

Stuck in "Apical": A Case of Apical Variant HCM presenting as NSTEMI

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INTRODUCTION

- Hypertrophic cardiomyopathy (HCM) is seen in 1/500 people.
- It is usually asymptomatic but can cause dyspnea on exertion, fatigue, angina, and syncope.
- We present a case of an apical variant HCM in a patient who presented with atypical angina thought to be related to a non-ST elevation myocardial infarction (NSTEMI).

CASE DESCRIPTION

A 43-year-old physically active male with no significant medical history presented with chest pain. He reported several hours of progressively worsening chest pain at rest that was substernal, moderate in severity, waxing and waning in nature and without associated diaphoresis. He denied preceding symptoms and previous episodes of chest pain. However, he reported an unusual two-week span of headaches and neck pain. Family history was negative for sudden cardiac death, HCM, amyloidosis, or sarcoidosis but notable for age-related coronary artery disease and stroke in parents.

Vitals: Temp 97.2 °F, BP 132/74, HR 75,, RR 16, SpO2 100%

Physical Exam: Patient was alert and oriented. No evidence of JVD, leg edema, murmurs, rubs, gallops. Lungs were clear bilaterally.

Workup: Chest x-ray was unremarkable. Electrocardiogram (EKG) showed normal sinus rhythm, prominent inferolateral ST depressions, and deep T wave inversions in the precordial leads (Figure 1). High sensitivity troponins became flat around 360 ng/L. A CT head was also obtained on admission given headaches and T wave inversions with results not indicating any acute intracranial abnormalities.

Troponins HS (ng/L): 364 > 345 > 367.

Troponin I (ng/L): 279

CLINICAL COURSE

Given atypical chest pain, troponin elevation, and EKG changes, he was initially treated for NSTEMI with aspirin, clopidogrel, and heparin drip. He underwent a left heart catheterization which did not reveal any coronary disease, however ventriculography displayed an "ace of spade" appearance. Echocardiogram was obtained revealing apical hypertrophy (Figure 2) with an ejection fraction (EF) of 60-65% and no wall motion abnormalities. These findings were concerning for apical variant HCM and the patient was ultimately followed in the outpatient setting for further evaluation.

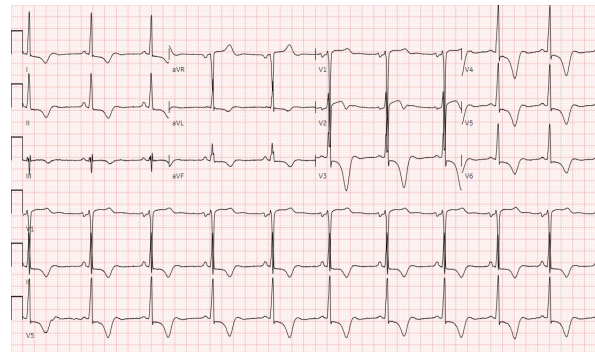


Figure 1. Nonspecific EKG that can be seen in NSTEMI or HCM

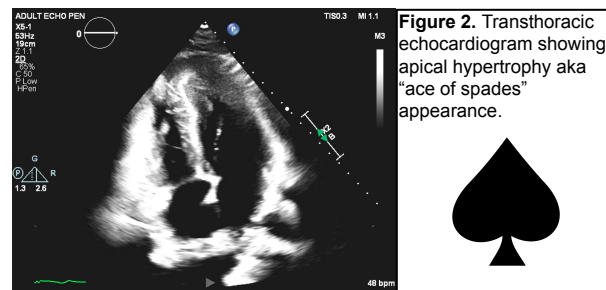


Figure 2. Transthoracic echocardiogram showing apical hypertrophy aka "ace of spades" appearance.

DISCUSSION

- Atypical chest pain with EKG changes showing deep T wave inversions in young patients should raise suspicion for HCM.
- Echocardiography should be utilized early to guide decision making, given that the treatment for one condition can potentially worsen the outcome of the other.
- For example, the use of nitroglycerin or diuretics in preload dependent states such as HCM with LVOT obstruction could lead to fatal complications such as sudden cardiac death, syncope, or shock.

CONCLUSION

- Differentiation between obstructive coronary disease and HCM in patients is crucial given that both disease processes can elicit symptoms of chest pain, dyspnea, lightheadedness, syncope, and palpitations.
- Early utilization with echocardiography to rule out wall motion abnormalities in the setting of atypical chest pain without a positive rise in troponin can prevent invasive testing such as coronary angiography.

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