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Case Report
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Apical Hypertrophic Cardiomyopathy Presenting as NSTEMI: A Case Report

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Abstract

Apical hypertrophic cardiomyopathy also known as Yamaguchi syndrome is a rare subtype of hypertrophic cardiomyopathy defined by giant negative T waves and spade like appearance of LV cavity on imaging. We present a 63 year old male who presented with NSTEMI in the setting of atrial fibrillation with newly diagnosed Apical Hypertrophic cardiomyopathy.

Keywords: Apical hypertrophic cardiomyopathy; Yamaguchi syndrome; Myocardial infarction

Case Presentation

A 63-year-old Caucasian man with half a pack daily cigarette use presented to the ED with complaints of left sided chest discomfort beginning in the morning upon waking seven hours prior to arrival. His chest pain was located centrally and radiated towards his upper back described as moderate in intensity. There were no aggravating or relieving factors for the patient's symptoms. The patient had associated symptoms of generalized fatigue throughout the day with episodes of diaphoresis and nausea. Upon further investigation he stated that a department of transportation physical evaluation uncovered an abnormal rhythm on Electrocardiogram (ECG) and he came to the ED per recommendation by his primary care physician.

His cardiovascular risk factors include obesity with a body mass index of 31.14 kg/m^2, the patient's history includes hypertension and dyslipidemia. He reports occasional alcohol and marijuana use. His home medications include amlodipine 10 mg daily, carvedilol 25 mg twice daily, and chlorthalidone 25 mg once daily.

Physical examination showed a well nourished patient complaining of active chest discomfort.

Vital signs showed heart rate of 110 beats/min, blood pressure of 133/85 mmHg, respiratory rate of 18 breaths/min saturation 97% on room air. He was alert and oriented. Cardiac examination revealed tachycardia with an irregularly-irregular rhythm, normal S1 and S2 with no murmurs or additional heart sounds. No jugular venous distention or carotid bruits appreciated. His lungs were clear to auscultation bilaterally. Abdomen was soft and non-tender and no lower extremity edema was noted.

EKG showed a ventricular rate of 117 with atrial fibrillation, left ventricular hypertrophy with repolarization abnormality, giant T waves in precordial leads and a prolonged QT interval as seen in Figure 1. Anteroposterior chest radiograph showed no evidence of any cardiopulmonary pathology. SARS-CoV-2 (COVID-19) viral testing was negative.

Troponin-I drawn was found to be 0.150 ng/mL which trended up to 0.540 ng/mL. Given his presenting symptoms and possibility of acute coronary syndrome the patient was administered aspirin 324 mg oral, clopidogrel 300 mg oral and enoxaparin 90 mg subcutaneously as per ACS protocol.

Repeat EKG revealed atrial fibrillation with marked T wave abnormality again seen and prolonged QT interval (QT/QTc of 408/510 ms). There was moderate voltage criteria for left ventricular hypertrophy as seen in Figure 2.

Transthoracic Echocardiogram (TTE) revealed an ejection fraction of 60% to 65%. The left

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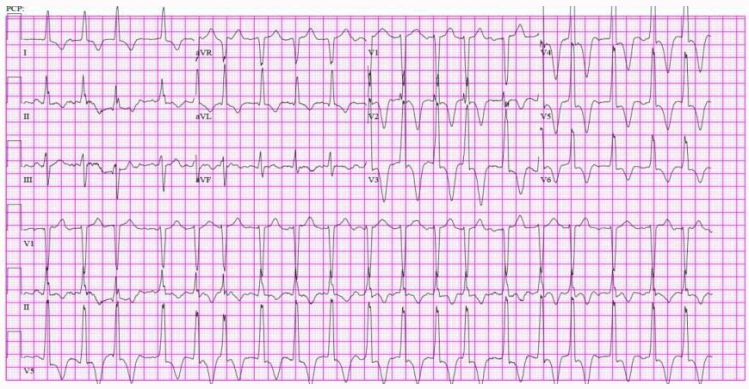

Figure 1: Revealing deep T wave inversion in anterior leads.

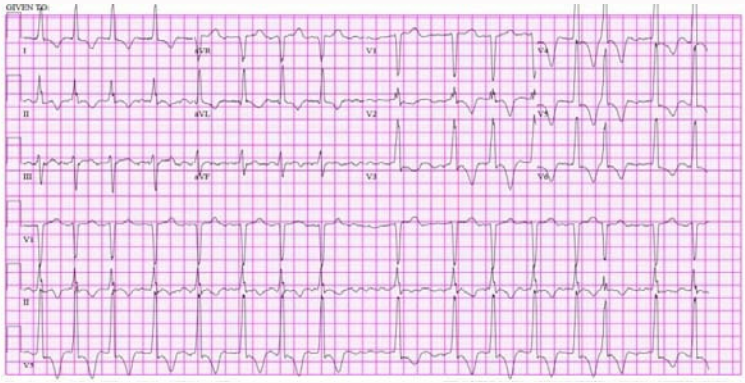

Figure 2: Showing atrial fibrillation with "giant" negative precordial T-waves & voltage criteria for LVH.

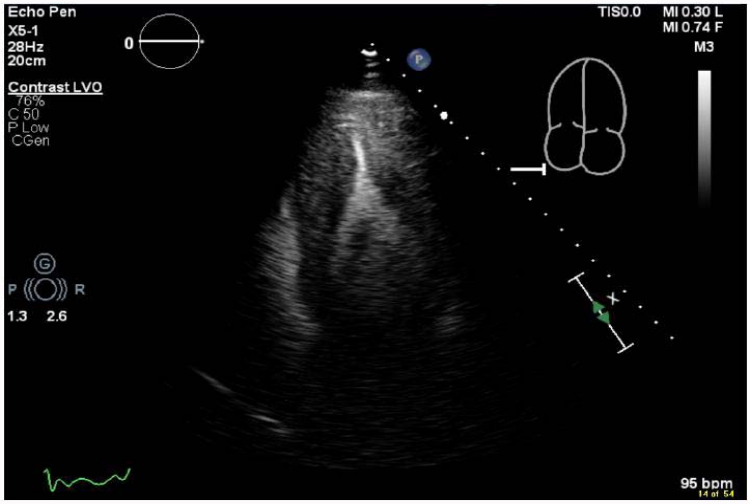

Figure 3: Showing apical four chambers with spade like appearance and severe apical hypertrophy.

ventricular cavity was small with hyperdynamic LV function. 2D echocardiogram didn't show clear apical hypertrophy therefore Definity contrast enhancement was used revealing a spade-shaped configuration with disproportionate hypertrophy of the apex, suggestive of an apical variant of hypertrophic cardiomyopathy as seen in Figure 3.

There was severe LV apical wall hypertrophy (25 mm) with remaining LV wall segments moderately thickened (15 mm) and no significant intracavitary gradient. Global Longitudinal Strain (GLS) was -2.5% and apical GLS pattern is severely depressed (0.0% to 0.8%) (Figure 4). There was mild mitral annular calcification without significant regurgitation and no evidence of mitral valve prolapse or stenosis. The right ventricle size and function was normal. The atria were grossly normal in size and there was no hemodynamically significant valvular disease appreciated.

The patient underwent left heart catheterization which showed no evidence of epicardial coronary artery disease with widely patent vessels including the right coronary, left circumflex, and the left

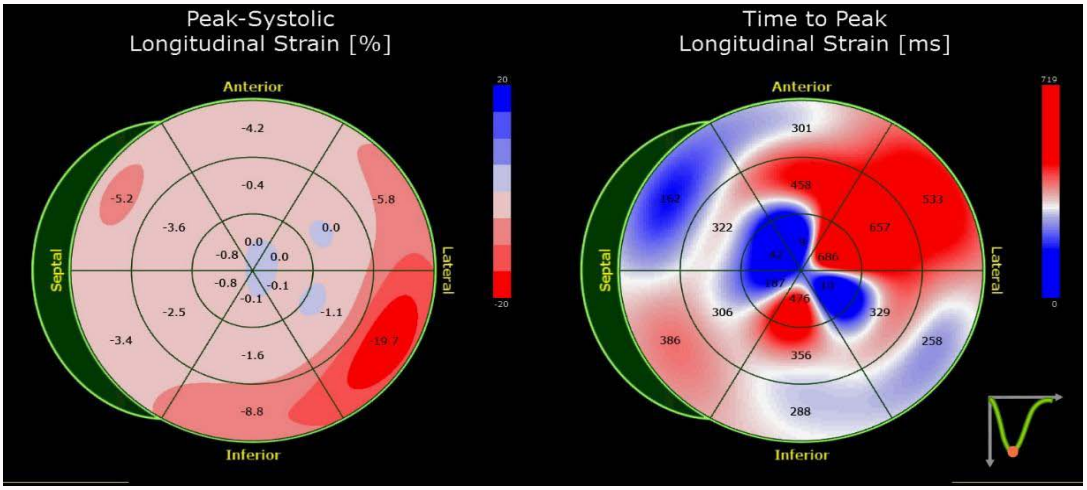

Figure 4: Strain pattern.

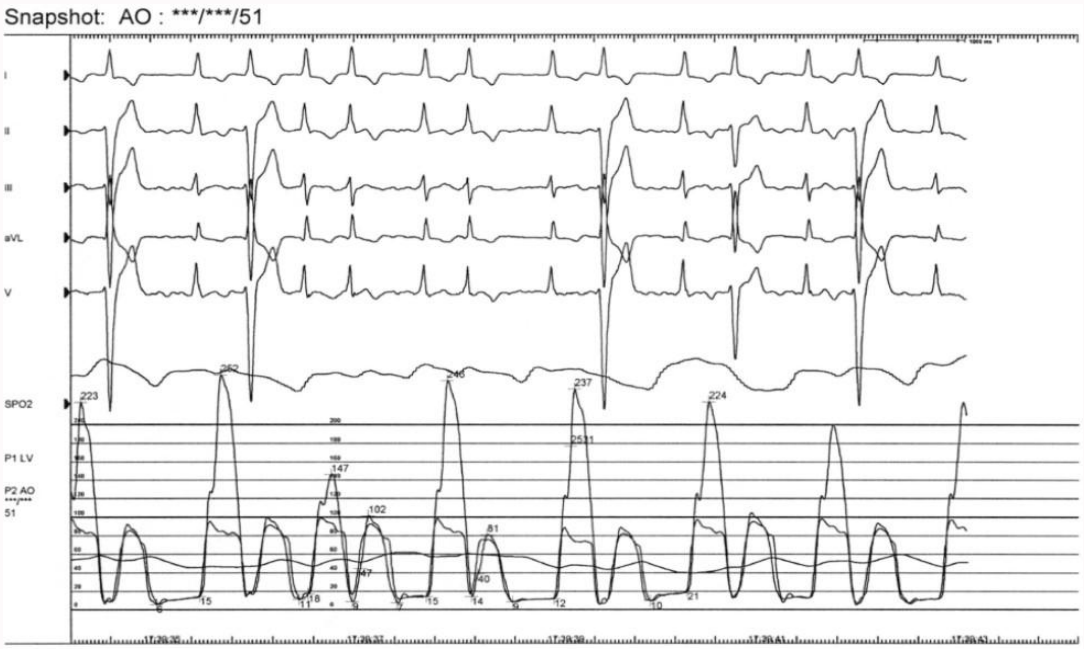

Figure 5: Showing hemodynamics during left heart catheterization.

main arteries. Hemodynamic findings include Left Ventricular End Diastolic Pressure (LVEDP) of 22 mmHg. There was a significant left ventricular apex to aortic gradient. Baseline pressure in the apical area was 24 mmHg and rising to 110 mmHg after an induced premature ventricular contraction (Figure 5). The gradient at the mid ventricle was 5 mmHg and the gradient at the base of the LV was 0 mmHg.

The patient was discharged home with diagnosis of apical hypertrophic cardiomyopathy and new onset atrial fibrillation. He was started on metoprolol succinate 100 mg oral once daily and apixaban 5 mg oral twice daily per CHADS-VASC score.

Discussion

Apical Hypertrophic Cardiomyopathy (AHCM) is a rare subtype of non-obstructive hypertrophic cardiomyopathy affecting 1 out of 500 people and 3% in the United States [1]. The most common clinical presentations are exertional dyspnea (38%), angina (8%), syncope (11%) and heart failure (6%) [2]. Sudden cardiac death, stroke or myocardial infarction are also described as first manifestations of AHCM [3,4].

Apical hypertrophic cardiomyopathy is characterized by decreased diastolic volume of the LV subsequently leading to decreased cardiac output. Left ventricular hypertrophy also results in myocardial ischemia and fibrosis. Increased filling pressures may lead to left atrial dilation, which increases the risks of atrial fibrillation as seen in our patient.

Hypertrophy of the apical myocardium can lead to mismatch in myocardial demand and coronary arterial supply resulting in myocyte injury and therefore troponin release in Apical HCM patients [5].

Typical ECG findings involve deep symmetrical negative T waves in precordial leads as well as LVH voltage criteria [4,6]. T wave inversions in precordial leads are commonly seen in most cases but

"giant" T waves are found in 47% of all patients with AHCM [7].

Doppler echocardiogram normally shows increased thickness of the left ventricular wall in the apical region (cut off value fixed at 15 mm as measured below the insertion of the papillary muscle). Apical four-chamber view resembles the image of an "ace of spades."

It is pertinent to use contrast enhancing echocardiogram to see obliteration of apex which could be lost in 2D echocardiogram.

Furthermore, longitudinal strain plots derived from 2D speckle tracking imaging can be helpful by demonstrating the absence of longitudinal deformation in apical segments. This is surrounded by the regions with normal strain values at the basal and mid segments [7].

Medical management of apical hypertrophic cardiomyopathies includes the use of beta blockers or calcium channel blockers in patients with preserved ejection fraction at maximum tolerated doses. In the case of reduced ejection fraction, standard heart failure medication can be used. Implantation of an Implantable Cardioverter Defibrillator (ICD) implantation should also be considered in patients with syncope, asymptomatic NSVT, family history of sudden cardiac death as well as left ventricular wall thickness >30 mm [8].

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