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Case report: Apical variant hypertrophic cardiomyopathy simulating an acute inferior myocardial infarction

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ABSTRACT

We present a female patient who arrived to the emergency department with chest pain without known medical history at the time of consultation. Considering her risk factors and electrocardiographic findings, it was decided to perform a coronary catheterization under the suspicion of acute myocardial infarction. The catheterization and another studies was negative for coronary artery disease. Additional studies were conclusive for apical variant hypertrophic cardiomyopathy or Yamaguchi syndrome.

Background

Hypertrophic cardiomyopathy (HCM) is a common disease. It is characterized by an increase of myocardial thickness (≥15 mm) [1]. The hypertrophy can occur in any myocardial region, although the best known presentation is the one that compromises the interventricular septum, due to its association with sudden cardiac death. However, there are other less frequent presentations. Among them is the apical variant or Yamaguchi syndrome (YS), first described in 1979 by Yamaguchi and collaborators in 30 Asian subjects [2]. Nowadays it is known that this variant can be expressed in subjects outside the Asian continent, with an estimated prevalence between 1 and 11% of the total of patients with HCM in North America and Europe [3]. Data in other regions of the world, such as Latin America, is unknown.

The YS presents some particular electrocardiographic findings, such as the increase of the QRS complex voltage in the precordial leads and a deep inversion of the T wave, of greater amplitude in V4 that usually exceeds 10 mm (giant T waves) [2,4].

YS has good prognosis, because it is not associated with sudden cardiac death and its course is usually benign, although arrhythmias or chest pain may be present [3].

Case report

An 81-year-old female patient arrived to the emergency department with oppressive, non-irradiated high intensity chest pain and dyspnea. Her symptoms had started 3 h earlier. As part of her background she mentioned that she required a cardiac catheterization some years ago, without remembering the results or an official report of the procedure available at the time of arrival. At the physical examination, her blood pressure was 152/60 mm Hg, heart rate was 62 beats per minute and she had no respiratory distress. No other remarkable findings were found.

A surface electrocardiogram (ECG) was performed, revealing sinus rhythm, left ventricular hypertrophy (LVH), inversion of the T wave in the precordial leads, elevation of the ST segment in DIII and aVF with ST depression in DI, aVL and V3 to V6 (Fig. 1). We considered an acute ST-segment elevation myocardial infarction (STEMI) with inferior wall lesion, activated the catheterization laboratory and took blood tests. The result of cardiac catheterization was negative for coronary disease. Her initial high sensitivity troponin I (hs-TnI) was 0.015 ng/mL and control 6 h later was 0.016 ng/mL (99th percentile cut off of 0.016 ng/mL).

The patient had a favorable evolution, without new episodes of chest pain. A control ECG was taken, without relevant changes regarding to the initial one. The patient was discharged with medical management.

Follow up

During the hospitalization, the patient's family brought the previous medical history. Among the tests performed were a previous ECG with a pattern similar to the current one, a cardiac magnetic resonance with findings suggestive of apical variant hypertrophic cardiomyopathy, where the left ventricle is observed in the right view at the end of diastole with "ace of spades" shape, characteristic of YS (Fig. 2). In addition, an echocardiogram with pharmacological stress performed months

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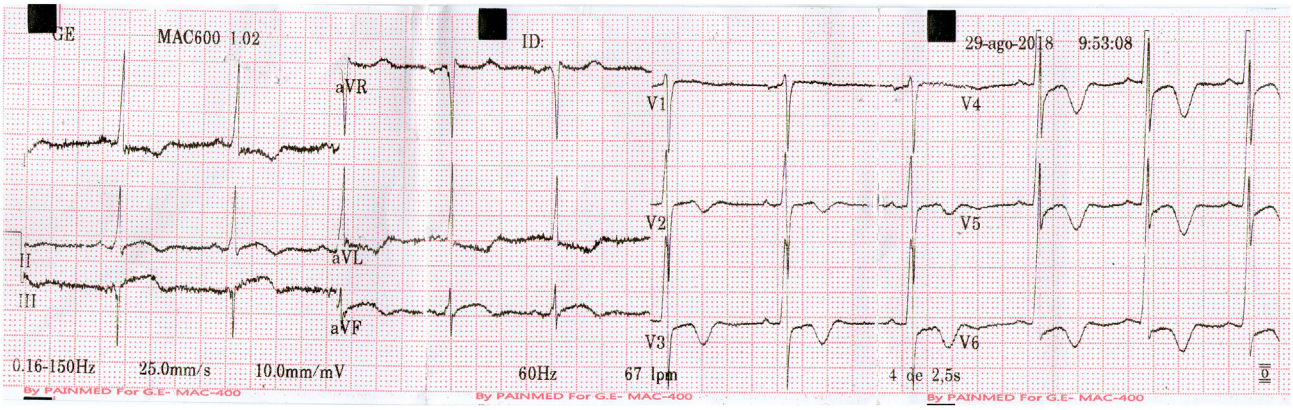

Fig. 1. ECG of the event in the emergency department. Sinus rhythm, left ventricular hypertrophy, ST segment elevation in DIII and aVF, ST segment depression in aVL, DI and V3 to V6, inversion of the T wave in V2 to V6. The artifacts in the baseline are due to muscle tremors.

before which were negative for coronary disease. We explained to the patient the nature of her disease and the reasons why it can be confused with a STEMI.

One week after the event, the patient remained asymptomatic. An ECG was performed, which remained unchanged with respect to the previous ones (Fig. 3).

Discussion

In the literature, several clinical situations have been described which can simulate a STEMI in the ECG. It has been estimated that up to 2.3% of patients who undergo catheterization with suspicion of STEMI may be due to ECG mimicry [5]. Previous cases have reported HCM as a mimic of STEMI, including patients with YS, specially by the inverted T wave in V3 to V6 [6]. Another factor that may aggravate this confusion is the borderline or slightly high value of hs-TnI [3], that was also seen in this case. Sometimes patients with YS may also experience chest pain [6].

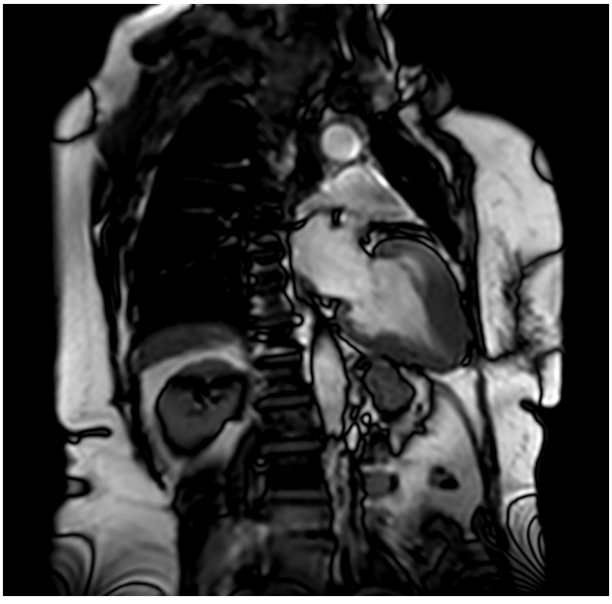

Fig. 2. Left ventricle is observed in the right view at the end of diastole with "ace of spades" shape, characteristic of Yamaguchi syndrome.

Previous case reports mentioning the YS simulating a STEMI, refer to changes of the T wave and ST deviation in the ECG secondary to HCM. Changes in ST due to HCM have been described with distinctive characteristics, compared to changes in ST as a result of ischemic myocardial injury. For example, in ST changes due to ischemia you can find reciprocal ST changes or "mirror changes", which is not usual with HCM [7,8].

In the patient of this case report, since her first ECG, ST deviation associated with severe chest pain was presented. Although ST-T abnormalities seen in DI and aVL could be probably due to HCM, It was striking to find ST elevation in DIII and aVF, that was interpreted as subepicardial lesion in inferior wall, which when accompanied by ST depression in DI and especially in aVL, have been considered as reciprocal ST changes and described as a highly sensitive sign for inferior wall myocardial infarction [9,10], but nevertheless these changes could be also due to LVH. And it was another reason for considering the realization of cardiac catheterization in this patient.

Coronary angiography was negative for coronary obstruction and considering the findings in complementary studies like cardiac magnetic resonance and echocardiogram, those indicates that the persistent ST elevation in the inferior wall and ST depression in DI and aVL presented in this patient were changes due to the apical variant HCM and perhaps not to ischemia. Establishing a situation where the apical variant HCM is presented as a false positive on the ECG for inferior wall STEMI.

The utility of non-invasive cardiac images is well known to clarify the etiology of chest pain and to avoid false positives that may lead to unnecessary invasive procedures in some patients [11]. Some electrocardiographic features may arise the suspicion of an apical variant HCM, including giant inverted T waves predominantly in V4, accompanied by a considerable increase in QRS voltage in V3 to V6 [2] or like this case ST deviation with apparent reciprocal changes. These ECG findings could lead to the clinician to the realization of non-invasive cardiac images increasing the diagnosis accuracy previous to consider invasive options.

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Conflict of interest and funding

The authors declare no conflict of interest.

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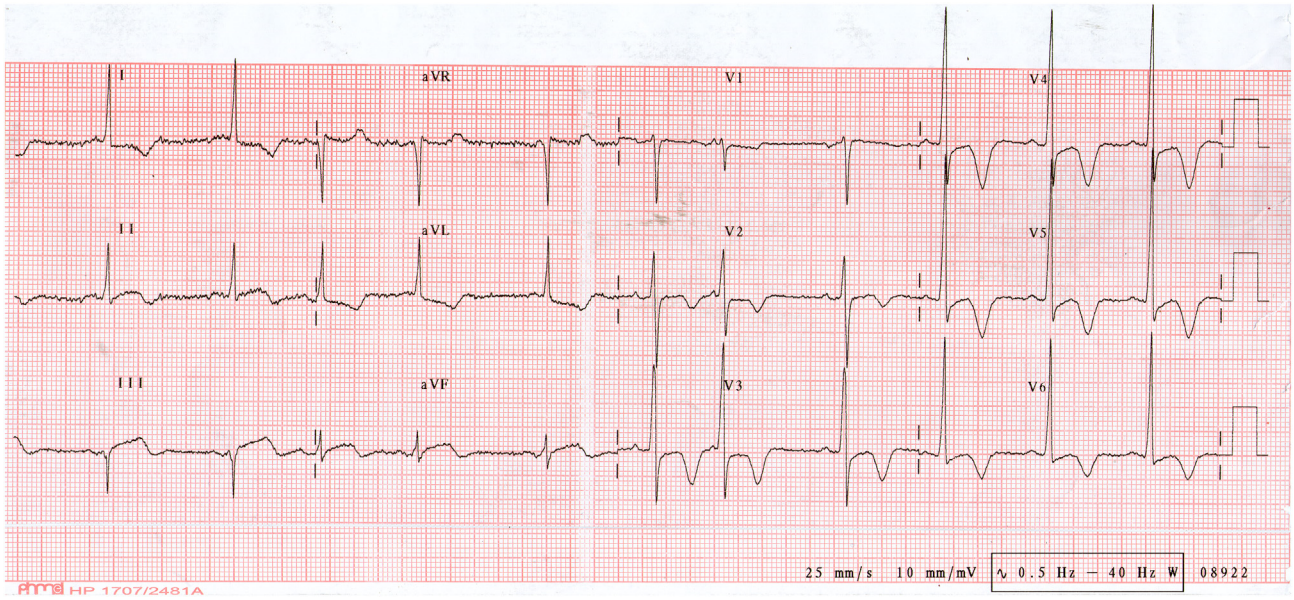

Fig. 3. ECG one week after the event. The ST segment deviation and T wave inversion persist without significant changes.

Informed consent

The patient gave her signature on the informed consent to make academic and scientific use of images and information in this case report.

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