

## CASE REPORT

# An unusual ST-segment elevation: Apical hypertrophic cardiomyopathy shows the ace up its sleeve

Francesco de Santis, Amedeo Pergolini, Giordano Zampi\*, Gaetano Pero, Paolo Giuseppe Pino, Giovanni Minardi

Department of Cardiovascular Science, "S. Camillo-Forlanini" Hospital, Rome, Italy

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### KEYWORDS

Electrocardiogram;  
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**Abstract** Apical hypertrophic cardiomyopathy is part of the broad clinical and morphologic spectrum of hypertrophic cardiomyopathy. We report a patient with electrocardiographic abnormalities in whom acute coronary syndrome was excluded and apical hypertrophic cardiomyopathy was demonstrated by careful differential diagnosis.

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### PALAVRAS-CHAVE

Eletrocardiograma;  
Miocardiopatia  
hipertrofica;  
Cardiopatias  
congenitas

**Elevação do segment St pouco usual: cardiomiopatia hipertrófica apical mostra o às na manga**

**Resumo** A miocardiopatia hipertrófica apical faz parte do amplo espectro clínico e morfológico da miocardiopatia hipertrófica. Nós reportamos um doente com alterações electrocardiográficas no qual foi excluída uma síndrome coronária aguda e foi demonstrada uma miocardiopatia hipertrófica apical usando um processo correto de diagnóstico diferencial.

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## Introduction

Apical hypertrophic cardiomyopathy (AHCM) is part of the broad clinical and morphologic spectrum of hypertrophic cardiomyopathy (HCM).<sup>1</sup> Cases of AHCM with and without apical aneurysm mimicking myocardial infarction have

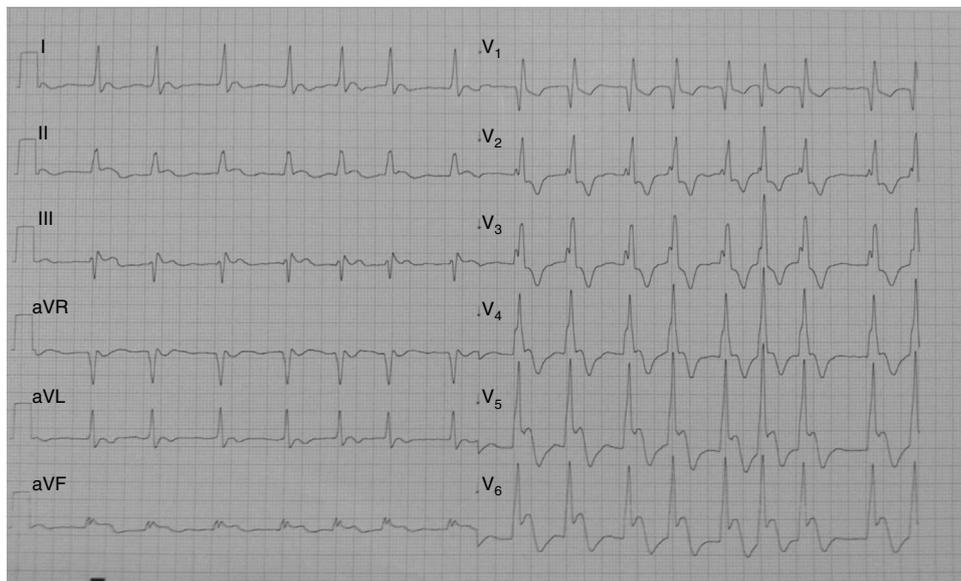
occasionally been reported.<sup>2-4</sup> We report a patient with abnormalities on the electrocardiogram (ECG) in whom acute coronary syndrome (ACS) was excluded and AHCM was demonstrated by careful differential diagnosis.

## Case report

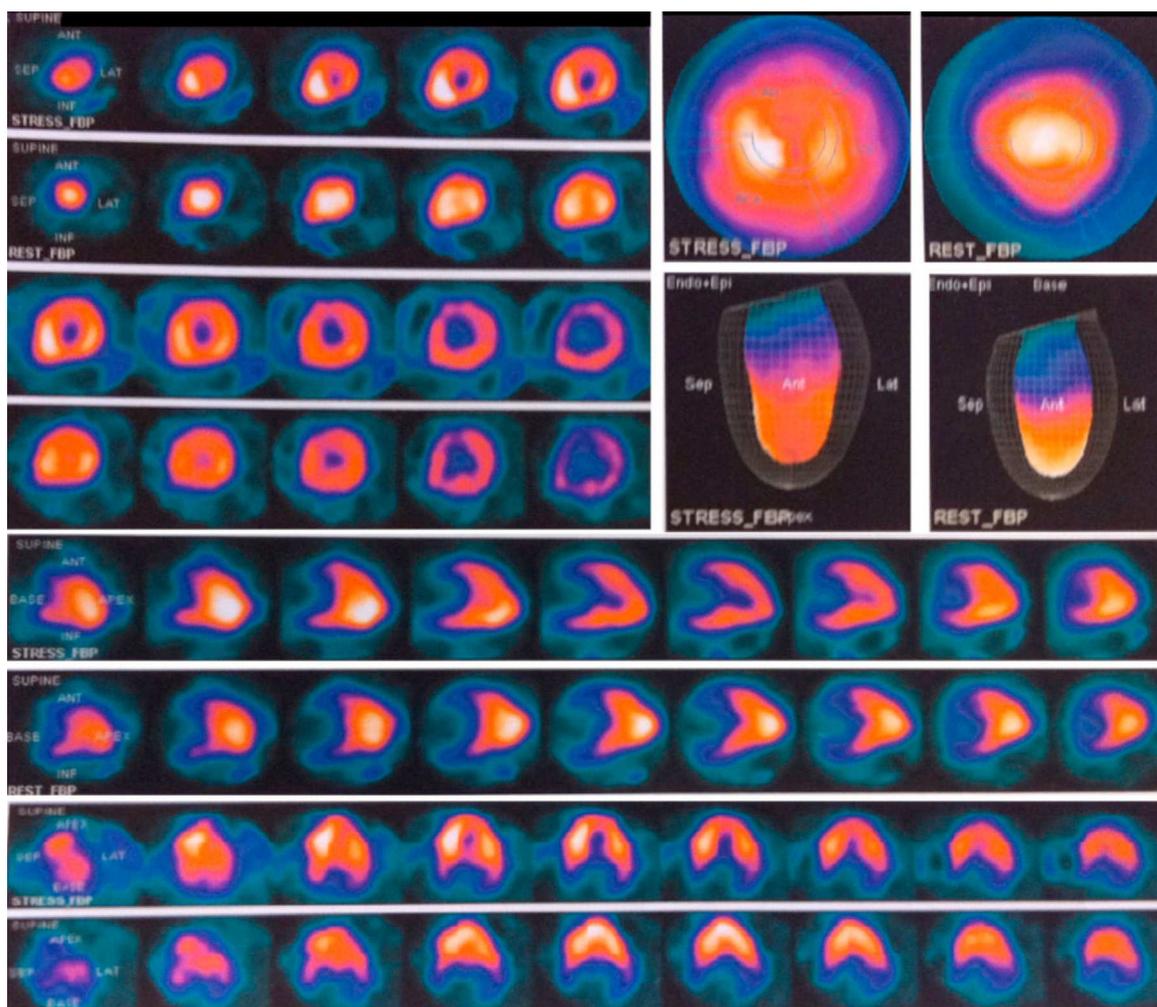
A 77-year-old Caucasian woman was examined by a primary care physician because of dyspnea and atypical chest pain. The ECG showed atrial fibrillation, mean heart rate of

\* Corresponding author.

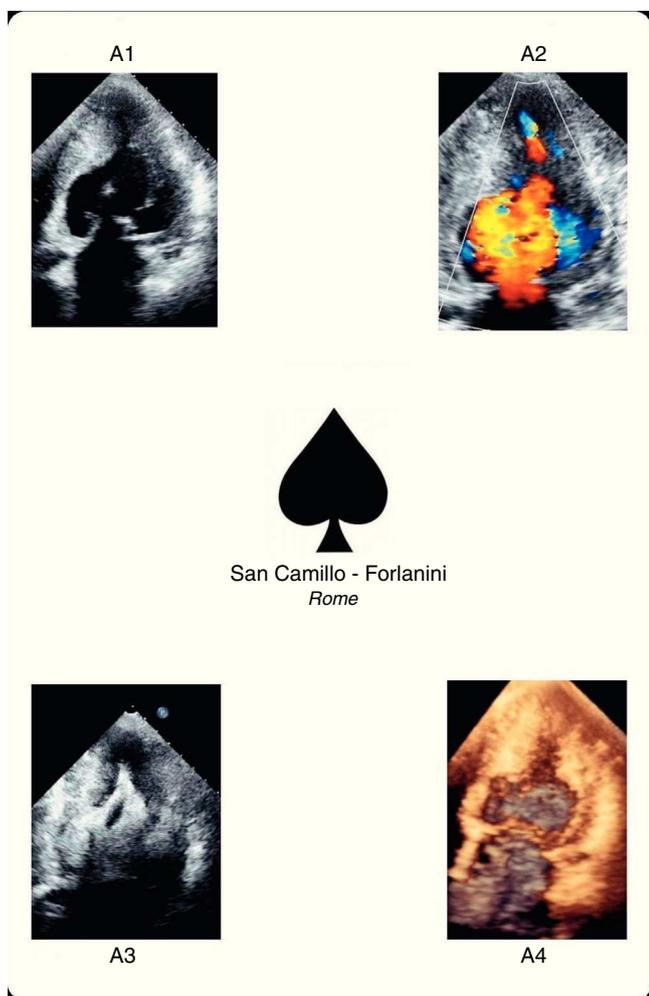
E-mail address: giordano.zampi@alice.it (G. Zampi).



**Figure 1** The ECG shows an unusual 4-mm convex ST segment elevation in C4–C6 with biphasic T waves.

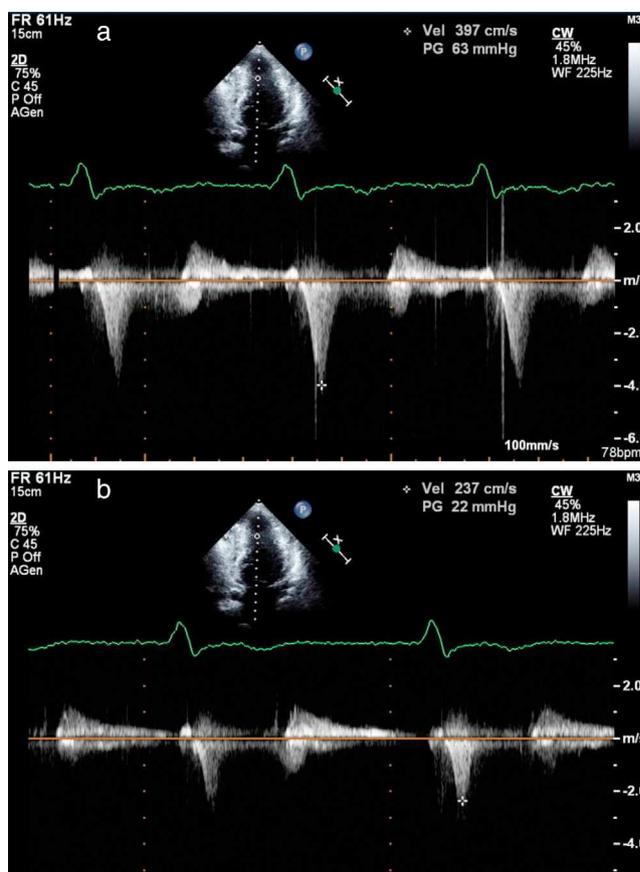


**Figure 2** Rest-stress myocardial perfusion scintigraphy revealing reduced left ventricular chamber volume suggestive of hypertrophic cardiomyopathy without signs of ischemia or previous myocardial necrosis.



**Figure 3** Apical hypertrophic cardiomyopathy shows the ace up its sleeve. A1: two-dimensional transthoracic echocardiography; A2: color Doppler transthoracic echocardiography; A3: contrast-enhanced transthoracic echocardiography; A4: real-time three-dimensional reconstruction.

85 bpm, right bundle branch block and 4-mm convex ST segment elevation in C4-C6 with biphasic T waves (Figure 1). Due to this atypical ECG, similar to a previous one performed two years before, rest-stress myocardial perfusion scintigraphy was prescribed. This revealed a reduction of left ventricular chamber volume suggestive of HCM without signs of ischemia or previous myocardial necrosis (Figure 2). The patient was referred to our echo lab in order to complete the diagnostic procedure. Transthoracic echocardiography (TTE) demonstrated AHCM, showing left ventricular (LV) apical hypertrophy with the typical ace of spades shape (Figure 3A1). The ace of spades was therefore studied in depth using color Doppler (Figure 3A2, Video 1), Sonovue contrast TTE (Figure 3A3, Video 2) and real-time three-dimensional echocardiography (Figure 3A4, Video 3). A small area of turbulent flow and a mid-ventricular gradient of about 63 mmHg were found (Figure 4a). In order to evaluate possible modification of the mid-ventricular gradient, an intravenous bolus of atenolol (3 mg) was administered. A significant decrease of about 40 mmHg was observed while the ECG was unchanged (Figure 4b).



**Figure 4** Transthoracic echocardiography, apical 4-chamber view showing (a) a mid-ventricular gradient of about 63 mmHg and (b) a significant decrease in the gradient after administration of an intravenous bolus of atenolol.

Beta-blocker therapy was prescribed indefinitely. At six-month follow-up, the patient was asymptomatic and in good clinical condition.

### Discussion

AHCM is characterized by segmental hypertrophy confined to the distal portion of the LV chamber. The prevalence of AHCM among HCM patients is 15% in Japan, whereas in the USA it is only 3%. Among AHCM patients 54% are symptomatic, the most common symptoms being chest pain, palpitations, dyspnea and syncope. This condition may cause various complications including atrial fibrillation, acute myocardial infarction, embolic events, ventricular fibrillation, congestive heart failure, apical aneurysm and cardiac arrest. Moreover, it may mimic other conditions, such as cardiac tumors, LV apical thrombus, isolated ventricular non-compaction, endomyocardial fibrosis and coronary artery disease. The most common ECG findings, seen in about 93% of patients, are negative T waves in the precordial leads (>10 mm deep in 47%), followed by signs of LV hypertrophy in 63% of cases. TTE usually shows LV apical hypertrophy and is thus usually the standard diagnostic tool for AHCM. Symptomatic patients may be treated with verapamil, beta-blockers and antiarrhythmic drugs, all of which may improve the signs and symptoms of this condition.

In conclusion, AHCM is a clinical condition that can cause various complications and simulate ACS. Its diagnosis enables ACS to be excluded in patients with atypical ST-segment elevation. TTE is usually sufficient to make the diagnosis, enabling appropriate medical treatment.

### Ethical disclosures

**Protection of human and animal subjects.** The authors declare that no experiments were performed on humans or animals for this study.

**Confidentiality of data.** The authors declare that no patient data appear in this article.

**Right to privacy and informed consent.** The authors declare that no patient data appear in this article.

### Conflicts of interest

The authors have no conflicts of interest to declare.

### Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at [doi:10.1016/j.repc.2012.11.013](https://doi.org/10.1016/j.repc.2012.11.013).

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