the patient rejected surgical treatment. Follow-up echocardiography revealed a gradual decrease in the LVOT-PG to 24 mmHg, and no episodes of fainting or syncope have recurred for 2 years after the initiation of bisoprolol.

<**Learning objective:** Abnormal papillary muscle (PM) is an unusual cause of left ventricular outflow tract (LVOT) obstruction, and cardiac magnetic resonance (CMR) imaging has been reported to be useful for diagnosis of abnormal PM. Abnormal PMs with LVOT obstruction are usually treated by surgical correction, and therefore, reports on medical treatment are limited. We report a case of LVOT obstruction due to abnormal PMs, which was accurately diagnosed by CMR imaging and successfully treated with a beta-blocker.>

Introduction

Abnormal papillary muscle (PM) is an unusual cause of left ventricular outflow tract (LVOT) obstruction [1], and is often found in patients with hypertrophic cardiomyopathy (HCM) [2]. Cardiac magnetic resonance (CMR) imaging has been reported to be useful for diagnosis of abnormalities of the PM and mitral valve in HCM patients [3–5]. Abnormal PMs with LVOT obstruction are usually treated by surgical correction [6,7], and therefore, reports on medical treatment are limited [8]. In the present report, we describe the case of an HCM patient associated with LVOT obstruction due to abnormal PMs, which was accurately diagnosed by CMR imaging and successfully treated with a beta-blocker.

Case report

A 65-year-old man with a history of hypertension was admitted to our hospital with fainting and syncope. The patient had experienced recurrent syncope since 20 years of age, and was diagnosed with epilepsy at 49 years of age. Treatment with anticonvulsants was soon discontinued because of skin rash. He had been taking amlodipine (5 mg/day) for hypertension. On admission, his blood pressure was 158/93 mmHg, pulse rate was 61 beats per minute, and systolic heart murmur of grade 4/6 was audible at the apex of the heart. Chest radiography showed mild cardiomegaly, and electrocardiography showed high voltage and mild ST-segment depression. Transthoracic echocardiography

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http://dx.doi.org/10.1016/j.jccase.2014.10.009
1878-5409/

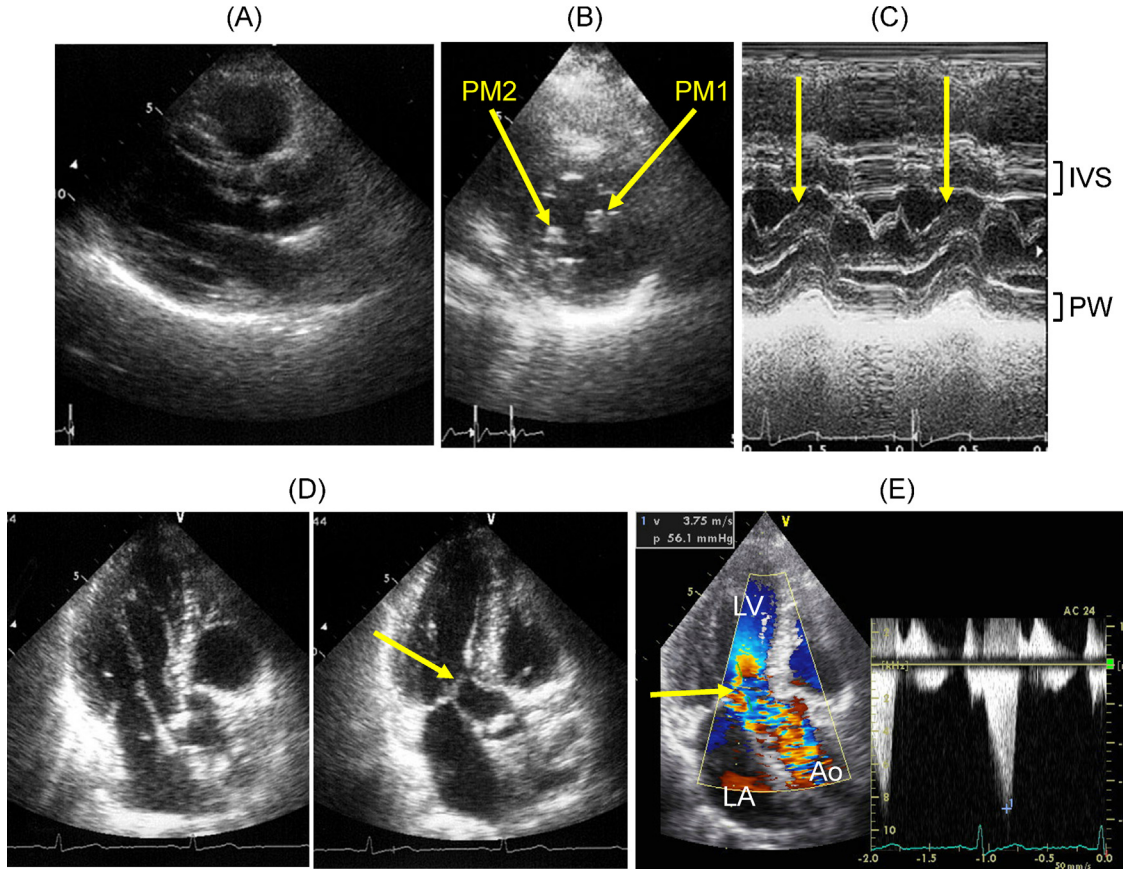

Fig. 1. Echocardiography on admission. (A) Parasternal long-axis view. Left ventricular hypertrophy was not apparent. (B) Parasternal short-axis view of the mid-left ventricle showing two anteriorly displaced papillary muscles (PM1 and PM2) (yellow arrows). (C) M-mode recording at the mitral valve level showing systolic anterior motion of the anterior mitral valve leaflet (AML) (yellow arrows). (D) Apical long-axis view showing elongation of the AML and protrusion of the AML into the left ventricular outflow tract (LVOT) (yellow arrow). (E) Color flow imaging showing accelerated flow at the LVOT (yellow arrow). The LVOT pressure gradient, measured by continuous wave Doppler, was 56 mmHg. IVS, interventricular septum; PW, posterior wall; LV, left ventricle; LA, left atrium; Ao, aorta.

revealed normal left ventricular wall thickness (septal wall: 12 mm, posterior wall: 10 mm, septal/posterior wall thickness ratio: 1.2), dimension (end-diastolic: 41 mm, end-systolic: 22 mm), and contractility (ejection fraction: 77%) (Fig. 1A). Parasternal short-axis views of the left ventricle showed two anteriorly displaced PMs (Fig. 1B). M-mode recording at the mitral valve level showed systolic anterior motion (SAM) of the anterior mitral valve leaflet (AML) (Fig. 1C). Apical long-axis views showed elongation of the AML and protrusion of the AML into the LVOT (Fig. 1D). Color flow imaging showed accelerated flow at the LVOT and continuous wave Doppler recorded a pressure gradient (PG) of 56 mmHg (Fig. 1E). CMR imaging revealed mild asymmetric septal hypertrophy (anterior septal wall: 14.1 mm, posterior wall: 6.9 mm) which was not apparent on echocardiography, and confirmed the presence of multiple accessory PMs, some of which were displaced anteriorly (Fig. 2A). Cine images clearly displayed SAM and LVOT obstruction due to anteriorly displaced PM (Fig. 2B–E). Left ventricular pressure measured at the apex of left ventricle and LVOT showed a PG of 40 mmHg (Fig. 3). Coronary angiography showed no significant stenosis. Based on these findings, the patient was diagnosed as having HCM and LVOT obstruction due to abnormal PMs. Oral administration of bisoprolol (2.5 mg/day) was initiated, because the patient rejected surgical treatment. The daily dose of bisoprolol was maintained at 2.5 mg because of bradycardia. Follow-up echocardiography performed 6 months and 2 years after the initiation of bisoprolol revealed a gradual decrease in the LVOT-PG to 42 and 24 mmHg, respectively. The systolic heart murmur also decreased to grade 2/6. In addition, no episodes of fainting or syncope recurred for 2 years after the initiation of bisoprolol.

Discussion

Hypertrophic obstructive cardiomyopathy is a common cause of LVOT obstruction, whereas abnormal PM, although unusual, has been reported to cause LVOT obstruction [1]. Roberts and Cohen classified abnormal PM morphology into the following groups: single PM, accessory PMs, abnormally large (or small) and malpositioned PM, and insertion of the PM directly into the mitral leaflet [9]. In the present case, the number of PMs was high, and a few of these PMs were anteriorly displaced; thus, the patient was diagnosed as having accessory PMs and malpositioned PMs. Although left ventricular hypertrophy was not apparent on echocardiography, CMR imaging revealed mild asymmetric septal hypertrophy; thus, the patient was diagnosed as having HCM. Previous studies reported that abnormalities of the PM and mitral valve were often found in patients with HCM [2], and CMR was useful for diagnosis of these abnormalities [3–5]. Harrigan et al. evaluated PM morphology in 201 patients with HCM using CMR imaging, and indicated that PM number and mass were increased in HCM patients, and anteriorly displaced PMs were associated with LVOT obstruction [3]. Furthermore, Kwon et al. assessed the relationship between abnormal PM morphologies and LVOT obstruction in 56 patients with HCM using CMR, and noted that patients with anteroapically displaced PMs had higher LVOT-PG,