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Pseudo ST segment elevation myocardial infarction in a case of apical hypertrophic cardiomyopathy

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ABSTRACT

Background: Apical hypertrophic cardiomyopathy (AHCM) is a relatively rare variant of hypertrophic cardiomyopathy. The condition is associated with numerous clinical and electrocardiogram (ECG) features, some of which are well documented in the literature.

Case Presentation: We present a case of a 77-year-old female who presented with a syncopal episode. Her ECG showed lateral ST segment elevation and her cardiac biomarkers were not in keeping with a myocardial infarction. She was diagnosed with AHCM based on characteristic findings on echocardiography. The association between AHCM and lateral ST segment elevation is one that has only been described in a handful of cases in literature.

Conclusion: The authors hope that in presenting this case we can add to the body of literature and remind readers to be aware of the possibility that lateral ST segment elevation may be an indicator of AHCM, especially when this electrocardiographic feature occurs in the absence of the other well-known causes of ST segment elevation.

Keywords: Apical hypertrophic cardiomyopathy, electrocardiogram, echocardiogram, ST segment elevation myocardial infarction, case report.

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Background

Hypertrophic cardiomyopathy is defined as a primary myocardial disease characterized by left ventricular hypertrophy in the absence of another cardiac or systemic disease [1]. Recent population-based studies suggest that the prevalence of the disease is 0.2% of the general population but the number of diagnosed cases is suspected to be 1/3,000 [1].

Apical hypertrophic cardiomyopathy (AHCM) is more common in the East Asian population representing 15% of Hypertrophic cardiomyopathy (HCM) cases in Japan and up to 3% of non-Asian HCM patients [2]. AHCM is a segmental cardiomyopathy that predominantly involves the apex of the left ventricle. Rather than causing left ventricle outflow tract obstruction, it results in mid-ventricular obstruction [3].

Case Presentation

A 77-year-old female with a background of essential hypertension, type two diabetes mellitus, and atrial fibrillation (AF) presented to the emergency room after a syncopal episode. The fainting episode occurred as she was in the kitchen making tea. There was no chest pain or shortness of breath. On examination, she was found to have a postural drop of 30 mmHg from lying to standing blood pressure measurement. Her observations were as follows: blood pressure of 172/86 mmHg (right arm, supine position), heart rate of 82/minute, and a respiratory rate of 19/minute. Her cardiovascular examination revealed an irregular heart rate with a normal first and second heart sound. There was no murmur. Her respiratory and abdominal examination was unremarkable.

She had no family history of sudden cardiac death and denied alcoholism or recreational drug use.

Her baseline Troponin I was 21 ng/ml (reference range <20 ng/ml) and the second sample taken 1 hour apart was 23 ng/ml. Full blood count, renal and liver function tests were normal. Her electrocardiogram (ECG) showed AF with ST segment elevation in leads V4-V6 (Figure 1).

A repeat ECG 30 minutes later showed similar findings (Figure 2).

The case was discussed with the regional cardiac center for consideration of emergent coronary angiogram +/- angioplasty; however, the on-call cardiologist advised echocardiography and management in the local hospital for suspected acute myocardial infarction.

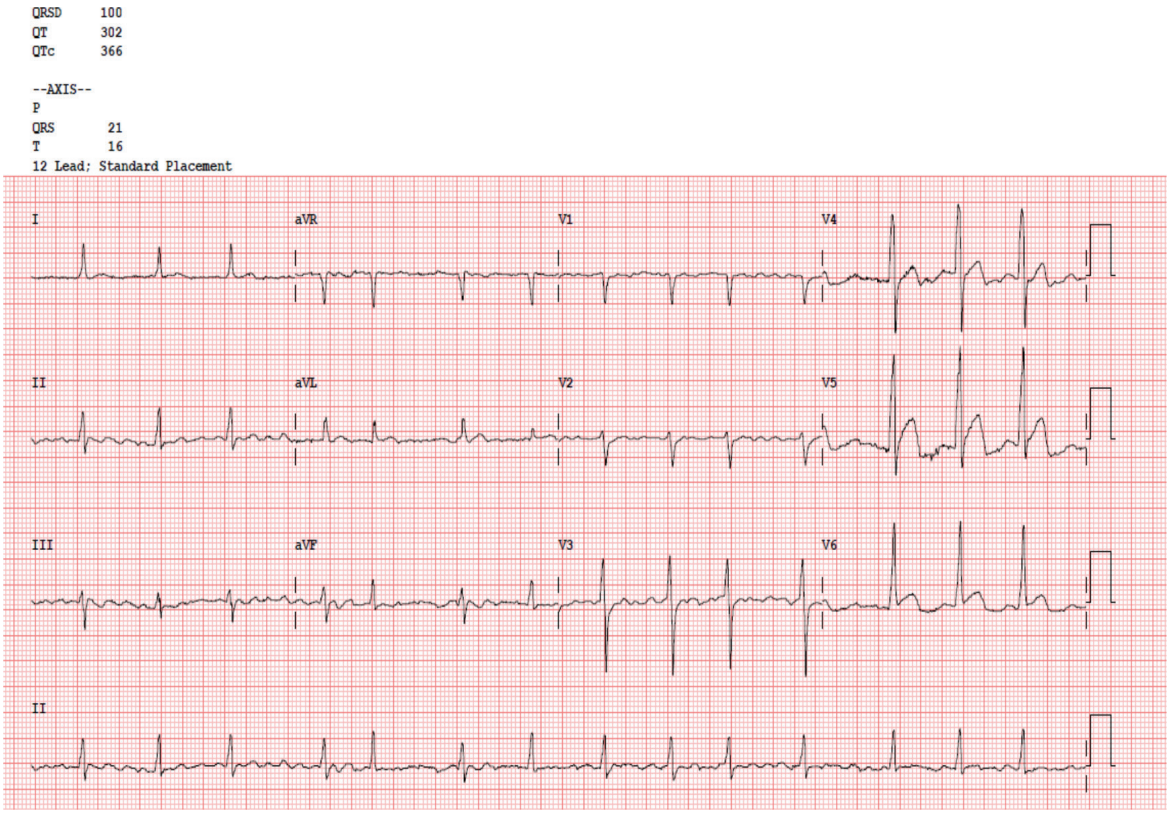

Figure 1. *ECG on presentation showing AF with ST segment elevation in leads V4-V6.*

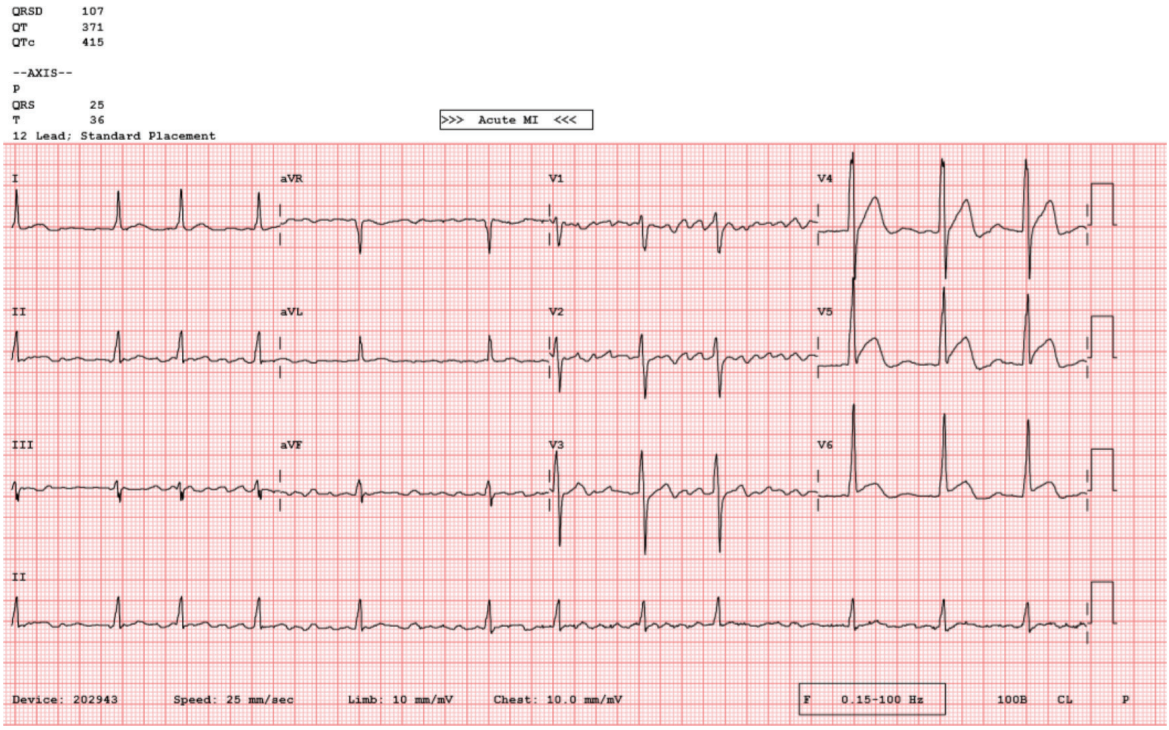

Figure 2. *Repeat ECG done 30 minutes after initial ECG which showed similar findings of AF and ST segment elevation in leads V4-V6.*

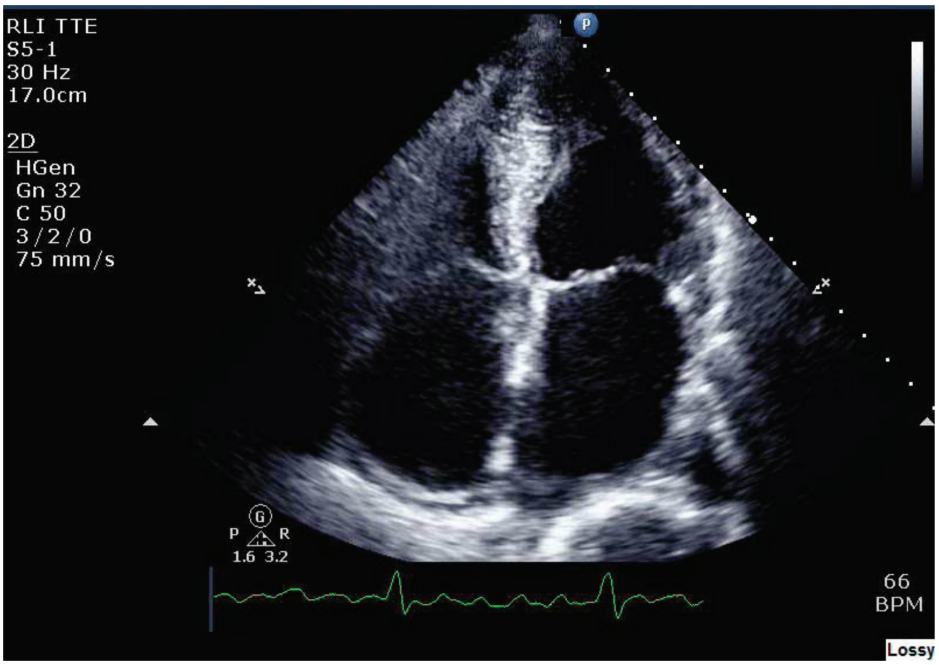

Figure 3. *Transthoracic echocardiogram four chamber view showing hypertrophy of the apicose ptal region.*

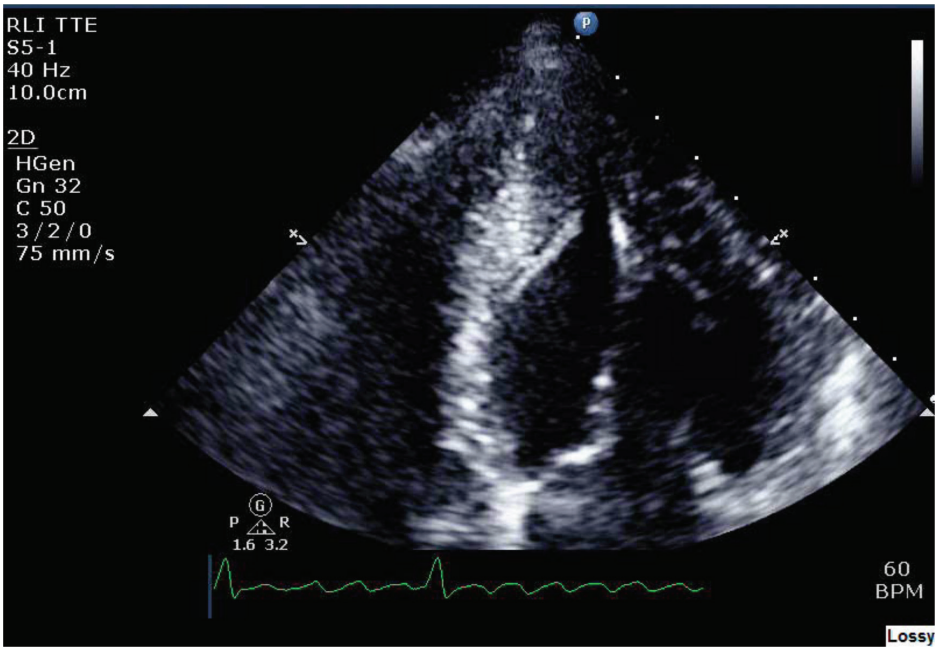

Figure 4. *Transthoracic echocardiogram focused on left ventricle:"ace of spades" appearance.*

A few hours later, she had a transthoracic echocardiogram, which showed a severely hypertrophied apicoseptal wall segment, abnormal left ventricular diastolic relaxation filling pattern, and a left ventricular ejection fraction of 65% (Figure 3 and 4). There were no doppler features of left ventricular outflow tract obstruction.

The next day, an ECG from her General Practice (GP) records which was done 14 months ago was obtained and it showed pre-existing lateral ST segment abnormalities, especially in leads V5-V6 (Figure 5).

She was diagnosed with "syncope induced by postural hypotension and apical hypertrophic cardiomyopathy". She was started on bisoprolol 2.5 mg once daily and an outpatient exercise tolerance test to investigate for induced arrhythmias was arranged. Genetic testing for HCM was also discussed as well as the importance of family screening. An outpatient cardiac magnetic resonance imaging (MRI) was also considered. However, the patient was admitted with significant morbidity 3 weeks later after presenting with right lower limb weakness and was

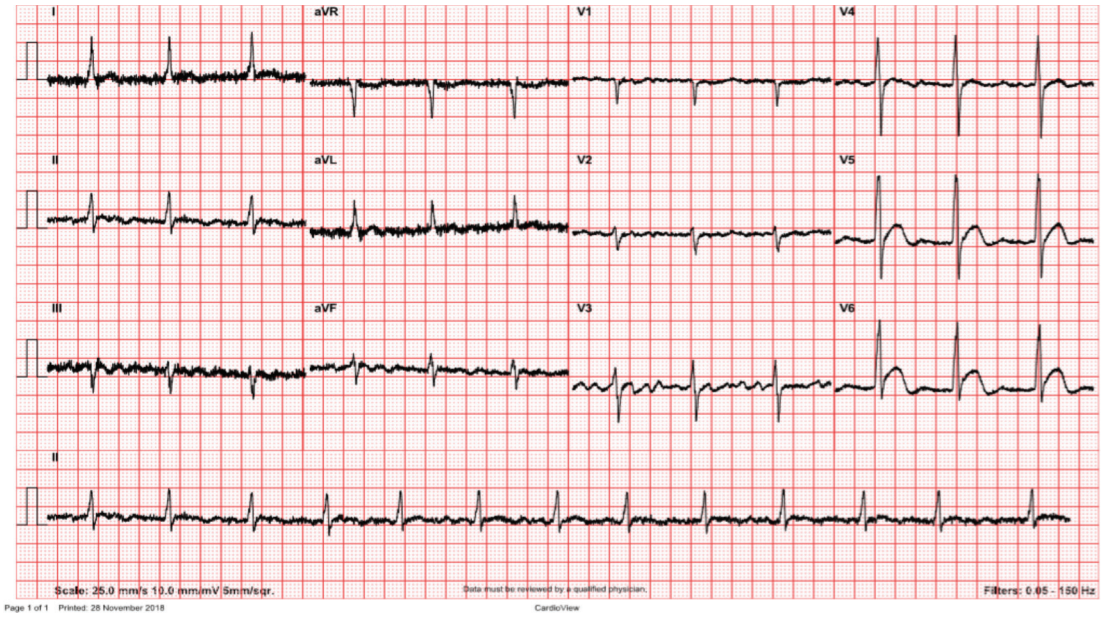

Figure 5. *Previous ECG done by her GP 14 months ago showed pre-existing ST elevation in V5-V6.*

diagnosed with a stroke. Her functional status declined significantly thereafter, and the previously scheduled outpatient investigations were postponed indefinitely.

Discussion

In majority of patients, AHCM is asymptomatic. However, some patients may develop angina, myocardial infarction, presyncope, syncope, heart failure, and arrhythmias such as atrial or ventricular fibrillation [3].

AHCM is caused by autosomal dominant mutations in genes that encode sarcomeric proteins particularly the sarcomere gene cardiac α-actin gene (ACTC) 1 (actin, alpha, cardiac muscle 1) - Glu101Lys missense mutation [4]. Although it is often suggested that the gold standard investigation for diagnosing HCM is cardiac magnetic resonance imaging [5], echocardiography is the most used tool. The American Heart Association/American College of Cardiology 2020 guidelines for the diagnosis and management of adult patients with hypertrophic cardiomyopathy advised that the clinical diagnosis of HCM can be established with 2D echocardiography or cardiovascular magnetic resonance [6].

Electrocardiographic abnormalities are common and well documented in AHCM. In general, the ECG features of AHCM are giant inverted T-waves, ST segment depression in leads II, III, aVF and V4-V6 [7]. Increased S-wave voltage in lead V1 + R-wave voltage in lead V5 up to 8.1 mv in addition to T-wave inversion has also been described [8]. There can be variations in these features with time as the highest R-wave amplitude in the precordial leads may remain stable, increase, or decrease greatly [9]. Newly developed left ventricular hypertrophy with giant T-wave inversion with the tallest R-wave in lead V4 may also be seen [10]. Evolution of the position of giant T-waves can transit between negative and positive directions over time [11]. This transition may also involve evolution from normal T-waves into negative T-waves and this process may occur rapidly or over a period of several years [12].

This particular patient did not have a past history of myocardial infarction; however, the authors are aware left ventricular aneurysms can cause ECG abnormalities such as ST Segment Elevation (STE), mostly with concave morphology, in leads I, aVL and V1-V6 [13]. The presence of ST segment elevation in the context of AHCM and in the absence of myocardial ischemia is rare. Some authors have investigated this relationship. Ozeke et al. [14] found an association between ST segment elevation in V4-V6 alongside giant T-wave inversion with the presence of apical aneurysm in individuals with HCM.

Maron et al. [15] found ST segment elevation and inverted T-waves in the lateral leads in 13 of 28 patients with apical aneurysm. There were two patients with ST elevation in V4-V6 in the absence of apical aneurysm [16]. One of these patients did not have a cardiac MRI [17] as in our case presentation.

Conclusion

It is useful for the clinician to evaluate the significance of any of these ECG findings when evaluating a patient with an abnormal ECG and syncope. While a careful evaluation for risk factors for coronary heart disease is essential, the clinician should also entertain the possibility of structural heart disease and as this case depicts, AHCM should be considered. Simple steps such as reviewing older ECGs, if available, is also often very helpful. It is important to emphasize the initial consideration and exclusion of life-threatening diagnosis such as ST segment elevation myocardial infarction and common clinical sequelae

What is new?

AHCM is known to present with certain ECG which have been well documented. This case report, however, presents a rare combination of lateral ST segment elevation secondary to AHCM with the absence of the aforementioned characteristic ECG findings.

such as apical aneurysm. Multi-modal imaging should be applied when the diagnosis is not clear-cut.

List of Abbreviations

AF	Atrial fibrillation
AHCM	Apical hypertrophic cardiomyopathy
ECG	Electrocardiogram
GP	General practitioner
MRI	Magnetic resonance imaging

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent for publication

The patient gave a written consent for the publication of this case report.

Ethical approval

Our institution does not require ethical approval for anonymized case reports.

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Summary of the case

1	**Patient details**	Female, 77 years old
2	**Symptoms**	Syncope
3	**Final diagnosis**	Syncope secondary to AHCM
4	**Clinical procedures**	Echocardiogram
5	**Clinical specialty**	Cardiology
6	**Interesting features**	Lateral ST segment elevation on ECG secondary to AHCM