CME Article

Electrocardiographical case. Asymptomatic patient with deep T-wave inversions

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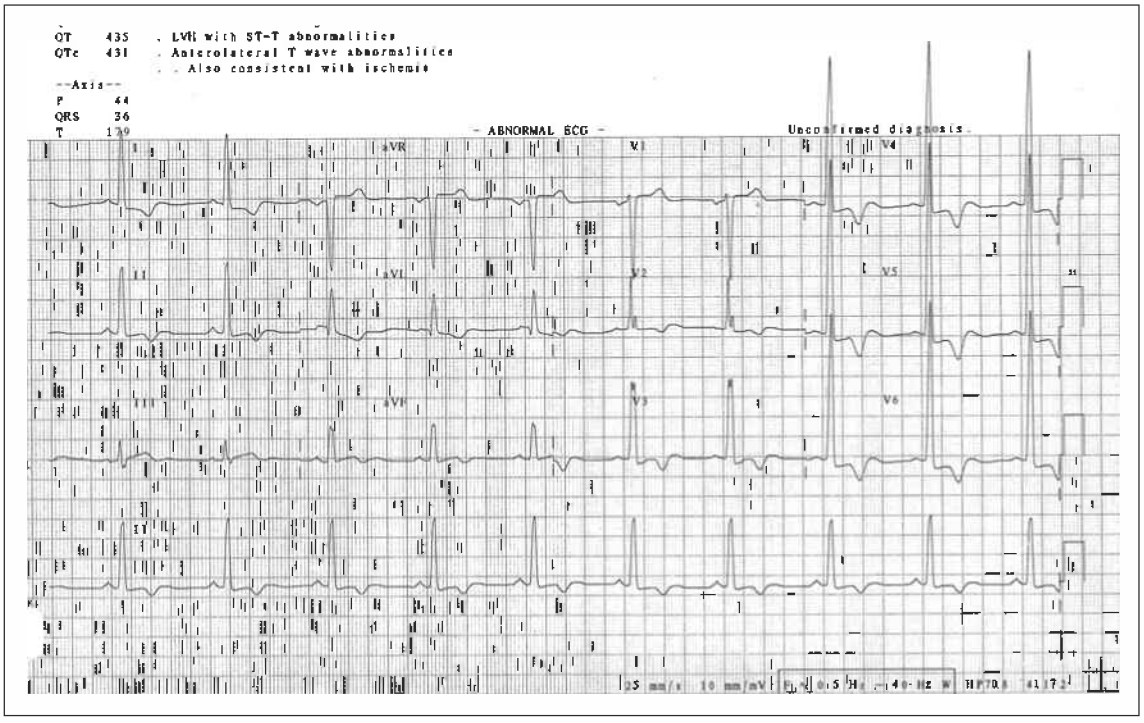

Fig. 1 12-lead electrocardiogram.

CLINICAL PRESENTATION

A 45-year-old man had an abnormal myocardial perfusion scan which showed mild ischaemia in the apex and distal inferior wall of the left ventricle. He was an ex-smoker, and had a past history of hyperlipidaemia. Otherwise, he had no complaints of chest pain, breathlessness or palpitations. Clinical examination was unremarkable. The 12-lead electrocardiogram (ECG) is shown in Fig. 1. What is the diagnosis?

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DIAGNOSIS

Apical hypertrophic cardiomyopathy.

ECG INTERPRETATION

The ECG showed sinus rhythm with QRS axis of +36° and absent septal Q waves. Increased QRS voltages are seen in leads V3–V6, ranging from 20 to 38 mm. Large negative T-waves are also noted in leads V3–V6, measuring up to 6 mm in depth (Fig.1). These ECG findings are characteristic of apical hypertrophic cardiomyopathy (HCM), where the apical segment potentials are most clearly reflected by large deep inverted T-waves in the precordial leads, particularly V4 and V5. Lesser degrees of T wave inversion and ST segment depression are the other most frequently noted abnormalities.

Many patients also have left ventricular hypertrophy by voltage criteria. Other electrographical abnormalities reported less frequently include left and right atrial enlargement, left axis deviation, and first-degree atrioventricular block. Prolongation of the QTc interval has also been noted.[1-5] Some important causes for deep T-wave inversion in the anterolateral precordial leads are listed in Table I.

Table I. Some important causes for T-wave inversion in the precordial leads.

Myocardial ischaemia
Myocardial infarction (Q-wave and non-Q wave)
Pericarditis
Acute myocarditis
Acute pulmonary embolism
Myocardial contusion (from trauma)
Central nervous system events e.g. subarachnoid haemorrhage
Idiopathic apical hypertrophy
Left ventricular hypertrophy with "strain"

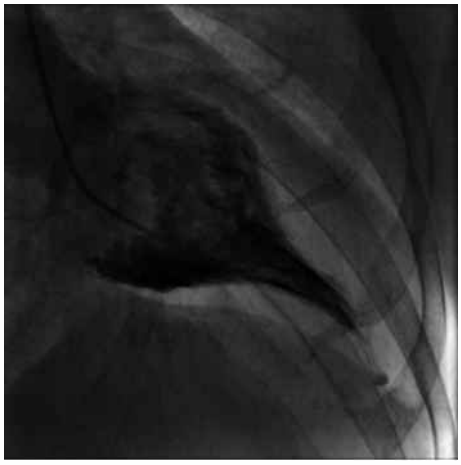

Fig. 2 Left ventriculogram shows obliteration of the apex during systole.

CLINICAL COURSE

A coronary angiogram was done, which revealed normal coronary arteries. Left ventriculogram showed thickening of the distal 1/3 and apex (Fig. 2). There was no significant intra-cavitary gradient. A transthoracic echocardiogram showed thickening of the left ventricular apex with obliteration of the ventricular apex at end systole (Figs. 3a & b). The valves and the ejection fraction were normal.

This patient's ECG is classical for apical HCM. It is however important to exclude coronary artery disease, particularly in the presence of an abnormal myocardial perfusion scan. The diagnosis was made on the basis of the left ventriculogram and transthoracic echocardiogram findings. He was managed expectantly as he had no evidence of outflow tract obstruction and was asymptomatic.

DISCUSSION

Apical HCM is an uncommon variant with predominant involvement of the apex of the heart. This condition was first described in Japanese males in 1976. Apical HCM is distinctly uncommon in other parts of the world and probably constitutes 1%–2% of those with HCM. In

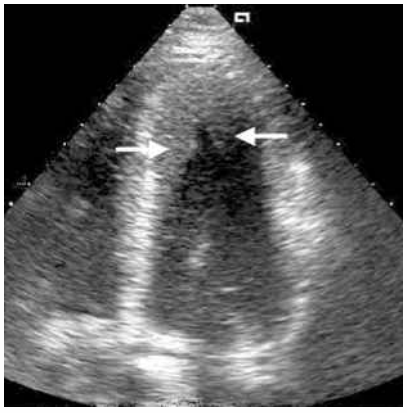

Fig. 3a US image (apical four chamber view) of the heart shows hypertrophy of the apical myocardium (white arrows).

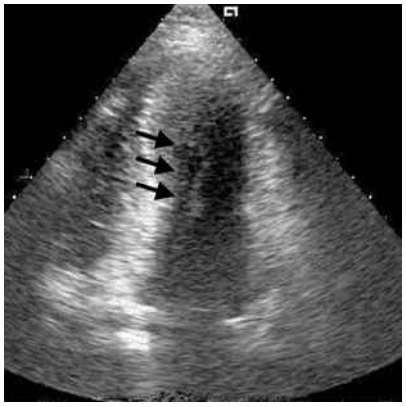

Fig. 3b US image shows obliteration of the ventricular apex during systole (black arrows).

Japan, this apical variant constitutes about 25% of patients with HCM.[1,6,7] Patients with apical HCM tend to have little or no symptoms. The most frequently-encountered symptom is atypical chest pain. Like classical HCM, they are prone to arrhythmias, either atrial or ventricular, and may present with palpitations or even syncope. Physical examination is of little use in apical HCM, which often has no murmur. Occasionally, the patient may have a left ventricular lift or loud fourth heart sound. This is in contrast to classical HCM, which has well-described characteristic physical findings.[3-5,8]

The diagnosis of apical HCM can be established with transthoracic echocardiography (Fig. 3a & b), which is the tool of choice in the diagnosis and evaluation of all types of HCM. Harmonic imaging or contrast echocardiogram imaging provides good delineation of the ventricular walls, in particular, the thick apex. If echocardiogram images are difficult to obtain or interpret, magnetic resonance (MR) imaging may occasionally be used to diagnose apical HCM, especially if the apex is difficult to visualise on transthoracic echocardiography.[3-5]

Cardiac catheterisation has some value in diagnosing HCM. Apical HCM has a distinctive left ventricular "ace of spades" appearance and obliteration of the apex during systole (Fig. 2). Its main role in HCM patients is to evaluate for concomitant coronary artery disease.[4] This is especially so in patients with coronary artery disease risk factors, or presenting with chest pain or exertional dyspnoea. Patients with HCM may have small vessel disease from increased collagen deposition and myocardial ischaemia due to myocardial oxygen supply and demand mismatch. This mismatch is driven primarily by the increased myocardial mass and not by any significant obstructive coronary artery disease. This may explain the finding of a reversible myocardial perfusion defect on radionuclide imaging with technetium in this patient even in the setting of angiographically-normal coronary arteries. However, advances in echocardiography imaging have made it the predominant means by which HCM is diagnosed.

Familial HCM occurs as an autosomal dominant Mendelian-inherited disease in about 50% of cases or can occur as spontaneous mutations. It is a genetically heterogeneous disease that can be caused by genetic defects in more than one locus. For apical HCM, the specific genetic mutations have, however, not been identified.[9] Apical HCM generally has a more benign course compared to other variants of HCM, and is usually not associated with any significant outflow tract obstruction, though like classical HCM, they are at a slightly increased risk of arrhythmias.[2,8,10-12] In these cases, it might be necessary to identify patients at risk of sudden death, and institute antiarrhythmic therapy or even an implantable cardioverter. In the asymptomatic patient, with no evidence of myocardial ischaemia or significant arrhythmia, no specific therapy is needed.

ABSTRACT

A 45-year-old man was found to have an abnormal myocardial perfusion scan. He was asymptomatic, with no chest pain, breathlessness or palpitations. Clinical examination was unremarkable. The 12-lead electrocardiography (ECG) showed increased QRS voltage in leads V3-V6, and deep T-wave inversions noted in leads V3-V6, with an absence of septal Q waves. These ECG features were characteristic of apical variant hypertrophic cardiomyopathy (HCM). He underwent a coronary angiogram that revealed normal coronary arteries, and a left ventriculogram which showed apical HCM. Transthoracic echocardiography further confirmed the diagnosis. No drug therapy was instituted as he was asymptomatic. Apical HCM is discussed.

Keywords: apical hypertrophy, hypertrophic cardiomyopathy, Japanese variant hypertrophic cardiomyopathy, T-wave inversion, transthoracic echocardiography

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 200706B)

	True	False
Question 1. T-wave inversion in the precordial leads can be found in the following:		
(a) Left ventricular hypertrophy.	☐	☐
(b) Duchenne muscular dystrophy.	☐	☐
(c) Subarachnoid haemorrhage.	☐	☐
(d) Proximal left anterior descending coronary artery occlusion.	☐	☐
Question 2. The following is true of apical hypertrophic cardiomyopathy:		
(a) It is more commonly seen in the Japanese population.	☐	☐
(b) Double or triple apical impulse is characteristic.	☐	☐
(c) It may be associated with serious arrhythmias.	☐	☐
(d) Cardiac murmur is common.	☐	☐
Question 3. Diagnosis of apical hypertrophic cardiomyopathy can be made with the following:		
(a) Transthoracic echocardiography.	☐	☐
(b) Cardiac MR imagimg.	☐	☐
(c) Contrast ventriculography.	☐	☐
(d) Electrocardiogram.	☐	☐
Question 4. Drug therapy for symptomatic apical hypertrophic cardiomyopathy in the absence of significant coronary artery disease may include:		
(a) Beta blockers.	☐	☐
(b) Nitrates.	☐	☐
(c) Aspirin.	☐	☐
(d) Calcium channel blockers.	☐	☐
Question 5. The following is true of apical hypertrophic cardiomyopathy:		
(a) It may be a cause of reversible or fixed myocardial defects on radionuclide imaging, even in the setting of normal coronary arteries.	☐	☐
(b) It occurs as autosomal dominant inherited disease in about 50% of cases.	☐	☐
(c) It carries a worse prognosis compared to other variants of hypertrophic cardiomyopathy.	☐	☐
(d) Cardiac defibrillator implantation is indicated in the presence of recurrent syncope and inducible ventricular arrhythmias during electrophysiological study.	☐	☐

Doctor's particulars:

Name in full: _____

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Email address: _____

SUBMISSION INSTRUCTIONS:
(1) Log on at the SMJ website: **www.sma.org.sg/cme/smj** and select the appropriate set of questions. (2) Select your answers and provide your name, email address and MCR number. Click on "Submit answers" to submit.

RESULTS:
(1) Answers will be published in the SMJ August 2007 issue. (2) The MCR numbers of successful candidates will be posted online at **www.sma.org.sg/cme/smj** by 15 August 2007. (3) All online submissions will receive an automatic email acknowledgment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.

Deadline for submission: (June 2007 SMJ 3B CME programme): 12 noon, 25 July 2007