Heart Science and Heart Surgery Journal

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Case Report *Volume 1; Issue 1*

Apical Hypertrophic Cardiomyopathy: When the ECG changes?

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Received Date: April 26, 2018; **Published Date:** May 21, 2018

Abstract

Hypertrophic cardiomyopathy is a relatively common form of inherited cardiac disease. Apart from the usually seen septal form of asymmetric hypertrophy, there is other less frequent and uncommon variants of hypertrophic cardiomyopathy. We would like to share one such presentation where the patient was diagnosed with an apical hypertrophic pattern of hypertrophic cardiomyopathy.

Keywords: Cardiomyopathy; Electrocardiographic

Abbreviations: HCM: Hypertrophic Cardiomyopathy; ECG: Electrocardiographic; BNP: B-type Natriuretic Peptide; CMR: Cardiac Magnetic Resonance

Introduction

Apical hypertrophic cardiomyopathy (HCM) is uncommon in North America and is usually discovered incidentally during the routine medical assessment in elderly patients. The electrocardiographic (ECG) findings are described as typical symmetric deep T wave inversion and generally this diagnosis portends a good prognosis. We present a patient with apical variant hypertrophic cardiomyopathy with an unexpected ECG finding suggestive of ischemic myocardial injury or pericardial injury.

Case

An 89-year-old female with a remote history of heart failure some twenty years earlier, was admitted to the cardiology service for syncope. The physical examination was unremarkable and basic laboratory data including complete blood count, electrolyte panel, renal, liver function and troponin levels were normal. The serum B-type natriuretic peptide (BNP) at 262 pg/ml (normal < 37 pg/ml). A 12 lead electrocardiogram revealed markedly abnormal ST and T wave changes in the precordial leads suggestive of acute myocardial or pericardial injury (Figure 1).

A transthoracic echocardiogram showed findings consistent with an apical variant of hypertrophic cardiomyopathy and an apical pouch which was a kinetic (Figures 2,3 & Videos 1,2). Because of the ECG finding and associated regional wall motion abnormalities the patient underwent a left heart catheterization that showed no obstructive epicardial coronary artery disease. One week later, the ECG had remained unchanged. Meanwhile, the telemetry monitoring showed asymptomatic episodes of nonsustained monomorphic ventricular tachycardia of 3-12 beats duration at a rate of 150-160 bpm.

Citation: Mookadam F, et al. Apical Hypertrophic Cardiomyopathy: When the ECG changes?. Heart Sci Heart Surg J 2018, 1(1): 180001.

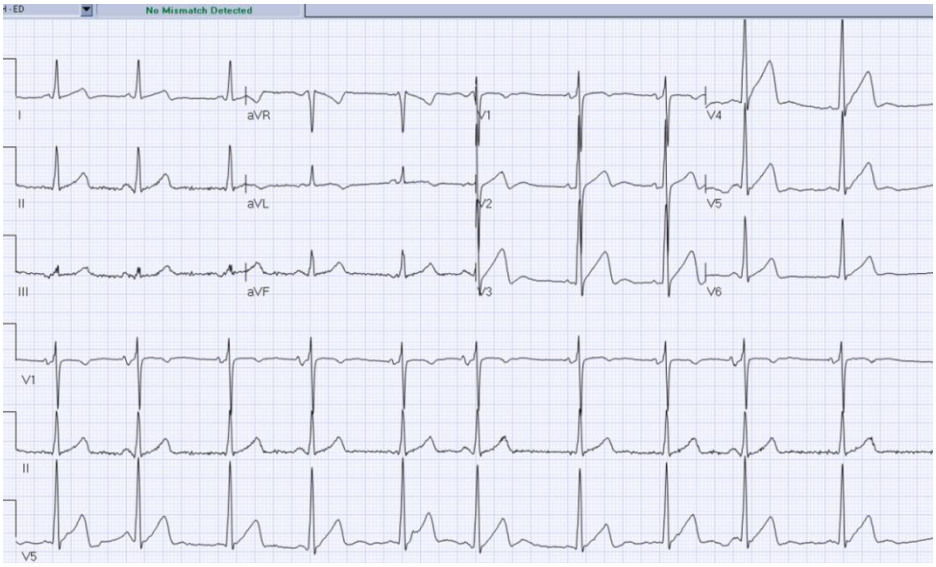

Figure 1: Patient's presenting ECG showing asymmetric up sloping precordial ST elevation.

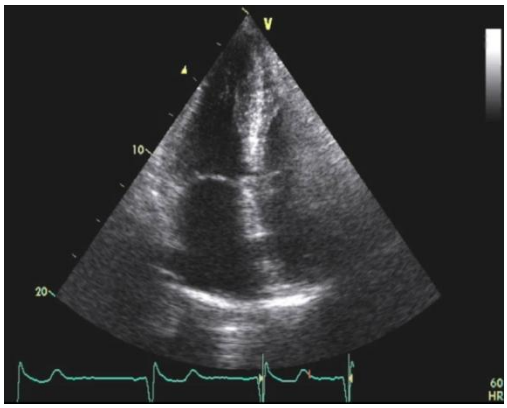

Figure 2: 4 chamber 2D echocardiographic view, in the Mayo format (with left sided structures to the left of the picture), showing apical hypertrophy with possible apical pouch.

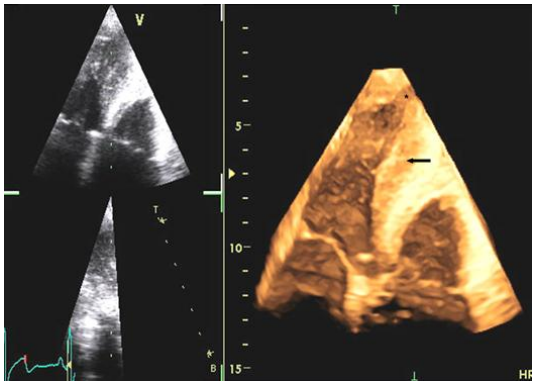

Figure 3: 4 chamber 3D echocardiographic view, in the Mayo format, showing LV apical hypertrophy (Arrow) and presence apical pouch (Asterisk).

A cardiac magnetic resonance (CMR) study confirmed the echocardiographic findings of apical HCM with an apical pouch and no thrombus (Figure 4, Video 3). The apical septum measured 2.1 cm in thickness. The apical pouch with adjacent delayed enhancement was estimated at less than 3% of the myocardial volume.

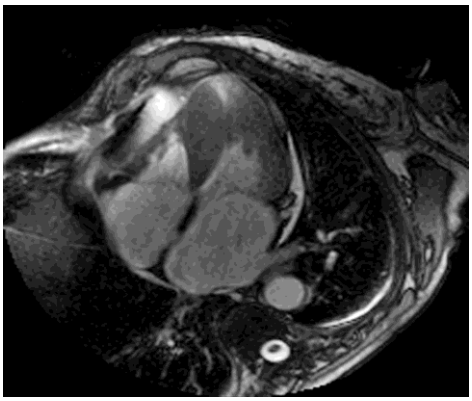

Figure 4: 4 Chamber T2 weighted cardiac magnetic resonance image revealing presence of apical hypertrophy with apical pouch.

Discussion

Apical hypertrophy variant of HCM is relatively rare in North America (3%-11% of all HCM cases) but can constitute up to 25% of all cases of HCM in Japan [1,2]. This variant of HCM is associated with a benign prognosis in both Japanese and North American patient populations; presumably due to the absence of dynamic obstructive physiology [2].

The classic ECG abnormality related to apical variant HCM is the presence of giant negative T waves seen in the precordial leads (Figure 5) [1]. Our patient's ECG actually showed asymmetric upsloping precordial ST elevation. The loss of T wave inversion and their replacement with ST elevation on ECG in patients with apical variant HCM has been previously described. Webb et al. [3] followed 26 patients (mean age 46 years) for a mean follow up period of 7.3 ± 6.2 years. All 26 patients had symmetric T wave inversions with 14 patients showing "giant T wave" inversions characteristic of apical HCM. On follow up only one patient showed loss of precordial T wave inversion and this was associated with the formation of an apical aneurysm which was ascribed to myocardial infraction and aneurysm formation [3]. Apical aneurysms in the setting of HCM are being increasingly recognized as an uncommon but important subgroup with increased mortality in the overall HCM population. The causes for apical aneurysm formation remain unclear but may include myocardial infarction related to increased myocardial demand with or without CAD, genetic factors and increased apical wall stress due to mid-cavitary LV obstruction [4]. It is associated with adverse clinical outcome which seems related to myocardial scarring that develops around the aneurysm. An evolving ECG from typical deep symmetrical T-wave inversion to an atypical appearance should raise the suspicion of new apical aneurysm formation. This followed by timely investigation with echocardiography or CMR may help in early diagnosis and institution of ICD placement or anticoagulation to prevent some of the complications associated with this condition.

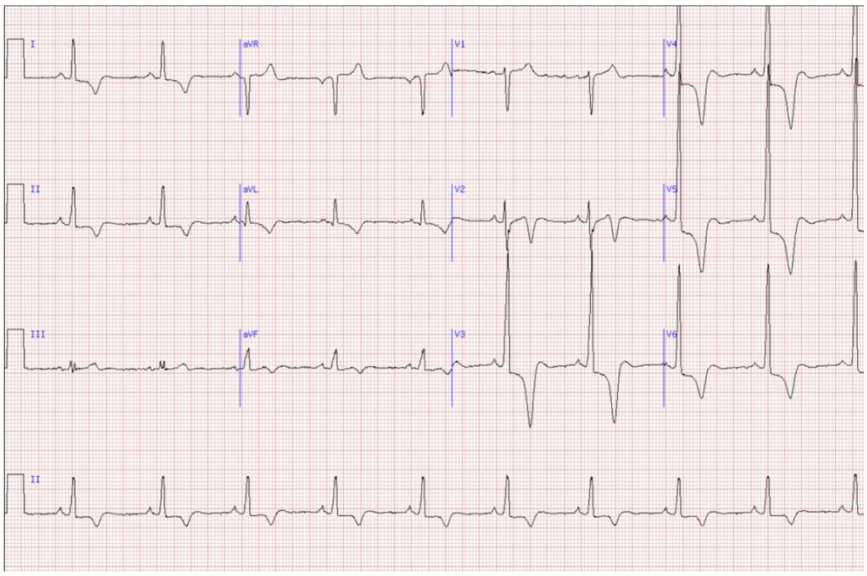

Figure 5: Typical ECG in patients with apical hypertrophy with deep symmetric inverted T waves.

Acknowledgement

The authors would like to thank Joan L Lusk, R.D.C.S, for her efforts in obtaining optimal echocardiographic images.

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