



Case Report

Appearance of myocardial fibrosis during long-term follow up in a patient with apical hypertrophy



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ABSTRACT

A 67-year-old man without symptoms presented with significant negative T wave on electrocardiography. No significant abnormality was detected by echocardiography. Cardiac magnetic resonance imaging (CMR) showed trivial hypertrophied myocardium in apex. During follow up of 9 years, serial CMR scans revealed the appearance and expansion of late gadolinium enhancement in apex. The patient had never complained of cardiac symptoms such as chest pain, palpitation, or syncope during follow up. We experienced the appearance of significant myocardial fibrosis in a patient with apical hypertrophy.

<Learning objective: Apical hypertrophy is characterized by hypertrophied apex and giant negative T wave. From this report, we appreciate the importance of serial cardiac magnetic resonance imaging to follow the progress of hypertrophied myocardium even for patients without significant gadolinium enhancement who can be in a premonitory state of irreversible fibrosis.>

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Introduction

Apical hypertrophy (APH) is a relatively rare type of hypertrophic cardiomyopathy. It is frequently reported from Asian cohorts, and typically features spade-shaped left ventricular cavity and giant inverted T wave [1]. This type of hypertrophy seems to have benign prognosis for cardiovascular mortality among hypertrophied cardiomyopathies if there is no apical aneurysm [1–3]. Myocardial fibrosis can be identified by late gadolinium enhancement (LGE) cardiovascular magnetic resonance (CMR), and it is known to be associated with cardiovascular mortality. In this case, we experienced an elderly male with trivial APH and managed to capture the appearance and expansion of myocardial fibrosis during 9 years of serial CMR.

Case report

A 67-year-old man presented with asymptomatic electrocardiogram (ECG) abnormality. No significant cardiovascular history except hypertension was identified. ECG demonstrated remarkable

T wave inversion in lead V3–6 (Fig. 1A). CMR (1.5 Tesla scanner, Gyroscan[®] ACS-NT, Phillips Medical Systems, Best, the Netherlands) was performed to evaluate cardiac morphology. Mildly hypertrophied myocardium in apex was suspected in cine-magnetic resonance imaging (MRI). LGE was not detected (Fig. 1A).

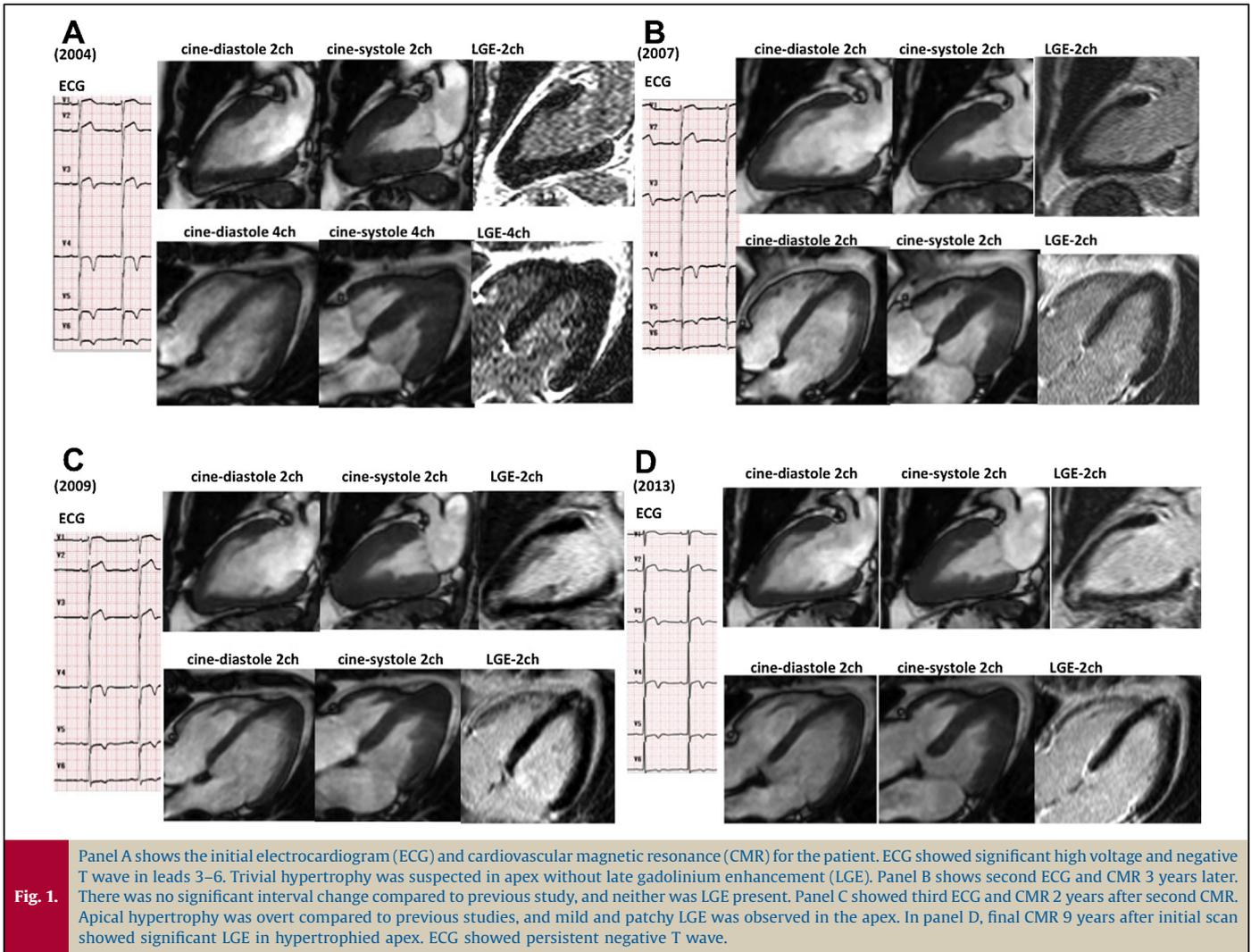
The patient was suspected of having APH, and follow-up CMR 3 years later demonstrated similar findings to the previous examination (Fig. 1B). However, 5 years later, a third CMR scan revealed focal-patchy signal had appeared in the apex (Fig. 1C). In follow-up scan 9 years later, LGE was more obvious and showed remarkable expansion in the apex. Compared to initial CMR, apical hypertrophy was found to be overt (Fig. 1D). During follow up, neither significant ECG change, arrhythmia, newly onset symptoms, or high brain-type natriuretic peptide (BNP) was detected. BNP was maintained between 40–80 pg/mL. No significant abnormal flow in apex appeared in echocardiography. And the analysis with wall motion from cine-MRI showed no significant progression of diastolic dysfunction (peak filling rate and time to peak filling were around 170–220 mL/s, and 700–900 ms).

Discussion

APH is frequently reported in Asian cohorts, and typically features predominant hypertrophy in the apex and giant negative T

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wave [4,5]. In this case, we managed to capture the appearance and expansion of myocardial fibrosis during 9 years of serial CMR. Compared to echocardiography, the advantage of CMR is to cover the whole heart including apex where echocardiography occasionally fails to penetrate. Myocardial infarction (MI) also needs to be differentiated. However, LGE in this case showed gradual expansion along many years which is unlike the natural course of MI. In the study of relatively large population for prevalence of LGE in APH, Yamada et al. reported the frequency of LGE was less than half [6]. The appearance and progression of myocardial fibrosis in hypertrophic cardiomyopathy is explained as premature myocyte death and collagen deposition by sarcomere mutation, and followed by small vessel disorder, ischemia, and remodeling [7]. The clinical course in this report indicated the early manifestation of fibrosis and focal hypertrophy could occur simultaneously. Thus, we propose the need for serial CMR scan even for the patients without significant LGE to identify the subjects at risk for future cardiovascular events.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.jccase.2018.12.008>.

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