Case study

HYPERTROPHIC CARDIOMYOPATHY MIMICKING ACUTE ANTERIOR MYOCARDIAL INFARCTION : CASE REPORT

Abstract

We report a case of 57 years old man, with history of smoking, presented to the emergency department with chest pain and electrocardiographic findings of a ST elevation in the precordial leads from V1 to V4. He was treated as an acute ST-elevation myocardial infarction and transferred to our catheter lab. The coronary angiography did not reveal any coronary lesion. Transthoracic echocardiography, and cardiac magnetic resonance imaging were consistent with hypertrophic cardiomyopathy.

Keywords: Acute myocardial infarction , Hypertrophic cardiomyopathy, ST segment elevation.

INTRODUCTION:

Hypertrophic cardiomyopathy (HCM) is the most common hereditary cardiac disease and the most frequently found cardiomyopathy [1]. It is caused by various genetic mutations that affect the structure, function and organization of heart muscle cells. HCM reflects a large nosological framework. It can be "hidden" by various clinical manifestations. Among these masks, there is a clinically painful form whose symptoms learn to stimulate acute coronary syndrome.

CASE REPORT:

We report a case of a 57 years-old man, who was admitted to the emergency for a constrictive, retrosternal chest pain. Past medical history was non contributory except for smoking

The patient’s vital signs included the following: blood pressure was 130/80 mm Hg, resting heart rate was 120 beats/min, respiratory rate was

16 breaths/min, oxygen saturation was 98%, and temperature was 37.0°C.

Cardiac auscultation revealed a 4/6 systolic murmur over the apex.

There were no congested neck veins. Neither lower limb edema nor signs of pulmonary congestion were observed.

The initial ECG revealed a ST elevation in the precordial leads from V1 to V4 (Figure 1). The initial diagnosis of acute coronary syndrome (ST elevation myocardial infarction) was established.

After initiating treatment by Aspirin (300 mg), Clopidogrel (300 mg) and intravenous heparin, the patient was immediately transferred to our catheter lab.

The coronary angiography did not reveal any coronary lesion.

The echocardiography showed signs of symmetrical parietal hypertrophy with a thickness of up to 17 mm at the side wall (Figure 2), with an intra-cavity obstruction with a peak systolic pressure gradient at 37 mm Hg at rest.

Holter monitor showed predominantly a sinus rhythm with infrequent asymptomatic premature atrial contractions and no ventricular ectopy.

The cardiovascular magnetic resonance (CMR) confirmed the diagnosis of hypertrophic cardiomyopathy (Figure 3).

The patient was discharged on medical treatment including a β-blocker. Upon follow up after 3 months, the patient was asymptomatic, whereas transthoracic echocardiography continued to show the same gradient of 20 mmHg across the left ventricular outflow tract obstruction (LVOT).

DISCUSSION:

The HCM is the most common genetic disease associated with more than 1000 mutations in 11 genes [1], and the most frequent cause of sudden death in young patients. This causes the heart to become abnormally thick, particularly at the septum between the left and right sides of the heart, which may cause obstruction of blood flow from the heart during each contraction. Once diagnosed, appropriate treatment is key to relieving symptoms and preventing serious complications, which include abnormal heart rhythms, heart failure and sudden death.

HCM can have many clinical presentations. While some individuals don't experience symptoms, others may experience chest pain, shortness of breath or fainting, especially during periods of exertion [2]. Chest pain on exertion results from an increased demand for blood flow to the body and to the thickened heart muscle itself and may be worsened by distorted coronary arteries.

Acute dynamic LVOT obstruction elevates left ventricular filling pressure, increasing myocardial oxygen demands, and ultimately leads to ischemia [3]

A microvascular dysfunction is a well-recognized feature of HCM, and its severity is a strong predictor of clinical deterioration and death. This dysfunction may precede clinical deterioration by several years [4]. Individuals can also experience palpitations due to abnormal electrical activity within the heart that causes abnormal heart rhythms due to thickening of the heart muscle and disturbance of the muscle cells. The clinical diagnosis of HCM is usually made using cardiac imaging, most commonly two-dimensional echocardiography and, increasingly, CMR [5]. Negative inotropes, such as beta-blockers or nondihydropyridine calcium channel blockers, are the most appropriate initial therapeutic interventions [6].

CONCLUSION:

The myocardial fibrosis in HCM is a progressive phenomenon caused by necrosis and leads to hypoplasia of small intramural coronary arteries, which may explain the ischemic clinical and electrical manifestations of the disease, with malignant ventricular tachyarrhythmias and risk of sudden death.

REFERENCES:

[1] Alpendurada F, Prasad SK. The missing spade: apical hy- pertrophic cardiomyopathy investigation. Int J Cardiovasc Imaging 2008; doi 10.1007/s10554-008-9335-z.

[2] B. J. Maron, “Contemporary insights and strategies for risk stratification and prevention of sudden death in hypertrophic cardiomyopathy,” *Circulation*, vol. 121, no. 3, pp. 445–456, 2010.

[3] Eriksson MJ, Sonnenberg B, Woo A, et al. Long-term outcome in patients with apical hypertrophic cardiomy- opathy. *J Am Coll Cardiol.* 2002;39:638-645.

[4] Jan MF, Todaro MC, Oreto L, Tajik AJ. Apical hyper- trophic cardiomyopathy: present status. Int J Cardiol 2016;222:745-59.

[5] Y.Daralammouri, M.El Garhy, K.Same and B.Lauer : Hypertrophic Cardiomyopathy Mimicking Acute Anterior Myocardial Infarction Associated with Sudden Cardiac Death- Case Reports in Medicine, 2012; 2012: 236154. Published online 2012 Aug 16.

[6] - F.Luzza, S.Carerj, G.Oreto - Hypertrophic cardiomyopathy with persistent ST segment elevation simulating acute myocardial infarction- BMJ journals- Volume 90, Issue 4 ; Online issue publication March 12, 2004.

[7] Ibrahim T, Schwaiger M. Diagnosis of apical hyper- trophic cardiomyopathy using magnetic resonance imaging. *Heart.* 2000;83:E1.

Abbreviations :

CMR	cardiovascular magnetic resonance
HCM	hypertrophic cardiomyopathy
LVOT	left ventricular outflow tract

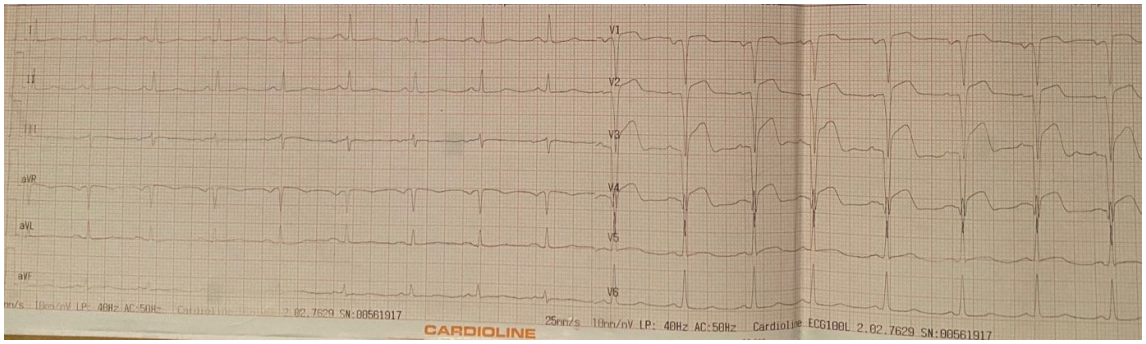

Figure 1: Standard 12-lead ECG shows sinus rhythm at 85 bpm, ST segment elevation in V1–V4.

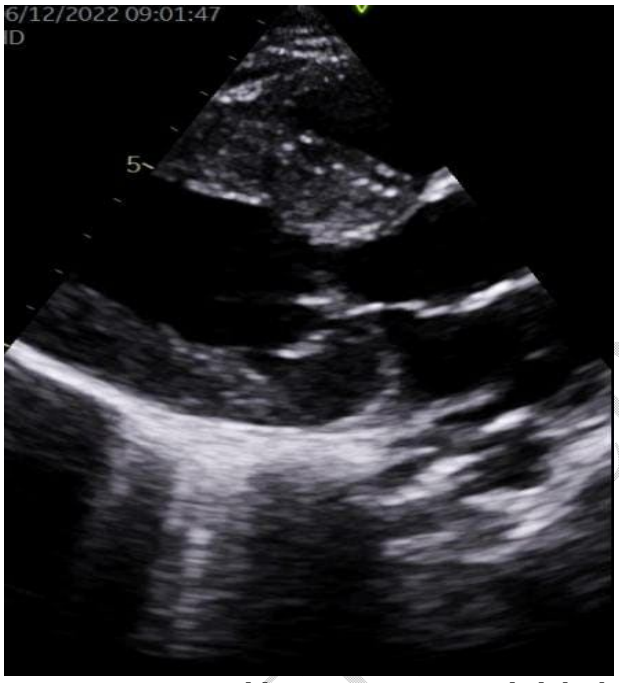

Figure 2: Parasternal long axis view revealed thickened left ventricular walls

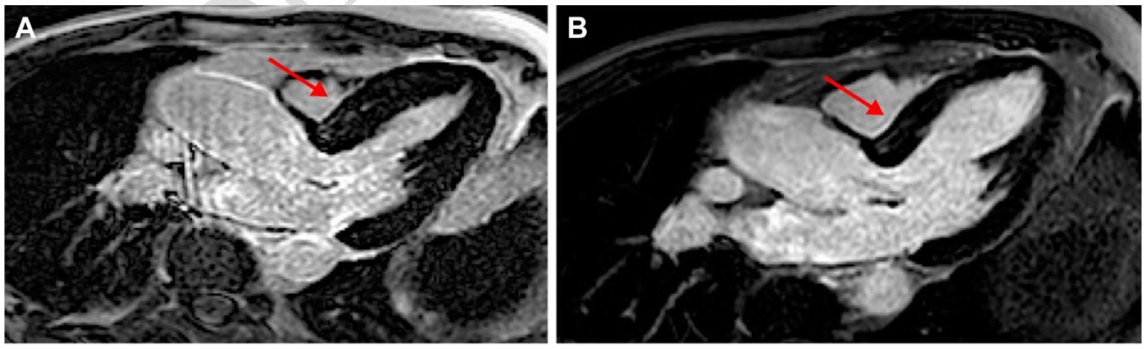

Figure 3: CMR 3-chamber view late Gadolinium enhancement images