Case Report

Mid-ventricular hypertrophic obstructive cardiomyopathy (MVHOCM) complicated with coronary artery disease: a case report

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ABSTRACT  Mid-ventricular hypertrophic obstructive cardiomyopathy (MVHOCM) is a rare type of cardiomyopathy that can be accompanied by apical aneurysm. We presented here a case report of MVHOCM with coronary artery disease. The sixty-four years old man was sent to hospital because of ventricular tachycardia. Large inversion T wave was showed on electrocardiography in the presence of abnormal coronary arteries and normal cardiac enzyme. Echocardiography showed an hourglass appearance of the left ventricle with an aneurysm in the apex and a pressure gradient between the outflow tract of left ventricle and the middle of the left ventricle was revealed by left-heart catheterization. (J Geriatr Cardiol 2008; 5:190-192)

Key words  hypertrophic obstructive cardiomyopathy; coronary artery disease; echocardiography

Mid-ventricular hypertrophic obstructive cardiomyopathy (MVHOCM) is a rare variant type of hypertrophic obstructive cardiomyopathy, which was first reported by Falicov et al in 1976 and is characterized by high pressure in the apical chamber and none-obstructive left ventricular outflow. Apical aneurysm formation can be found in some cases and significant pressure gradient exists between the apical and basal sites of the left ventricle. Here we present a case report of a patient with MVHOCM combined with coronary artery disease.

A 64-year-old man with hypertension and smoking history was admitted to our coronary care unit for ventricular tachycardia. No family history of cardiomyopathy was mentioned. Just one week before his hospitalization, β-blockers were prescribed to control his occasional episodes of amnesia and palpitation. The initial physical examination showed, BP 140/90mmHg, HR 60 bpm, no cardiac murmur. No cardiomegaly or pulmonary congestion was found by chest X-ray. The 12-lead electrocardiogram showed large negative T wave in lead V2-V6 (0.5-1.0mV) with no ST-segment elevation (Fig 1). Echocardiogram showed normal left ventricular diameter (45mm at end-diastolic diameter, 30mm at end-systolic diameter), marked left atrial dilatation (51mm), significant mid-ventricular hypertrophy (interventricular septum of 20mm/ left ventricular posterior wall of 9 mm; ratio 2.22:1) and an apical aneurysm (Fig 2). There was no evidence of systolic anterior motion of the mitral valve in the echocardiogram. Ejection fraction was 63%.

Laboratory examination displayed normal levels of creatine kinase-MB and troponin I. To confirm the diagnosis of acute coronary syndrome and possible underlying MVHOCM, coronary angiography and left ventriculography were performed. The results of coronary angiography showed 70-75% regional stenosis in the middle of the left anterior descending artery. Left ventriculography revealed an apical aneurysm formation and a distinct pressure gradient of 15mmHg between the outflow tract of the left ventricle and the middle of the left ventricle (152/24mmHg at the middle of the left ventricle, 137/22mmHg at the outflow tract of left ventricle) (Fig 4). β-Blocker was prescribed for the patient at discharge. Consideration of other therapies such as implantable cardioverter defibrillator (ICD) implantation was rejected by patient.

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Mid-ventricular obstructive hypertrophy cardiomyopathy (MVOHCM), a rare subtype of hypertrophic cardiomyopathies (HCM) accounting for only one percent of HCM, is characterized by the presence of a pressure gradient between the apical and basal chambers of the left ventricle. It is frequently associated with an apical aneurysm even in patients without significant atherosclerotic coronary artery disease. Although this patient has several risk factors of coronary artery disease, such as hypertension and smoking history, the cause of HCM may be a hereditary myocardial disorder.

Frequent complications of life-threatening ventricular arrhythmias and sudden death make the management of MVOHCM difficult. Though beta-blockers are generally prescribed as the first choice of treatment for patients with subaortic obstructive hypertrophic cardiomyopathy, the optimal treatment for MVOHCM has not yet been established. Dual-chamber pacing and percutaneous myocardial ablation have been proposed as non-surgical treatments, but their long-term prognosis and procedural safety need be further observed in a large patient population.

References
