Making Waves: A Case of the Apical Form of Hypertrophic Cardiomyopathy

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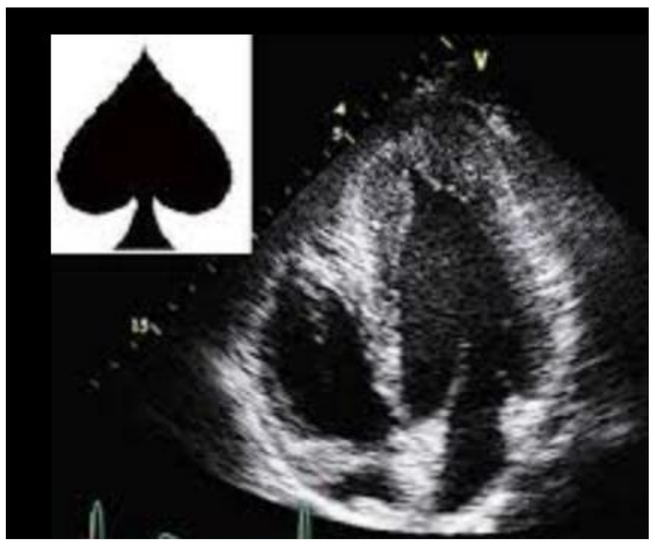
Introduction

Hypertrophic cardiomyopathy describes a group of myocardial diseases characterized by left ventricular hypertrophy (LVH). In these conditions, LVH arises due to alterations in cardiac myofiber organization rather than cardiac loading conditions.[2]

The prevalence in the general population is estimated to be 1:500-3,000.[2-4] An uncommon type predominantly involves the apical region of the myocardium, accounting for 1-10% of hypertrophic cardiomyopathies in non-Asian populations.[2] Although this is an uncommon hypertrophic variant, it can be alarming for a provider to encounter its dramatic EKG changes which can mimic findings of acute coronary syndrome. We present a case of a 75-year old man found to have apical hypertrophic cardiomyopathy (ApHCM) after presenting with atypical chest pain.

On echocardiogram and cardiac MRI imaging, ApHCM is characterized by an "ace-of-spades" LV appearance from localized apical hypertrophy with a wall thickness > 15mm (Figure 1).[3] On EKG, giant T-wave inversions are commonly seen in the precordial leads.[3]

Figure 1. Four-view transthoracic echocardiogram image demonstrating the ace-of-spades appearance with massive hypertrophy of the LV apex with relative sparing of the base.



Note. By Ahmadi M, Khameneh Bagheri R, 2017, licensed under CC 3.0.[1]

Case Description

A 75-year old man with a history of resistant hypertension presented to the ED with several hours of dull left parasternal chest pain, precipitated with ambulation and persisting at rest. There was no diaphoresis, nausea, vomiting, dyspnea, palpitations, or syncope. He denied a history of angina or family history of cardiac conditions or sudden death. He had no functional limitations at home.

On initial assessment, the patient's vitals signs were stable. EKG was remarkable for LVH by voltage criteria and marked T-wave inversions in the lateral and precordial leads (Figure 2). Further chart review revealed that EKG findings were consistent with those seen in multiple EKGs dating back 16 years (Figure 3) with initial involvement of only lateral leads. Serial high-sensitivity troponins were negative. He was given three sublingual nitroglycerin tablets and IV hydromorphone with resolution of his chest pain for the rest of his course.

Transthoracic echocardiogram showed substantially increased mid and apical LV wall thickness with an LV end diastolic diameter of 44 mm. The patient had normal systolic function and no regional wall abnormalities. An angiogram demonstrated non-obstructive coronary artery disease with 50% stenosis of the mid LAD. He was discharged with initiation of aspirin, statin, and continuation of his antihypertensive regimen including beta blocker therapy. He followed up with outpatient cardiology for evaluation for ICD placement.

Figure 2. Admission ECG with LVH and diffuse and marked T-wave inversions in lateral and precordial leads.

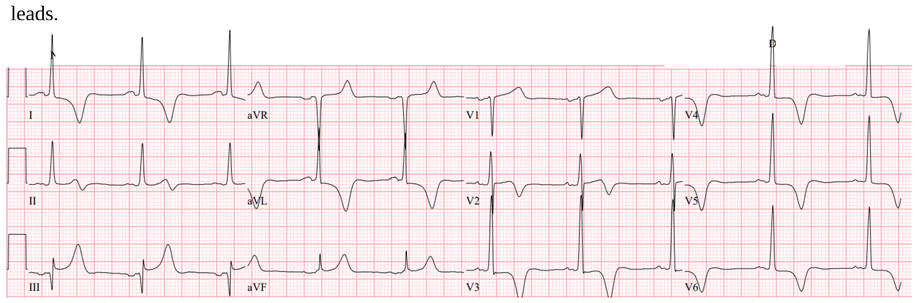
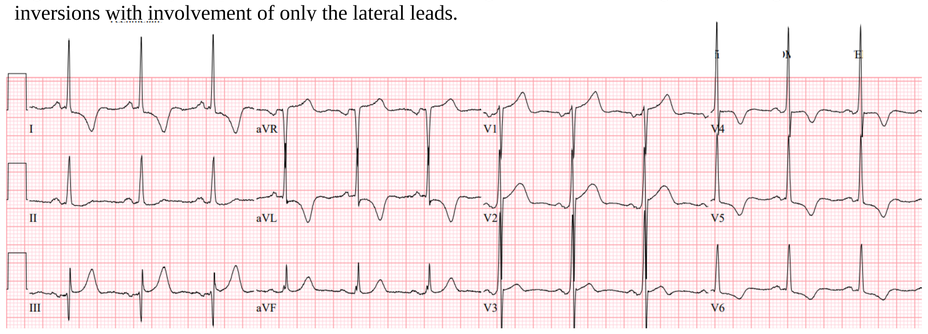


Figure 3. EKG 16 years prior to admission demonstrating LVH and lesser magnitude of large T-wave inversions with involvement of only the lateral leads.



Discussion

Clinical presentation of ApHCM is variable ranging from a complete lack of symptoms to nonspecific symptoms including atypical chest pain, dyspnea with exertion, and syncope in association with characteristic echo and/or EKG findings.[2] Management is similar to that of classic HCM and includes Holter monitoring and beta blockers.[5]

This case shows that, while his history of resistant hypertension certainly influenced his LVH, it is unable to account for the degree and pattern of hypertrophy alone and underscores the importance of having comprehensive awareness of this condition's characteristic EKG and echocardiography findings in order to ensure timely diagnosis and treatment. Perhaps most notably, the case highlights the association of marked precordial T wave inversions with atypical chest pain in ApHCM, overall demonstrating the importance of recognizing this condition as a potential mimic of the acute coronary syndrome.

References

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