Case Report

Atypical ECG Presentation of Hypertrophic Cardiomyopathy stimulating Acute Myocardial Infarction

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ABSTRACT

Hypertrophic cardiomyopathy (HCM) is an idiopathic disease of myocardium characterized usually by marked asymmetric myocardial hypertrophy without underlying pressure or volume overload. This can cause spectrum of anatomic and hemodynamic consequences ranging from being asymptomatic to sudden cardiac death. HCM can have various ECG changes including significant ST-T changes. Rarely, the condition may sometimes present with ST elevation of acute ischemic injury type stimulating myocardial infarction. We here present a case where the patient presented with ST elevation and was initially evaluated as myocardial infarction. Further detail evaluation ruled out acute the myocardial infarction and confirmed the diagnosis of HCM. Patient subsequently improved with the treatment. Appropriate clinical evaluation along with investigation will avoid wrong management and unwanted intervention.

Keywords: Electrocardiogram, ST segment elevation, sudden cardiac death

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INTRODUCTION

Hypertrophic cardiomyopathy is an idiopathic heterogeneous disease of myocardium and recognized to be an important cause of arrhythmic sudden death, heart failure, and atrial fibrillation.[1] Electrocardiogram (ECG) may show a wide variety of abnormal patterns, some of which are distinctly altered or even bizarre, although none is typical or characteristic of the disease.[2] Sometimes, abnormal ECG can mimic the acute

myocardial infarction causing diagnostic dilemma.

We here present a rare case where the patient presented with chest discomfort and ST segment elevation in electrocardiogram. Subsequent evaluation ruled out myocardial infarction in our patient. Further evaluation confirmed the diagnosis of hypertrophic cardiomyopathy and treated accordingly.

CASE PRESENTATION

A forty-seven years female presented in our OPD with history of on and off chest pain and external shortness of breath for last 3 months. For last two days, she gave history of continuous vague chest discomfort. She did not have history of palpitation, dizziness or syncopal attack. She was post-menopausal, non-hypertensive, non-diabetic and was not on any medication.

On general exam, she was not in obvious distress. BP was 130/ 70 mmHg and pulse rate was 70 beats per minute. On cardiovascular examination, apex was at fifth intercostal space and normal character. The first and second heart sound was normal. There was systolic murmur of grade 3 at the apex. Systemic examination was not significant. ECG done as routine revaluation revealed ST elevation of 3 milivolt in V2 – V4. She was transferred to emergency and evaluated for acute myocardial infarction. She was given with antiplatelets and considered for revascularization. Meanwhile, echo screening revealed marked hypertrophy of anteroseptal wall and apex with normal left ventricular systolic function. No regional motion wall abnormality was noted.

Diagnosis of acute ST elevation MI was reconsidered. Initial CPK MB and troponin I were both negative. Both tests were repeated after six hours and after twenty-four hours. All reports were negative. Serial ECG showed persistence of same pattern of ST elevation with no dynamic changes. It was decided to keep the patient in close observation and treat medically. Coronary angiogram was performed to rule out coronary artery disease and it showed normal coronaries. Subsequently, she was treated with low dose diuretic and metoprolol 25 mg twice a day. Hospital stay for next two days was uneventful. Patient came in follow up after one week, one month and three months. Patient symptomatically improved. ST elevation in ECG persisted with no new changes.

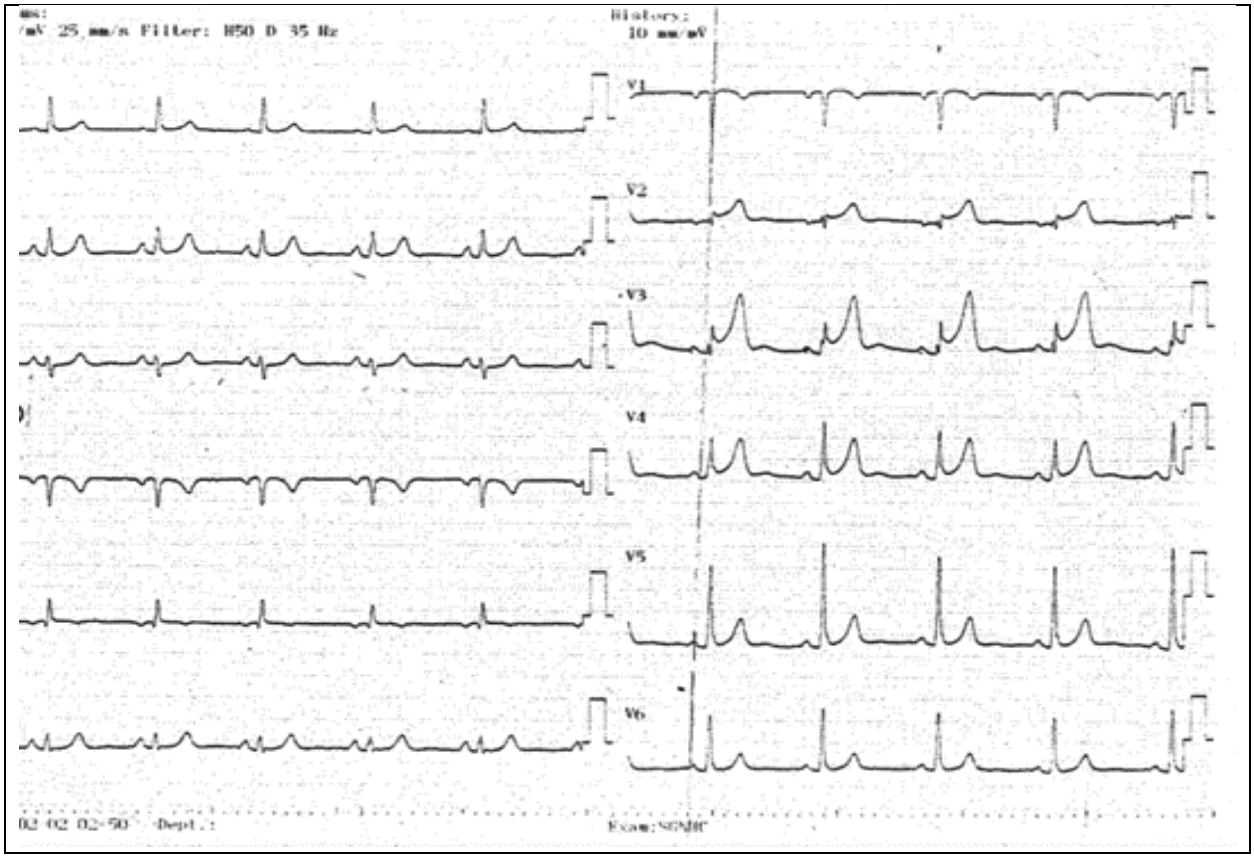

Figure1: ECG of the patient

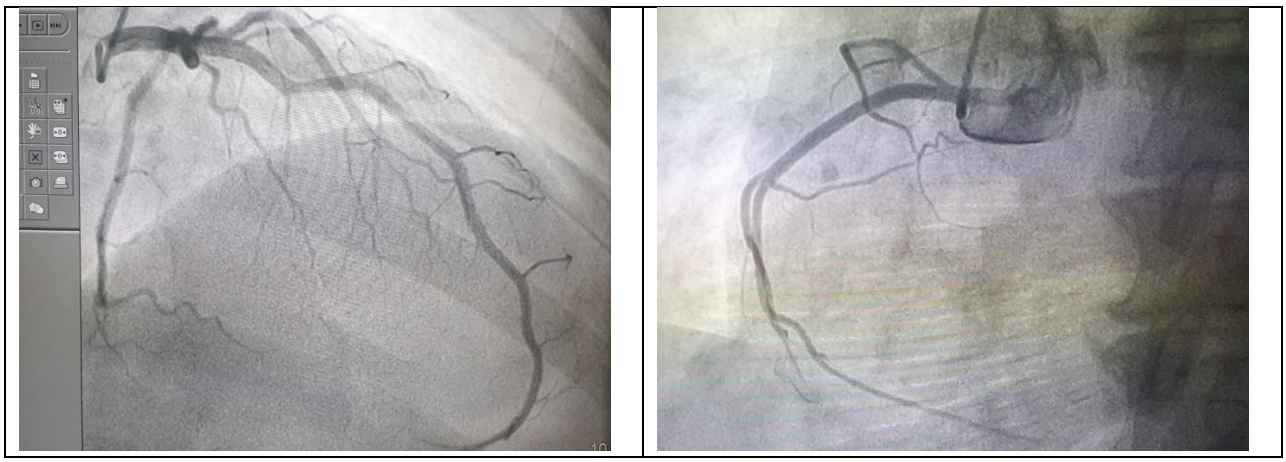

Figure 2: Coronary Angiogram of the patient

DISCUSSION

As mentioned earlier, hypertrophic cardiomyopathy is a familial disease primarily of myocardium. It is usually inherited by autosomal dominant pattern of inheritance, but up to half of cases may be sporadic.[3] The natural history and clinical course of hypertrophic cardiomyopathy is widely variable ranging from an asymptomatic course with a normal life span to sudden cardiac death.[4]

Anatomically, myocardial hypertrophy may be diffusely concentric, involving the whole of the left ventricle, or asymmetric with a maximum at a certain part of the ventricle. The interventricular septum is the most common site for hypertrophy, which may involve the entire septum or only its base, middle, or apical portion. Hypertrophy may be maximum not only at the septum, but also at the apex, free anterolateral wall, posterobasal wall and midventricular portion of the left ventricle.[5,6] Asymmetric septal hypertrophy and diffuse concentric hypertrophy are more prone to produce left ventricle outflow tract obstruction and left ventricular intracavity pressure gradient.

We can suspect hypertrophic cardiomyopathy on the basis of abnormalities in clinical examination and investigations. Classic findings in ECG include evidence of left ventricular hypertrophy on electrocardiography. ST-T changes including marked T wave inversion in the lateral precordial leads, left atrial enlargement, deep and narrow Q waves, and diminished R waves in the lateral precordial leads are also commonly found.[7] There are many conditions where ECG of the patient may have non ischemic ST elevation. Usually, they can be explained due underlying conditions like left ventricular hypertrophy, bundle branch blocks, Wolff- Parkinson-White syndromes, early repolarization syndromes, dyskinetic ventricular wall, vasospastic angina and acute pericarditis.

Rarely, a patient with HCM may have ST elevation stimulating acute injury type of myocardial infarction. Few previous reports have indicated that hypertrophic cardiomyopathy may mimic myocardial infarction. Khan IA et al[8] reported a case of sixty-two-year female with ST elevation and marked diffuse left ventricular hypertrophy. Their patient was clinically asymptomatic and had no evidence of myocardial ischemia. Sayin et al.[9] and Lin CS et al.[10] had reported similar cases. However, these reports are mainly for apical hypertrophic cardiomyopathy. The chronic increase in apical pressure and ischemia due to cavity obliteration and midventricular obstruction secondary to the apical hypertrophy may stimulate infarction in this subset of patients.

In our patient, she had sepal hypertrophy in antero-septal wall and apex with no evidence of pressure gradient. So, we believe that hypertrophic cardiomyopathy may present bizarre ECG changes that cannot always be explained with current knowledge and routine investigation. In this case, timely recognition of the condition saved unwanted result.

CONCLUSION

ST segment elevation in ECG with suspected acute coronary syndrome is considered to be medical emergency. However, frontline health care workers should be aware that not all patients with ST elevation are diagnosed with myocardial infarction. We should collaborate all our findings including investigations to make a diagnosis. HCM is myocardial disease which may have array of ECG changes including ST elevation. This may stimulate acute myocardial infarction as in the present case.

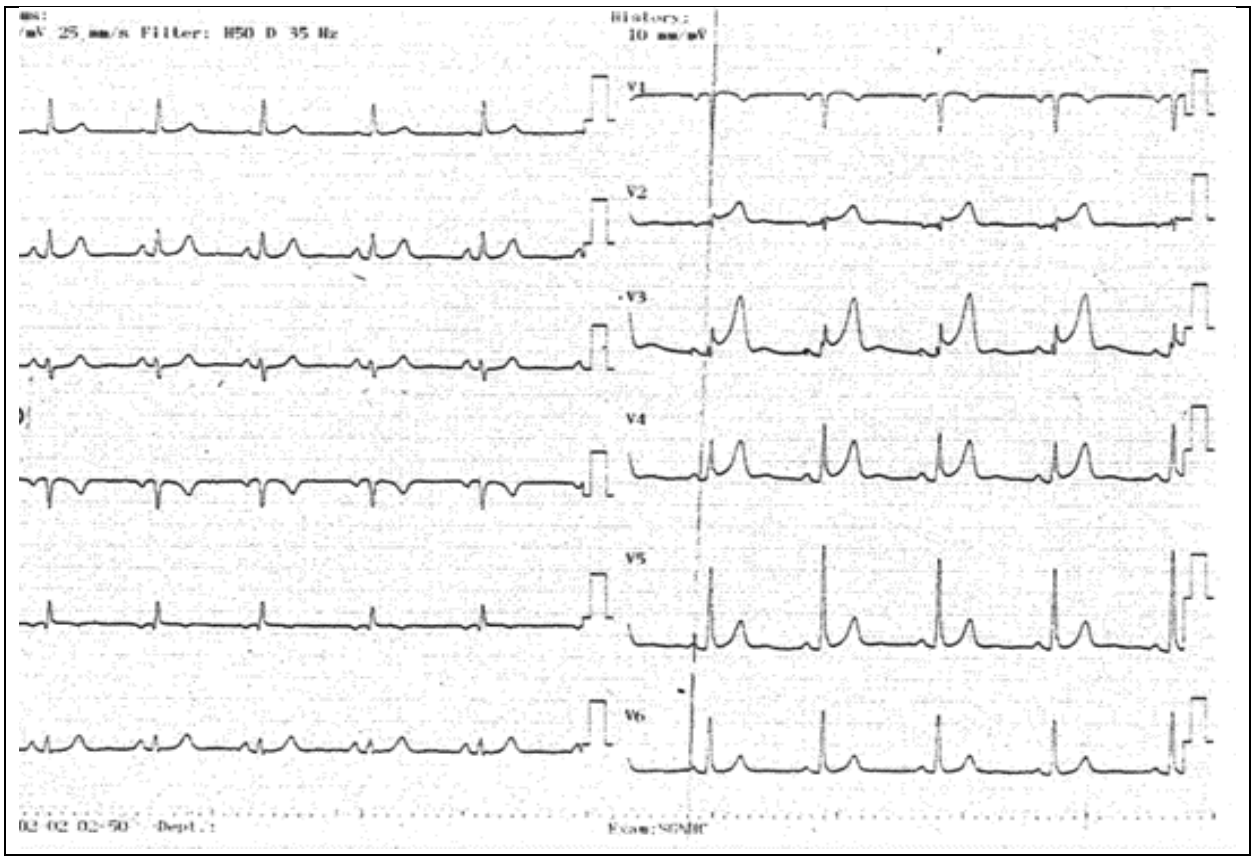

Figure1: ECG of the patient

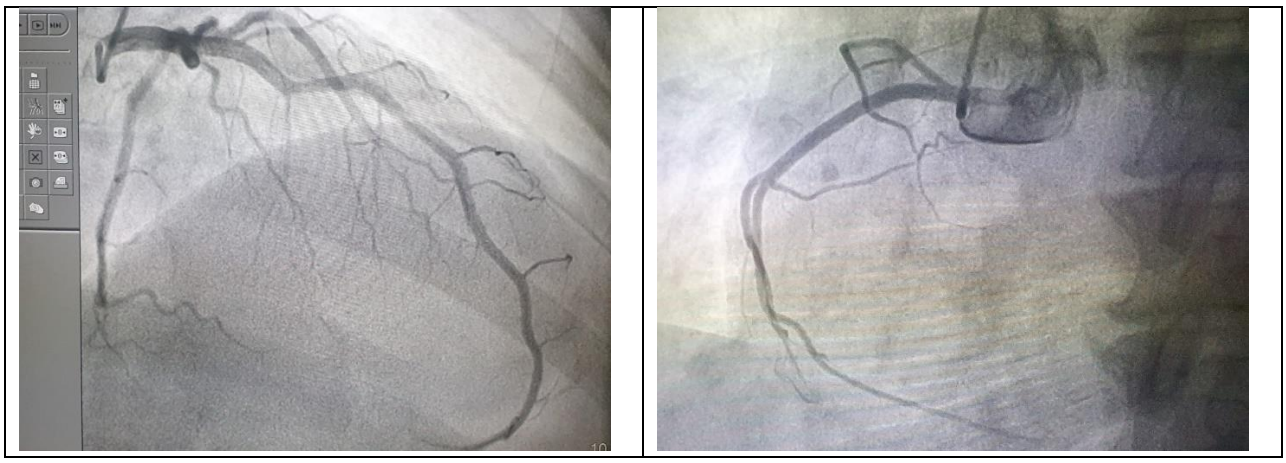

Figure 2: Coronary Angiogram of the patient

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