CASE REPORT

Electrocardiographic Changes Announcing the Rapid Development of Apical Hypertrophic Cardiomyopathy in an Adult Male

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We presented the case of an adult male without structural heart disease, who in the period of 3 years developed apical hypertrophic cardiomyopathy. ECG changes preceded the development of ventricular hypertrophy. We discussed the appearance of ventricular enlargement during adulthood, in some cases of hypertrophic cardiomyopathy (HC), and how the ECG abnormalities may precede the onset of ventricular hypertrophy.

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CASE REPORT

A 47-year-old male was assessed in our hospital for chest pain. The patient had been studied in another hospital 3 years before, in February 2010 with the same symptoms. An ECG showed no abnormalities at that moment (Fig. 1). Several months later (October 2010), exercise testing was done. The ECG had changed, and showed a slight ST segment depression and negative asymmetric T waves in V₄–V₆ leads (Fig. 2). The stress test was positive electrically. The echocardiogram was normal. It was decided to perform a coronary angiography, which showed no abnormalities in the epicardial coronary arteries.

In March 2013, the patient went to the emergency room of our hospital sustaining that he had episodes of atypical chest pain at rest. He smoked about 10 cigarettes per day and had a 4-year history of hypertension with good pharmacological control, taking 10 mg of enalapril daily. The blood pressure was 135/85 and the physical examination was normal. An ECG, performed without chest pain, showed striking symmetrical deep negative T waves in most leads (Fig. 3). CPK and I Troponin levels were normal. An echocardiogram showed severe hypertrophy of all apical segments of the

left ventricle, and apex obliteration during systole, with preserved global and segmental contractile function. An MRI showed similar findings (Fig. 4). A diagnosis of apical hypertrophic cardiomyopathy was made and treatment was started with a daily dosage of 10 mg of bisoprolol instead of enalapril.

DISCUSSION

HCM is a disease entity caused by autosomal dominant mutations in genes encoding protein components of the sarcomere and its constituent myofilament elements.¹ Disorganization (disarray) of muscle cells and excess of connective tissue² can be seen. Ventricular hypertrophy, without cause justifying it, is the basis for clinical diagnosis, usually made by echocardiography.³

Hypertrophic cardiomyopathy is a heterogeneous disease. The typical evolution of a person carrying a genetic mutation is the absence or presence of very mild ventricular hypertrophy through adolescence, developing from that moment on a substantial thickening of the ventricular walls, usually reaching its final morphology with the end of growth of the individual, at around 18 years of age.⁴ However, in some cases, the onset

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Figure 1. February 2010 ECG. No significant alterations.

Figure 2. October 2010 ECG. A slight ST segment depression and negative asymmetric T waves in V₄–V₆ observed.

of hypertrophy is delayed until adulthood,⁵ and even at an old age.⁶ It is also possible that some carriers of genetic mutations may never develop hypertrophy,⁷ or that alterations are restricted only to a histological level.⁸ The clinical diagnosis is made primarily by imaging techniques (echocardiography or MRI) because, as is implicit in its name, the quintessence of this pathology is the hypertrophy of the septum and/or the ventricular walls. Interestingly, sometimes ECG abnormalities may be observed in patients with genetic mutations

that have not yet developed hypertrophy.^{5,9} These alterations often precede or announce the future development of hypertrophy. For this reason, the ECG may be a more sensitive method to detect the possible involvement of relatives of patients with hypertrophic cardiomyopathy, or to announce the future development of ventricular hypertrophy in individuals with a known genetic mutation but with normal wall thickness present.¹⁰

In our patient, electrocardiographic changes appeared in 2 ECG, separated by an interval of

Figure 3. March 2013 ECG. Deep negative T waves can be appreciated, almost symmetrical in all leads.

Figure 4. Cardiac MRI in March 2013.

8 months (ischemic heart disease was suspected). However, at that moment the echocardiogram was normal. Ventricular hypertrophy subsequently developed in what would be considered a small time span. After a period of 30 months, the ECG changes were substantial (and typical of apical HM), and imaging techniques showed thickening of the apical segments of the left ventricle walls.

This case shows the late appearance of apical hypertrophic cardiomyopathy in a relatively short time, with electrocardiographic changes preceding its development.

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