Myocardial infarction in a patient with hypertrophic cardiomyopathy

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Introduction

Hypertrophic cardiomyopathy (HC) is a disease characterised by marked heterogeneity in its morphology and natural history. The prevalence of significant coronary artery disease in this population has been estimated to be just over 10%.[1] On the other hand, the prevalence of transmural myocardial infarction in the absence of significant coronary atherosclerosis is about 15% in a population of patients who have died from HC.[2] Although electrocardiographic criteria for diagnosis of acute myocardial infarction (AMI) in adults are well known and accepted, no general criteria exist for diagnosis of AMI in patients with HC. Further, there are no clear-cut guidelines for the management of patients with HC who present with a suspected AMI.

Case report

A 39 year old non-smoker presented to the Accident and Emergency Department (A&E) with a history of 2½ hours of typical cardiac chest pain associated with sweating and vomiting. The pain was somewhat relieved by sublingual glyceryl trinitrate (GTN) administered by the paramedics. His cardiac risk factors included hypertension, hyperlipidaemia and a history of angina. He had been recently diagnosed elsewhere as having a cardiomyopathy, which was later confirmed to be hypertrophic non-obstructive cardiomyopathy. His medication on admission was atenolol and atorvastatin.

Physical examination revealed the pulse to be 90/minute and regular, with a blood pressure of 148/90 mmHg, normal heart sounds and a clear chest.

The initial electrocardiogram (ECG) showed a sinus tachycardia (100/minute), normal axis (30°), normal PR interval, normal QTc interval and marked T wave inversion in leads I, II, aVL, V5 and V6 with upright T waves in aVR. There was no ST elevation in any of the leads (figure 1).

Initial cardiac enzyme measurements showed a creatinine kinase (CK) of 774 IU/L (normal = 25–200) and a lactate dehydrogenase (LDH) of 1,610 IU/L (normal = 350–700).

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Figure 1. The patient's ECG on admission

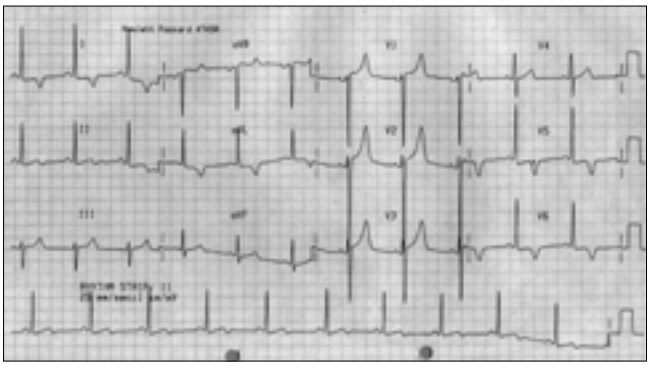
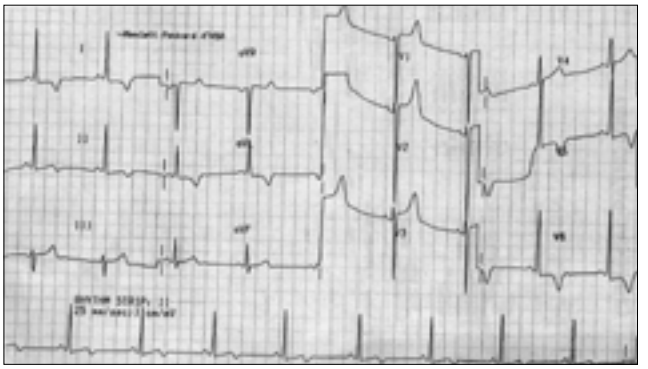


Figure 2. The patient's follow-up ECG



Although an acute myocardial infarction (AMI) was strongly suspected, since there were no ECG criteria for thrombolysis he was treated with aspirin and intravenous heparin. Nitrates were not used because of his underlying cardiomyopathy.

Serial ECGs did not change (figure 2) and no Q waves or ST elevation appeared. On review of ECGs performed a month prior to his admission, the T wave changes noted at that time were present and identical.

Serial CK levels were 1,240, 878 and 584 IU/L whereas serial LDH levels were 1,055, 1,236 and 1,120 IU/L. A CK(MB) performed on the first sample was raised at 47 IU/L (normal < 8).

He made a good recovery and was discharged with a diagnosis of AMI after four days, to be followed up in out-patients

Key messages

- Significant coronary artery disease occurs in 10% of patients with hypertrophic cardiomyopathy (HC)
- Conventional ECG criteria for diagnosis of acute myocardial infarction (AMI) can be unreliable in patients with HC
- Coronary angiography when the patient presents with suspected AMI is desirable, to determine whether angioplasty or thrombolysis might be beneficial

by the cardiologists. He underwent a transthoracic echocardiogram, which showed marked symmetrical left ventricular hypertrophy, systolic anterior motion of the mitral valve and no evidence of outflow tract obstruction. Diagnostic cardiac catheterisation revealed angiographically normal coronary arteries.

Discussion

This patient presented with a good history of cardiac pain on a background of several coronary risk factors. He was not thrombolysed because of the absence of conventional ECG criteria but subsequent cardiac enzyme measurements confirmed the diagnosis of AMI. This case illustrates the difficulty in interpreting ECGs of patients with HC in the context of AMI: this may lead them to be denied treatment aimed at revascularisation. Conversely, several case reports have described patients with HC presenting with chest pain who have ECGs with ST elevation mimicking an AMI. These patients have been subsequently demonstrated to have normal epicardial coronary arteries and have therefore been unnecessarily subjected to thrombolysis.[3,4] Thus conventional ECG criteria for thrombolysis of AMI can be misleading in patients with hypertrophic cardiomyopathy and cannot be relied upon. We present a third possible scenario where AMI occurs in a patient with apparently normal coronary arteries.

It is well recognised that up to 97% of patients with HC have abnormal ECGs,[5] typically with hypertrophic changes, deep inverted T waves, deep Q waves or slurred QRS complexes. Since typical cardiac chest pain is also a common symptom in patients with HC,[6] a strategy to resolve this diagnostic dilemma is required.

It is obviously important to offer appropriate revascularisation to HC patients who have an AMI. Patients with HC have an annual mortality of 2–6%,[7] so an AMI in this population will clearly contribute to a more rapid deterioration in their ventricular function and put them at even higher risk of sudden death from arrhythmias.

Conclusions

First, there should be greater awareness of the unreliability of using ECG abnormalities to diagnose AMI in patients with known or suspected HC, given the likelihood of pre-existing abnormalities and the disturbance in the normal progression of changes in AMI seen in other patients. As with other conditions where abnormal conduction or repolarisation is a feature, such as left bundle branch block pattern or a paced rhythm, the ECG should not be relied upon to diagnose or exclude AMI. The only exception to this may be if a previous ECG is available and there are obvious new changes suggestive of AMI.

Second, this high-risk group stands to benefit substantially from appropriate interventions towards revascularisation in the context of AMI if, as in 85% of patients, it is due to coronary thrombosis.[2] Hence coronary angiography at the time of presentation with suspected AMI is desirable in these patients to determine whether they would be candidates for primary angioplasty or thrombolysis.

If cardiac catheterisation facilities are not available then we propose that the clinical presentation alone should be used to make decisions regarding thrombolysis, given the probable high benefit: risk ratio. We know that older patients with coexistent coronary risk factors and non-obstructive forms of HC seem to be at highest risk of having significant coronary artery disease.[1] The use of troponin T estimation may also assist in the early diagnosis of AMI.[8] This latter approach, however, has not been validated in clinical trials. Guidelines on the management of a suspected AMI in this group of high-risk patients would be useful.

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