

## Case Report

## Hypertrophic Cardiomyopathy with Late Enhancement of the Non-Hypertrophied Left Ventricular Segments

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## Key words:

**Hypertrophic cardiomyopathy, cardiac imaging, magnetic resonance imaging, late enhancement.**

A 56-year-old asymptomatic man was referred for further evaluation because he displayed a rapid T-wave change on the ECG, from positive T waves to giant negative T waves in the anterolateral precordial leads, within 2 years. Transthoracic echocardiography revealed mild left ventricular apical hypertrophy without obstruction. Cardiac magnetic resonance imaging showed apical hypertrophic cardiomyopathy with focal hyperenhancement of the non-hypertrophic basal lateral segment of the left ventricle and absence of hyperenhancement of the hypertrophied apical segments.

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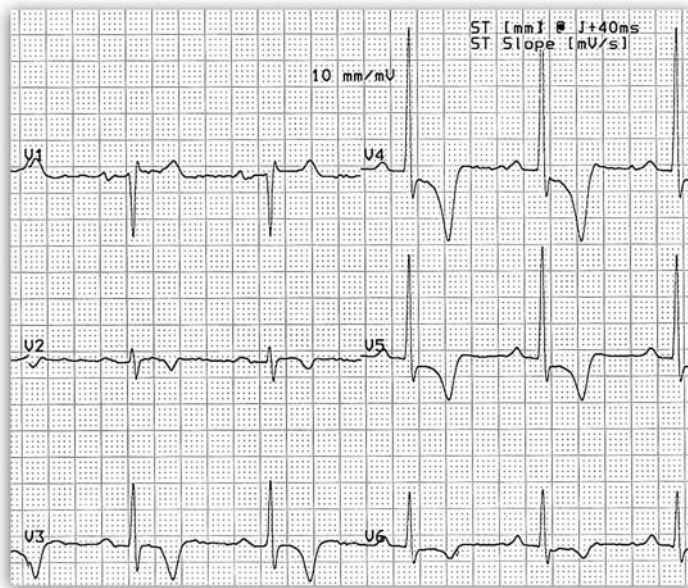
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**H**ypertrophic cardiomyopathy (HCM) is a relatively common primary cardiac disease with a great diversity of morphological, functional, and clinical features.<sup>1</sup> Areas of delayed enhancement on cardiac magnetic resonance imaging (MRI) in HCM patients, presumably attributable to foci of collagen deposition, have recently been described in asymptomatic or minimally symptomatic patients and associated with markers of sudden death and progressive ventricular dilatation.<sup>2-4</sup>

### Case presentation

A 56-year-old asymptomatic man was referred to our department for further evaluation because he displayed a rapid T-wave change on the ECG, from positive T waves to giant negative T waves in the anterolateral precordial leads, within 2 years, resembling an apical type hypertrophic cardiomyopathy (Figure 1). He had no chest discomfort, dyspnoea or syncopal episodes. The

patient's blood pressure and plasma glucose levels were normal and he was a non-smoker. He was not on any medication and there was no positive family history for HCM or premature sudden death. Transthoracic echocardiography revealed mild left ventricular apical hypertrophy without obstruction. Cardiac MRI showed apical HCM with focal hyperenhancement of the non-hypertrophic basal lateral segment of the left ventricle and absence of hyperenhancement of the hypertrophied apical segments (Figure 2). The patient had a normal exercise test, achieving 13.5 METS and 85% of the maximum predicted heart rate. He did not complain of any chest pain or dyspnoea, while ECG changes compatible with myocardial ischaemia were not evidenced. The blood pressure response to exercise was normal and no episodes of non-sustained ventricular tachycardia were detected in the 24h ECG recording. No medication was recommended. His 21-year-old daughter was checked by personal history, ECG and



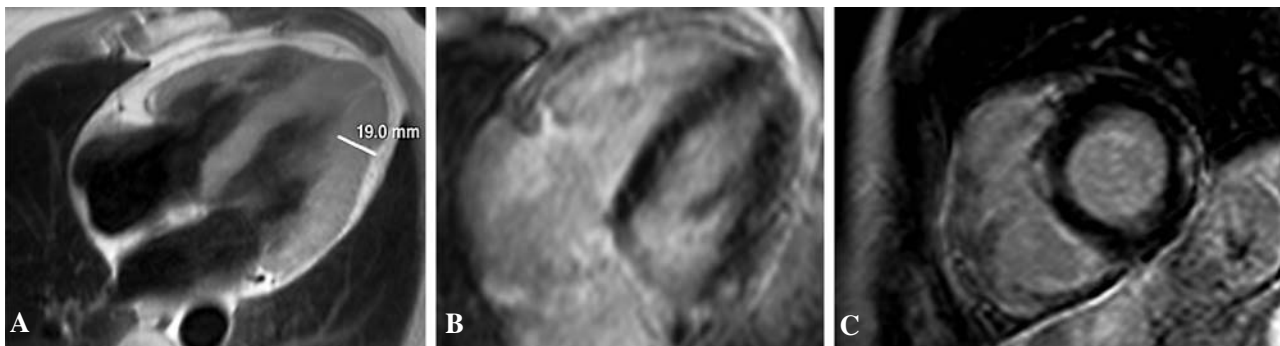
**Figure 1.** ECG displaying negative T waves in anterolateral precordial leads.

echocardiography and was found to be unaffected. The patient remains asymptomatic at 1-year follow up without any ECG or echocardiographic changes.

### Discussion

Here we present a case of apical HCM with focal hyperenhancement of the non-hypertrophic basal lateral segment of the left ventricle. Germans et al<sup>5</sup> showed that in HCM carriers who have not yet developed overt hypertrophy, cardiac MRI can detect crypts in the inferoseptal left ventricular wall, even when echocardiography and ECG are normal. Their location is similar to the typical location of the delayed contrast enhancement in HCM patients, which suggests that crypts, as

well as formation of focal replacement fibrosis—the histological counterpart of late contrast enhancement—might both reflect two different stages of the same disease process that ultimately results in manifest HCM. In general, hyperenhancement can occur not only in collagenous scar tissue, but also in any condition accompanied by a larger volume of distribution of gadolinium, such as disruption of cellular membrane, oedema, and increased blood volume. Therefore, hyperenhancement in HCM may be related not only to fibrosis but also to other conditions, such as disarray, inflammation, myolysis, and necrosis.<sup>6,7</sup> A recent study<sup>8</sup> found that 9 out of 53 patients (17%) showed delayed enhancement in segments with normal wall thickness, either as an isolated finding or associated with perfu-



**Figure 2.** Four-chamber turbo spin echo image (A) with apical hypertrophic cardiomyopathy, demonstrating thickening of the apical segments of the left ventricle (thickness of apical lateral segment: 1.9 cm). Four chamber (B) and basal short axis (C) delayed enhancement images demonstrating focal hyperenhancement at the non-hypertrophied basal lateral segment of the left ventricle. Note the absence of hyperenhancement of the hypertrophied apical segments of the left ventricle (C).

sion defects or hypokinetic wall motion at the respective areas of late enhancement.

In conclusion, late enhancement of the non-hypertrophied segments in HCM should not be considered exceptional, since the myopathic process (i.e. disarray, fibrosis or necrosis) affects the entire left ventricle irrespectively of the wall thickness.<sup>9</sup>

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