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Progression of electrocardiographic changes in a patient with apical hypertrophic cardiomyopathy

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ABSTRACT

A 58-year-old man asymptomatic from the cardiovascular point of view and with no known relevant family history was found by transthoracic echocardiography to have apical hypertrophic cardiomyopathy (AHCM). His electrocardiogram (ECG) revealed prominent precordial R-waves, particularly in V3–V4 leads, and "giant" (>1.0 mV), inverted T-waves, previously associated with AHCM. ECGs recorded 17 and 13 years previously, did not disclose such abnormalities, as the ones of his current ECG. The presented case illustrates a potential role of serial ECGs (along with serial imaging testing) in detecting the development and progression of regional left ventricular hypertrophy in patients with AHCM, and probably in other hypertrophic cardiomyopathy phenotypes.

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Case report

A 58-year-old man with a past medical history of diabetes, hyperlipidemia, and hypothyroidism was referred to cardiology clinic for a preoperative risk assessment prior to cataract surgery, due to an abnormal electrocardiogram (ECG) (Fig. 1). The patient had no cardiac symptoms and no known family history of structural heart disease, cardiomyopathy, arrhythmias, or sudden cardiac death. Transthoracic echocardiogram (TTE) was notable for apical hypertrophy with maximal wall thickness of 17 mm (Fig. 2), normal bi-ventricular function, no major valvular disease, and no apical aneurysm. No prior TTE or alternative cardiac imaging was available for comparison, to ascertain the temporal course of the left ventricular (LV) apical hypertrophy. The patient underwent successful cataract surgery without cardiovascular adverse events. Baseline ECG (Fig. 1) was abnormal. Review of previous ECGs obtained 17 (Fig. 3A) and 13 (Fig. 3B) years prior to his most recent (Fig. 1) ECG assessment are also shown.

Discussion

The most recent ECG (Fig. 1) is suggestive of apical hypertrophic cardiomyopathy (AHCM), also known as Japanese-variant of hypertrophic cardiomyopathy, due to its high prevalence in the Japanese population. ECG in this condition is often characterized by prominent precordial R-waves, particularly in V3–V4; in addition to "giant" (>1.0 mV), inverted T-waves [1]. The automated ECG measurements for the R-waves of leads V3, V4, and V5 (Fig. 1), provided by the electrocardiograph analysis algorithm, were 2.184 mV, 2.831 mV, and 2.341 mV, respectively, whereas the corresponding measurements for the ECG of Fig. 3B were 0.45 mV, 0.77 mV, and 0.86 mV, amounting to a 485%, 368%, and 273%, increase in the amplitude of the R-waves, in the intervening 13 years.

The mechanism for the "giant" R-waves in lead V4 is primarily attributed to the location of LV hypertrophy in patients with AHCM (opposite to the *non-muscular* elements of the fibrous cardiac skeleton of mitral valve and annular plane, which entails an *unopposed* depolarization with vectorial forces directed towards the apex, and resulting in enormous R waves [1]. In other types of LV hypertrophy, like for example the one encountered after myocardial infarction (MI), LV hypertrophy affects only the opposite side of the myocardium because of remodeling, and therefore it is not a circular LV hypertrophy [2]. LV hypertrophy in our patient was of similar non-circular type, confined to the LV apex, and thus it requires an explanation (i.e. an exclusion of an old MI). Our patient was asymptomatic, without history of coronary artery disease, but one could not exclude a silent MI in a diabetic patient. However, the absence of regional contraction abnormalities in the echocardiogram (Fig. 2), and the alternative explanation provided above (i.e., an unopposed LV hypertrophy vector in AHCM), explains the mechanism of the altered ECG (Fig. 1).

Hypertrophic cardiomyopathy (HCM) is a heterogenous condition with varying clinical presentations ranging from incidental discovery in asymptomatic patients to sudden cardiac death. Age-related penetrance resulting in delayed appearance of LV hypertrophy has been previously described [3]. We speculate that our patient exhibits an apical

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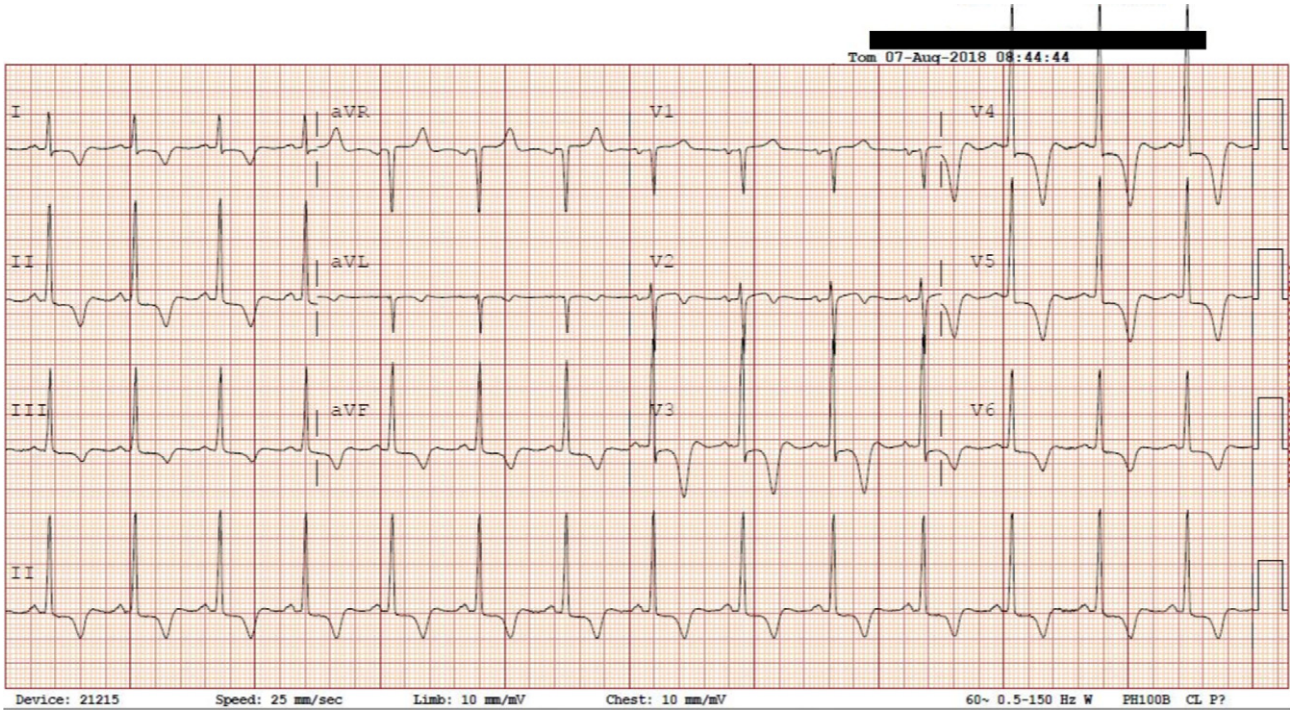

Fig. 1. ECG obtained in 2018.

variant of HCM, where LV hypertrophy developed during his fifth or sixth decade. No alternative explanation could account for the changes observed in the ECG throughout the years. Although there are several other methods useful in detecting the development and progression of LV hypertrophy, including echocardiography, other imaging modalities, and ECG body surface mapping, this unusual case illustrates the significant role that ECG could play in the diagnosis of HCM, particularly in patients who may develop left ventricular hypertrophy later in life, via the advantages of serial ECGs (a ubiquitously available and cost-effective diagnostic method).

AHCM variant results in selective hypertrophy of the LV apex, and does not typically result in obstructive physiology. This heterogeneous condition has varied clinical presentation, ranging from incidental discovery in asymptomatic patients to sudden cardiac death. Apical aneurysm is an increasingly recognized subset of AHCM and is believed to confer higher risk for arrhythmic sudden death and thromboembolic events. Age-related penetrance, resulting in delayed appearance of LV hypertrophy, has been previously described [3]. We speculate that our patient developed AHCM during his fifth or sixth decade of life, although this could not be corroborated by comparison of his current TTE with previous TTEs or other imaging testing.

The management of AHCM is the same as in the other more common HCM phenotypes, but the apical variant generally portends a more benign prognosis [4]. This case illustrates the significant role that the ECG could play in the diagnosis of AHCM, particularly in patients who develop LV hypertrophy later in life. Indeed, serial ECGs could be used to monitor the emergence and further progression of LV hypertrophy in AHCM, and probably in other HCM phenotypes. The present case report suggests that serial ECGs and corresponding imaging tests may be revealing in regards to the development and progression of LV hypertrophy in patients

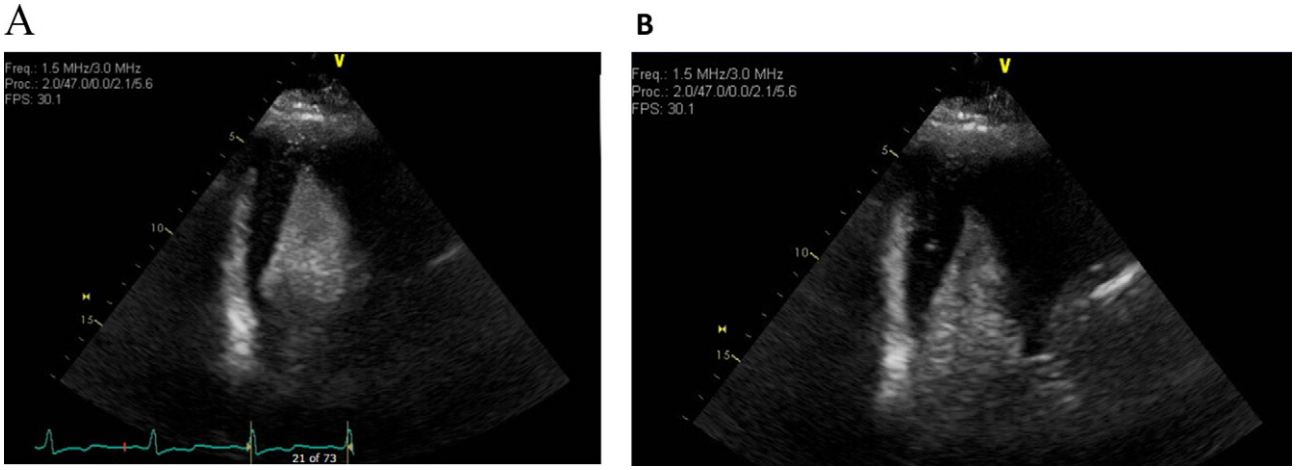

Fig. 2. Transthoracic echocardiogram in diastole (A) and systole (B) showing marked apical hypertrophy and normal left ventricular function.

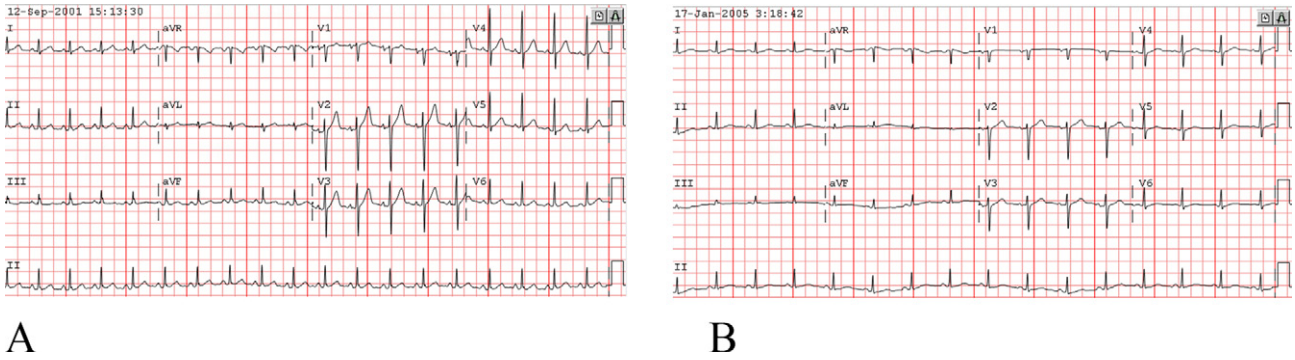

Fig. 3. ECGs obtained in 2001 (A) and 2005 (B).

with HCM, and such undertaking could be accomplished by scrutiny of follow-up data available in registries of patients with HCM, followed longitudinally [3].

Funding

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Declaration of competing interest

None.

References

[1] Madias JE. Electrocardiogram in apical hypertrophic cardiomyopathy with a speculation as to the mechanism of its features. Neth Heart J 2013;21:268–71.
[2] Mozos I, Hancu M, Jost N. Isointegral body surface maps and left ventricular hypertrophy in post-infarction heart failure patients. Acta Physiol Hung 2012;99:18–24.
[3] Maron BJ, Maron MS. Hypertrophic cardiomyopathy. Lancet 2013;381:242–55.
[4] Eriksson MJ, Sonnenberg B, Woo A, et al. Long-term outcome in patients with apical hypertrophic cardiomyopathy. J Am Coll Cardiol 2002;39:638–45.