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Case Report

Hypertrophic cardiomyopathy mimicking inferior wall myocardial infarction

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ABSTRACT

Electrocardiogram is most often the first-hand diagnostic tool with a cardiologist. Promptly identifying life threatening arrhythmias and myocardial infarction with ECG saves many lives. Quite often the electrocardiogram may have dubious findings and further testing helps arriving at the right diagnosis. Herein, we present a case of hypertrophic cardiomyopathy where the ECG mimicked inferior wall myocardial infarction along with a raised high sensitive Troponin T (hsTnT). A coronary angiogram failed to reveal any acute or chronic obstructive lesion in the coronary arteries. We discussed the varied ECG patterns in hypertrophic cardiomyopathy and causes of troponin elevation apart from myocardial infarction. We also discussed other causes of 'pseudo-infarct' pattern on ECG. This provides insight into a more comprehensive approach in management of each patient.

Keywords: Hypertrophic cardiomyopathy, Myocardial infarction, Cardiac biomarker

INTRODUCTION

The electrocardiographic (ECG) changes in hypertrophic cardiomyopathy are varied. The ECG pattern is abnormal in almost 90% patients and approximately 75% of their asymptomatic first-degree relatives.[1] Although no pattern is highly specific for the disease, left ventricular hypertrophy, deep inverted T waves and early repolarisation abnormalities are most often found. Other common findings are left axis deviation, left atrial enlargement and nonspecific ST-segment abnormalities. Such a nonspecific pattern on ECG may mimic other pathologies like myocardial infarction. Cardiac biomarkers like hsTnT are sine quanone for diagnosing acute MI. But these biomarkers may also be elevated in the presence of ventricular hypertrophy, sustained tachyarrhythmias, DC cardioversion and renal dysfunction.[2] Although ventricular tachycardia may elevate troponin levels in the absence of myocardial infarction, serial elevation to 4.5 times the previous value and approximately 200 times the 99th percentile upper reference limit as in our case is unusual. To have a strong suspicion of myocardial infarction is obvious in such a case.

CASE REPORT

A 40 years old male patient presented to the hospital with syncope which was preceded by palpitations for around thirty minutes. He was pulseless on admission and an urgent electrocardiogram recorded wide complex tachycardia (Figure 1A). A diagnosis of ventricular tachycardia was entertained and external transthoracic direct current (DC) cardioversion was done in view of hemodynamic instability. After reversion to sinus rhythm, the patient regained sensorium and his vital parameters were stable. On detailed history then, he admitted of having exertional dyspnea of NYHA functional class II for last 6 months. He had sought no medical advice for that. There was no history of angina, recurrent palpitations, presyncope, stroke or aborted resuscitation attempts in the past. The patient was previously normotensive with no

relevant drug intake history. There was no family history of sudden cardiac death or any relevant cardiac illness.

After cardioversion, his blood pressure was 104/66 mmHg with a regular pulse of 78 beats per minute. On auscultation, there was a medium-pitch ejection systolic murmur at the left lower sternal border. This murmur increased on Valsalva straining and decreased on sudden standing from lying down position. Electrocardiogram on presentation was suggestive of ventricular tachycardia (rate 220 per minute and regular) with wide QRS (168 msec) and positive concordance in all precordial leads. There was atrio-ventricular dissociation and an initial R wave in aVR lead (Figure 1A). After electrical cardioversion, the electrocardiogram was puzzling. It showed normal sinus rhythm, deep inverted T waves (>4 mm) in leads V4-V5 (Figure 1B). There was right bundle branch block with ST-T changes. There were pathological Q waves (40 msec in duration, 5-7 mm deep and >25% of respective lead R wave) with convex ST segment elevation of >2 mm in leads II, III, aVF and V6 along with ST-T depression in lead aVL. The ECG was suggestive of Inferior STEMI with reciprocal depressions. However, there was no evolution of ECG changes on subsequent examinations. High sensitivity Troponin T sent at the time of admission was 0.625 ng/ml. This biomarker value increased to 2.85 ng/ml after 5 hours. The patient's complete blood count, basic metabolic panel, and liver function tests were all within the normal range. 2D echocardiogram revealed normal biventricular functions with hypertrophied interventricular septum. The maximum end diastolic thickness at basal septum was 1.8 cm. There was resting left ventricular outflow tract gradient of 38mmHg, which increased to 60 mmHg on provocation with sublingual nitrate. A diagnosis of hypertrophic obstructive cardiomyopathy (HOCM) was made. As he presented with sustained ventricular tachycardia, he was a candidate for coronary angiogram but those pathological Q waves with convex ST changes in inferolateral leads, along with significantly rising hsTnT levels prompted us for an early diagnostic angiogram. He was taken up for coronary angiogram after stabilisation which revealed normal epicardial coronaries (Figure 2). He was advised an ICD (Implantable-cardioverter-defibrillator) along with medical therapy for hypertrophic obstructive cardiomyopathy including beta blocker.

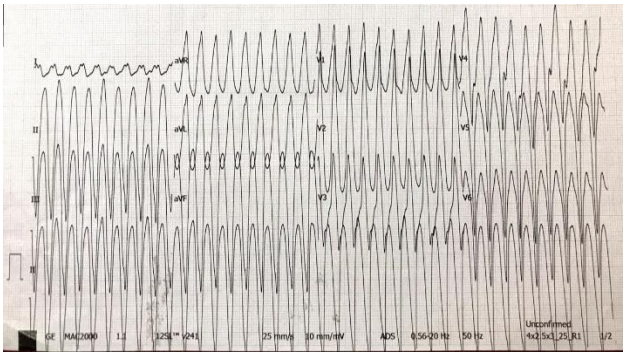

Figure 1A: Electrocardiogram: on presentation.

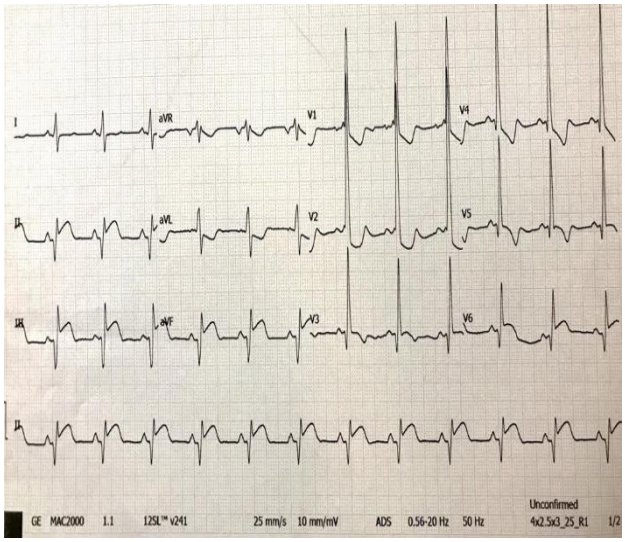

Figure 1B: Electrocardiogram: Post DC cardioversion.

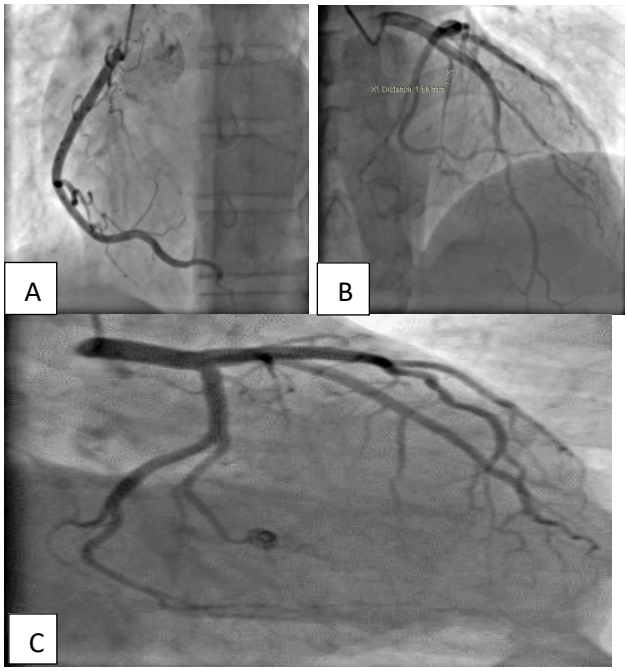

Figure 2. Coronary angiography.

DISCUSSION

Hypertrophic cardiomyopathy (HCM) is one of the most common genetic cardiac disorder with a prevalence of 1:500 in the general adult population.[3] Majority of the patients are asymptomatic and are diagnosed incidentally by an abnormal ECG or a murmur on examination.[4] Others may present with angina, dyspnoea, palpitations or syncope. Syncope is often the result of ventricular tachyarrhythmias as in our patient and may be the first presentation of the disease.[4] The resting ECG findings in patients with HCM are extremely heterogenous. Left ventricular hypertrophy, deep inverted T waves and early repolarisation abnormalities are most often found. Other common findings are left axis deviation, left atrial

enlargement and nonspecific ST-segment abnormalities.[1] Table 1 lists the various ECG changes found in HCM.[1,5] In LV apical involvement, giant T wave inversions (>10 mm) may be seen in precordial leads. Pathological Q waves may be seen in the inferolateral leads and are associated with replacement fibrosis.[5] It is difficult to differentiate from inferior wall myocardial infarction in such a case. There have been incidences where ECG appeared like anterior wall myocardial infarction in patients with hypertrophic cardiomyopathy.[6,7] Like in our case, there were pathological Q waves along with convex ST-segment elevation in leads II, III, aVF and V6. There was ST-T depression in aVL which appeared like a reciprocal depression. hsTnT was also elevated (0.625 ng/ml) and increased to 2.85 ng/ml which is almost 200 times the 99th percentile upper reference limit. This further supported our diagnosis of inferior wall myocardial infarction. With no evolution of ST-T changes in inferolateral leads, what can this high troponin be attributed to? It is known that troponin levels may rise due to ventricular tachyarrhythmias, left ventricular hypertrophy, hypertrophic cardiomyopathy and DC cardioversion. But in a patient with preserved renal functions, can such a high troponin elevation be due to all this alone was concerning. Table 2 lists the causes of elevated troponin values.[2]

Table 1: ECG changes in HCM.[1,5]

Left ventricular hypertrophy
T wave inversion in lateral precordial leads
Left axis deviation
Left atrial enlargement
Early repolarisation pattern
Pathological Q waves in inferior leads

Table 2: Various causes of elevated levels of Troponins.[2]

Myocardial Ischemia and infarction
Coronary artery dissection
Sustained tachyarrhythmia
Defibrillator shocks
Severe left ventricular hypertrophy
Heart failure
Cardiomyopathy
Coronary revascularisation procedures
Catheter ablation
Hypotension or shock
Sepsis
Severe Anemia
Cardiac contusions
Pulmonary embolism
Chronic kidney disease

Coronary angiogram was done and revealed no obstructive coronary artery lesion. This showed that the ECG changes and the elevated cardiac troponin were not due to inferior wall infarction. The diagnosis of hypertrophic cardiomyopathy is conventionally made with an echocardiogram. It is a learning point that it may have such a diverse ECG pattern and mimic acute myocardial infarction. Also, there may be numerous causes of troponin elevation and may even cause such a high level. So, very witfully one needs to rule out acute coronary syndrome in such a case. Dynamic LV outflow tract obstruction and raised LV filling pressures lead to elevated pulmonary capillary wedge pressure. Due to ventricular hypertrophy and high ventricular filling pressures, myocardial demand-supply mismatch occurs and leads to myocardial ischemia.[1] Beta blockers are indicated to reduce LV filling pressure and LV outflow tract obstruction.[8] Such negative ionotropic agents lead to symptom relief. An ICD is indicated for secondary prevention of SCD in such a case who presented with sustained VT.[8]

It is not just hypertrophic cardiomyopathy which may mimic as acute myocardial infarction. We need to know various other conditions also which may appear like acute myocardial infarction on the ECG. Such conditions are listed in Table 3.[9] Correlating with the clinical scenario and other diagnostic modalities helps rule out ACS in such situations.

Table 3. Causes of 'Pseudo-infarct' pattern on ECG.[9]

Early repolarisation pattern
Electrolyte abnormalities (e.g. hyperkalemia)
Myopericarditis
Intracranial hemorrhage
Left ventricular aneurysm
Pulmonary embolism
Hypertrophic cardiomyopathy
Non-thrombotic vasospasm
Hypothermia

CONCLUSION

While ECG and cardiac biomarkers help us rule in or rule out various possible diagnosis, they may even confuse a clinician at times. Knowing the different ECG presentations of diseases like hypertrophic cardiomyopathy, causes of false positive troponin elevations and various other diseases with 'pseudo-infarct' pattern on ECG helps us reach the right diagnosis without delay.

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