Apical Hypertrophic Cardiomyopathy revisited

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Citation

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Abstract

A 43 year old gentlemen with h/o cocaine use presented to the ED with chest pain. EKG was consistent with left ventricular hypertrphy with acute ST-T changes. Troponin was elevated. CT chest and was negative for pulmonary embolism. Patient was taken for cardiac catheterization to rule out acute coronary syndrome. Left ventriculogram revealed severe hypertrophic cardiomyopathy with apical systolic cavity obliteration. Transthoracic echocardiogram revealed massive apical hypertrophic cardiomyopathy. Case discussion and review of literature and images are presented.

CASE

A 43 year old African American male with history of crack cocaine use presented to the Emergency room with chest pain and dyspnea of one day duration. The pain was central thoracic, episodic, unassociated with faintness or syncope. It was determined on the basis of the patient's history that there was an intermediate probability of ischemic pain.

Clinical exam revealed an Afro middle aged obese man who is in no acute distress, his pulse rate was 110, blood pressure was 110/68.The Jugular venous pressure was not elevated and he did not demonstrate the Bernheim phenomenon. Kussmaul sign was absent. The pulse was small and there was a prominent left ventricular thrust. There was no evidence of right ventricular enlargement. There was no pericardial friction rub.

The electrocardiogram showed sinus tachycardia, PR interval 140 ms, QT corrected of <400 ms, and QRS which had a normal duration and axis. There was a p wave > 2 mm. Giant inverted T waves 10-12 mm preceded by ST segment depression were present in precordial leads V3 to V6.

Cardiac enzymes showed troponin elevation at 1.00ng/mL. Electrolytes and blood counts were normal. Chest X ray showed no cardiomagely or venous congestion. There was no left atrial enlargement. There was no organomegaly in the abdomen, no ascites and no pedal edema. The CT angiogram of the chest was negative for pulmonary embolus.

The provisional diagnosis of acute coronary syndrome was made and the patient was referred for cardiac catheterization. Coronary Angiography was negative for obstructive coronary disease, while left ventriculography revealed severe hypertrophic cardiomyopathy with apical systolic obliteration of left ventricular cavity (Fig 2.), and impaired diastolic filling (Fig 3).

Figure 1

Figure 2. Obliteration of LV cavity during mid systole

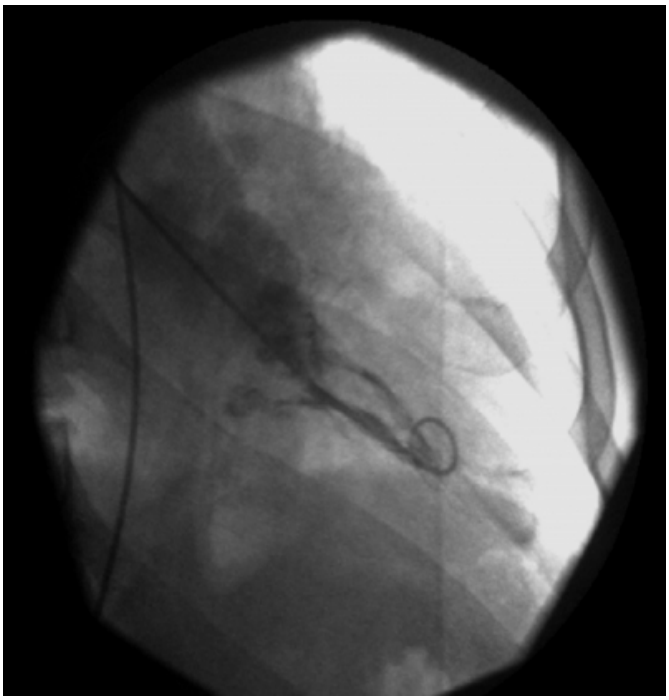
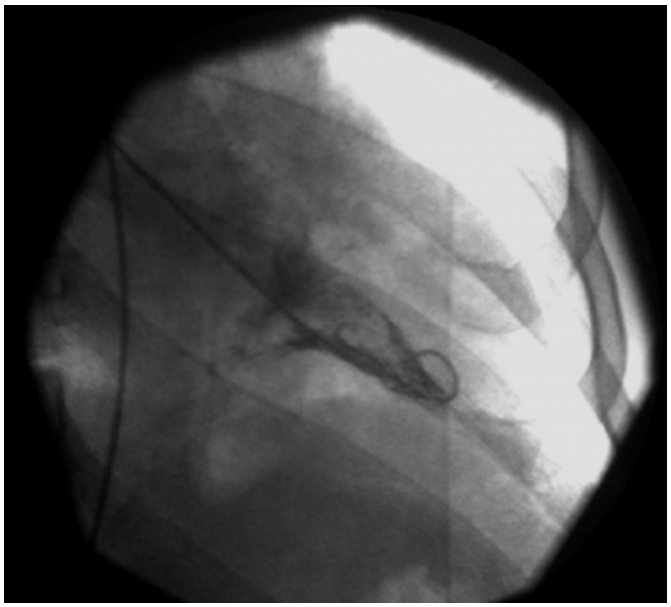


Figure 2

Figure 3 Impaired diastolic filling



Trans thoracic Echo revealed restrictive pattern of diastolic filling due to massive apical hypertrophy with. There was diffuse hypokinesis of the left ventricle. Left atrium was 4.7 cm. The septal wall thickness was 2.9 cm. Apical wall thickness was 4.5 cm. Ejection Fraction was 45 %.

DISCUSSION

Apical hypertrophic cardiomyopathy (APCM) is a less common variant of hypertrophic cardiomyopathy (HC) first reported in Japan by Sakamoto and then subsequently by Yamaguchi10.This condition is characterized by asymmetric myocardial hypertrophy of the apex of the left ventricle (LV) with wall thickness >1.5 cm confined to the most distal region of the LV wall at the apex below the papillary muscles with the Apical wall to posterior wall ratio (APW/PW) of greater than 1.5 7. It is associated with a classic electrocardiographic pattern of giant negative T waves (>10 mm) in the precordial leads and a “spade” deformity of the LV at end diastole on ventriculogram1. Invasive angiographic and hemodynamic features of this condition include an hour-glass shaped left ventricular cavity, systolic mid cavity obliteration, a distinct apical chamber and a systolic pressure gradient at the mid ventricular level13.

In our patient giant negative T waves were observed. The precise distribution of hypertrophy at the LV apex may in part be responsible for the development of giant negative T waves. The elevated troponins could represent myocardial infarction of the apex which is common in these patients. Another explanation could be transient vasospasm of the coronary arteries leading to myocardial injury as is common in cocaine users. Our patient is non-complaint and has not followed up for further imaging

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