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Case Report

Relevance of increased negative T waves in apical hypertrophic cardiomyopathy with progressive myocardial damage: Insights from repeat cardiac magnetic resonance studies ☆,☆☆

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ABSTRACT

In patients with apical hypertrophic cardiomyopathy (HCM), progressive electrocardiographic changes are observed during long-term follow-up. However, it is difficult to correspond these changes to the specific myocardial changes. Cardiac magnetic resonance (CMR) imaging can elucidate myocardial changes by late gadolinium enhancement. Here, we present the long-term follow-up (>18 years) on a patient with apical HCM, whereupon, precise and continuous changes in the myocardium, causing ST segment and T wave changes on electrocardiography, were observed on CMR images. The combination of electrocardiography and CMR facilitates management of patients with apical HCM because it helps explain and understand the nature of electrocardiography changes over time.

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Introduction

Apical hypertrophic cardiomyopathy (HCM) is characterized by myocardial hypertrophy at the apex of the heart, a spade-shaped left ventricular lumen, and the presence of giant negative T waves with an amplitude >1 mV in the precordial leads on electrocardiography (ECG) [1]. Although the disease progresses without any symptoms in most cases, cardiovascular events, including nonfatal arrhythmia and myocardial infarction may occur in few, and thus, necessitate follow-up observation [2]. In patients with HCM, ECG changes occur continuously over time as the disease progresses. However, precise analysis of the types of ECG changes that reflect the specific myocardial changes over a long-term follow-up period has not been reported. Although myocardial biopsy might be the ultimate technique to definitively determine the pathological changes in the myocardium correlating with ECG changes, it is invasive and difficult to perform [3]. Meanwhile, late gadolinium enhancement (LGE) observed on cardiac magnetic resonance (CMR) imaging indicates myocardial fibrosis, which confirms myocardial damage [4], and may allow considering the relation between the changes in ECG and myocardium with increased ability to depict the cardiac morphology and the movement. Herein, we report a case of apical HCM in which CMR imaging revealed serial cardiac changes related to ST segment and T wave changes in ECG during a long-term follow-up period of 18 years.

Case report

A male patient with an ECG abnormality visited our hospital for the first time at the age of 34 years. The patient was healthy, with no noteworthy medical or family history of cardiac or other systemic diseases. Changes in the limb and precordial leads over time are shown in Fig. 1. The ECG at that time showed a negative T wave in V4–6 leads (V4, 0.3 mV), but no left ventricular hypertrophy was observed (SV1 + RV5 [the Sokolow–Lyon voltage] was 3.12 mV; Table 1) [5]. In the precordial leads, the Sokolow–Lyon voltage showed an increasing tendency over time (3.12 mV at 34 years, 5.68 mV at 45 years, and peaked at 5.86 mV at 50 years of age) [5]. The amplitudes of negative T waves continued to increase in V4–6 leads from the age of 34 years and reached 2.5 mV in the V4 lead at the age of 45 years and then continuously decreased until the end of observation at the age of 52 years (V4, 1.4 mV). Minimal ST-segment depression appeared in limb leads II, III, and aVF at the age of 42 years, which peaked at the age of 45 years and then disappeared. At the age of 42 years, ST-segment depression appeared in precordial leads V4, V5, and V6, which peaked at the age of 45 years (V4: S=−0.5 mV) and then improved slightly but persisted until the age of 52 years.

A spade-shaped left ventricle was observed on cine CMR images (Fig. 2). Myocardial weight increased over time (from 137 g at the age of 42 years to 183 g at the age of 52 years; Table 1). Regarding left ventricular contractility, left ventricular ejection fraction remained between 56% and 69% during follow-up. The presence of LGE could not be confirmed at 42 and 43 years of age; however, faint LGE localized to the middle layer of the apical myocardium was first observed at the age of 45 years. Thereafter, no increase in the volume of the LGE area, although it became clearer over time, was observed (Fig. 2). In addition, the patient remained asymptomatic throughout the follow-up period without any complications.

Discussion

In the present case, during the 18-year long-term follow-up of a patient with apical HCM, the following observations were made based on simultaneous ECG and CMR observations. First, although cine CMR imaging revealed continued progression of myocardial hypertrophy, the amplitudes of negative T waves in the precordial leads increased initially and decreased later, while the Sokolow–Lyon voltage continued to increase until the age of 50 years. Second, ST segment depression in V4–6 leads occurred during LGE appearance on CMR images. As for

Table 1 – Electrocardiogram and cardiac magnetic resonance findings.

Age (y)	34	35	39	42	43	45	46	50	52
ECG findings									
Heart rate (beat/min)	64	61	60	57	67	55	58	68	48
QRS (sec)	0.095	0.096	0.102	0.100	0.103	0.102	0.107	0.096	0.108
QTc (sec)	0.412	0.392	0.416	0.396	0.411	0.426	0.423	0.477	0.408
SV_1 (mV)	1.37	1.81	2.17	1.95	1.94	2.05	1.81	2.07	1.84
RV_5 (mV)	1.75	2.17	2.67	3.75	2.39	3.63	3.60	3.79	2.99
Neg T in V4 (mV)	0.3	0.8	1.2	1.4	1.4	2.5	1.7	1.6	1.4
CMR findings									
LVEDV (mL)				136.1	150.8	145.1	146.2	140.0	130.0
LVESV (mL)				54.1	67.0	44.8	61.4	47.0	50.7
LVEF (%)				60.3	55.6	69.1	58.0	66.5	61.0
LV mass (g)				137.4	153.9	174.9	156.6	175.7	182.7
LGE				-	-	+	+	+	+

CMR, cardiac magnetic resonance; ECG, electrocardiogram; LGE, late gadolinium enhancement; LV, left ventricular; LVEDV, left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end systolic volume; Neg T, negative T wave.

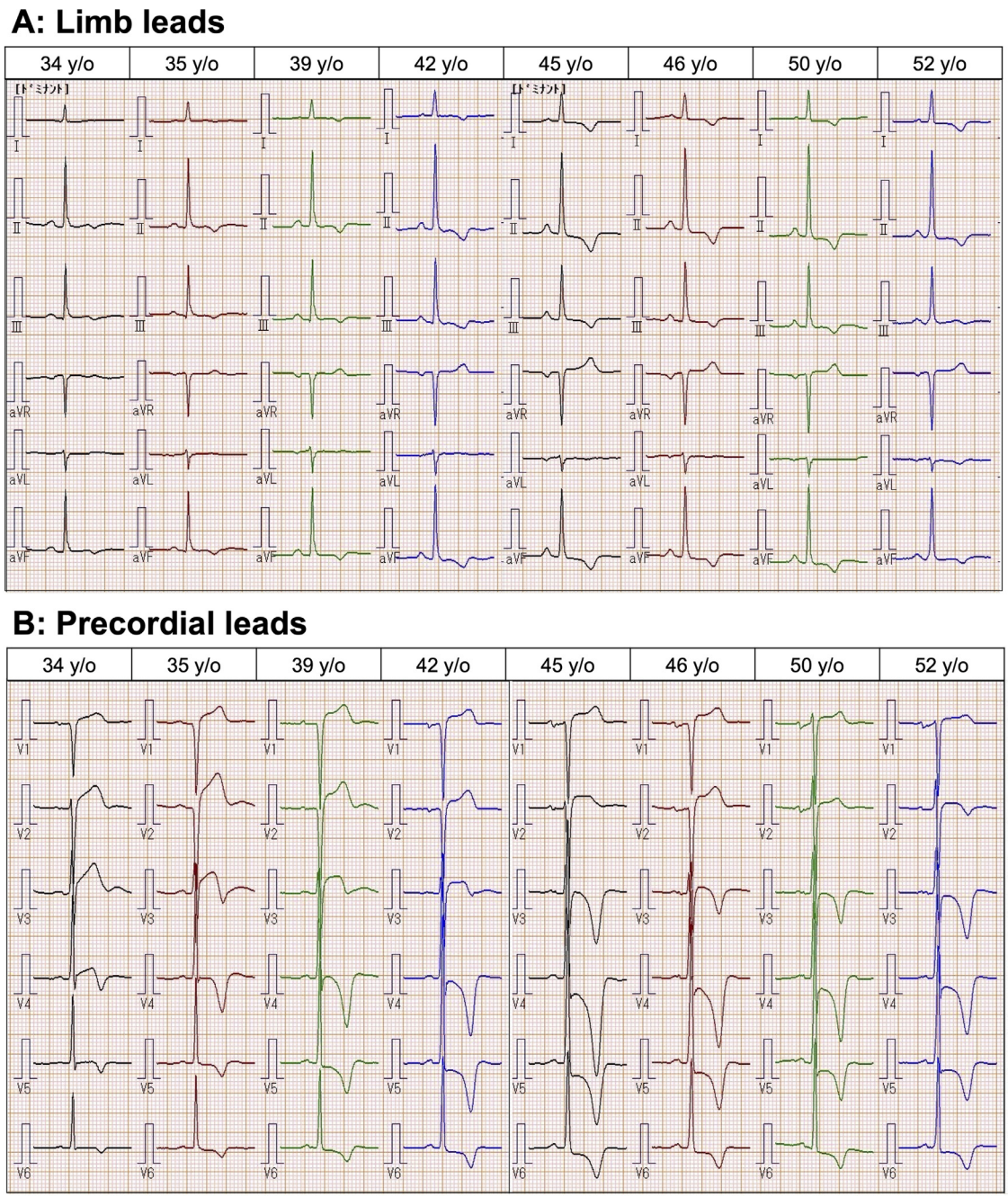

Fig. 1 – Electrocardiographic changes over 18 years. The upper row shows limb leads: at the age of 42 years, minimal ST segment depression appeared in II, III, and aVF leads, which peaked at age 45 years and then disappeared. The lower row shows precordial leads: at the age of 34 years, a negative T wave was detected in the V4 lead, and the amplitude of this negative T wave increased yearly until the age of 45 years. At the age of 42 years, the ST segment depression in the V4 lead decreased slightly. At the age of 45 years, both ST segment depression and amplitude of the negative T wave in the V4 lead decreased substantially. The amplitude of the negative T wave reduced, but ST segment depression persisted thereafter.

the progression of myocardial hypertrophy and electrocardiographic changes, the Sokolow–Lyon voltage, which is an indicator of left ventricular hypertrophy, was 3.12 mV at the age of 34 years, but after the age of 35 years, it exceeded 3.5 mV and peaked (5.86 mV) at the age of 50 years, indicating progression of wall thickening. CMR imaging confirmed a 34% increase in myocardial weight over 10 years (from 137 g [42 years] to 183 g [52 years]). However, the amplitudes of negative T waves increased over time until the age of 45 years and then decreased; this maybe an outcome of prolongation of the action potential duration of the hypertrophied myocardium and the progression of electrical remodeling [6]. In the present case, after

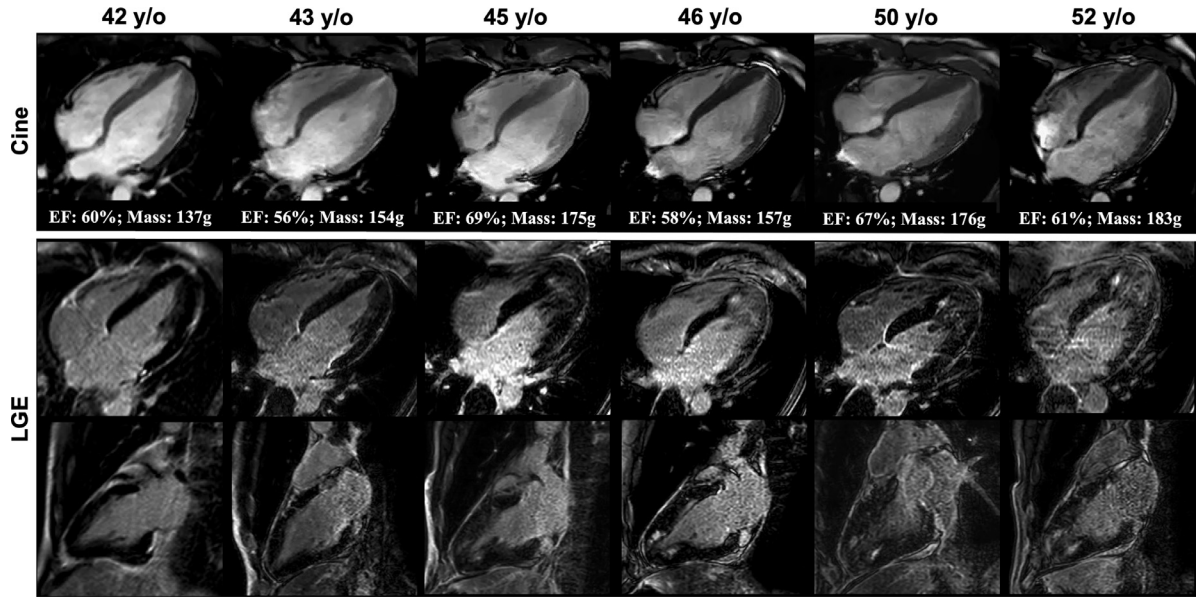

Fig. 2 – Changes revealed using cine and late gadolinium enhancement (LGE) cardiac magnetic resonance (CMR) imaging over 10 years. The upper row shows 4-chamber cine images, and the middle and lower rows show 4-chamber and 2-chamber LGE images, respectively. CMR imaging revealed an increase in cardiac weight and no change in ejection fraction (EF). At the age of 45 years, LGE appeared in the apical mid-layer myocardium. No new area with LGE appeared thereafter.

45 years of age, the amplitude of the T wave decreased despite an increase in myocardial mass as revealed on cine CMR imaging. In a previous report, the disappearance of negative T waves was confirmed in 71% of long-term follow-up studies conducted over 10 years [7]; the etiology of this depletion has not been entirely clarified. In the present case, however, it might have been due to decreased electrical potential on the intimal side caused by myocardial necrosis since fibrotic areas localized to the inner side were observed on CMR imaging. Furthermore, some reports have mentioned the possibility of hypertrophy extending beyond the apex toward the base of the heart, canceling out the negative component of the T wave [7]. In the present case, because the hypertrophy progressed, as revealed by CMR imaging, a similar phenomenon might have occurred.

In the present case, ST depression of 0.4 mV suddenly appeared at the age of 45 years and continued thereafter. We believe that this rapid development of ST depression suggests myocardial injury leading to fibrosis in the hypertrophic region, as suggested by the LGE on CMR imaging performed at the same age. Previous studies using ^{123}I-beta methyl iodophenyl pentadecanoic acid imaging have reported that fatty acid metabolism does not occur in the enlarged part of the area of apical HCM, resulting in ischemia and an anaerobic metabolic state [8]. Severe ischemia or necrosis might have occurred owing to a lack of oxygen as myocardial hypertrophy progressed. In addition, careful observation of the ECG revealed minimal ST depression in the V5 lead from the age of 39 years. However, no LGE indicative of myocardial fibrosis was revealed on CMR imaging at 42 and 43 years of age. Reportedly, capturing fibrosis as LGE might be difficult unless the amount of collagen in the myocardium exceeds 15% histologically. We believe that minimal subendocardial ischemia or fibrosis, which could not be visualized using delayed contrast imaging, might have slowly progressed from the time of minimal myocardial fibrosis. The manifestation of minimal myocardial fibrosis as minimal ST changes may require attention as a precursor to severe ischemia or infarction [9].

This study however, has a limitation. It is a single case study obtained from a single institution. However, there are few reports of detailed comparisons of ECG and CMR imaging changes in patients with apical HCM during long-term follow-up. Our results suggest that CMR imaging may help explain and understand the nature of ECG changes over time.

Based on our observations, we conclude that important myocardial changes may be suggested by changes in ECG, especially the initiation of a decrease in the height of negative T waves and the sudden appearance of ST depression. Careful follow-up using ECG and CMR imaging can prove beneficial for patients with apical HCM.

Patient consent

I hereby confirm that written informed consent was obtained from the patient for the publication of their case details and any accompanying images. The patient was thoroughly informed about the nature of the publication, the extent of the information to be disclosed, and the potential risks and benefits involved. The patient has voluntarily agreed to the publication, understanding that their identity will remain confidential and that all personal identifiers will be removed or anonymized to the fullest extent possible.

Ethical statement

The study protocol was approved by the ethics committees of Fukuokaken Saiseikai Futsukaichi Hospital (approval no. 468). All procedures were followed in accordance with the Declaration of Helsinki.

Declaration of generative AI in scientific writing

No generative AI tools were used in the writing of this research. All writing and analysis were performed by the authors.

Data availability statement

The data that support the findings of this study contain personal information and are not publicly available due to privacy and ethical considerations. Access to the data is restricted and requires approval from an ethics committee and an appropriate data use agreement. For more information, please contact the corresponding author.

REFERENCES

[1] Jan MF, Todaro MC, Oreto L, Tajik AJ. Apical hypertrophic cardiomyopathy: present status. Int J Cardiol 2016;222:745–59. doi:10.1016/j.ijcard.2016.07.154.

[2] Eriksson MJ, Sonnenberg B, Woo A, Rakowski P, Parker TG, Wigle ED, et al. Long-term outcome in patients with apical hypertrophic cardiomyopathy. J Am Coll Cardiol 2002;39:638–45. doi:10.1016/s0735-1097(01)01778-8.

[3] Cooper LT, Baughman KL, Feldman AM, Frustaci A, Jessup M, Kuhl U, et al. The role of endomyocardial biopsy in the management of cardiovascular disease: a scientific statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology. Circulation 2007;116:2216–33. doi:10.1161/CIRCULATIONAHA.107.186093.

[4] Mahrholdt H, Wagner A, Judd RM, Sechtem U, Kim RJ. Delayed enhancement cardiovascular magnetic resonance assessment of non-ischaemic cardiomyopathies. Eur Heart J 2005;26:1461–74. doi:10.1093/eurheartj/ehi258.

[5] Sokolow M, Lyon TP. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. Am Heart J 1949;37:161–86. doi:10.1016/0002-8703(49)90562-1.

[6] Tsunakawa H, Wei D, Mashima S, Harumi K. Study on the genesis of giant negative T wave in apical hypertrophic cardiomyopathy using a three-dimensional computer model. Jpn Heart J 1991;32 799–09. doi:10.1536/ihj.32.799.

[7] Koga Y, Katoh A, Matsuyama K, Ikeda H, Hiyamuta K, Toshima H, et al. Disappearance of giant negative T waves in patients with the Japanese form of apical hypertrophy. J Am Coll Cardiol 1995;26:1672–8. doi:10.1016/0735-1097(95)00377-0.

[8] Akutsu Y, Shinozuka A, Huang TY, Watanabe T, Yamada T, Yamanaka H, et al. Hypertrophic cardiomyopathy with apical left ventricular aneurysm. Jpn Circ J 1998;62:127–31. doi:10.1253/jcj.62.127.

[9] Moon JC, Reed E, Sheppard MN, Elkington AG, Ho SY, Burke M, et al. The histologic basis of late gadolinium enhancement cardiovascular magnetic resonance in hypertrophic cardiomyopathy. J Am Coll Cardiol 2004;43:2260–4. doi:10.1016/j.jacc.2004.03.035.