Acute Myocardial Injury: An Entity of Acute Coronary Syndromes

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Summary

A 46-year-old Indian man was admitted with unstable angina (Braunwald's class IIIB1) - the chest pain at rest was associated with inferolateral ischaemia on the ECG at admission. His serial total creatine kinase levels were elevated but CKMB was not detected. Serial serum troponin T levels were significantly raised suggesting the presence of significant acute myocardial injury.

Key Words: Chest pain, Acute myocardial injury

Introduction

Unstable angina is associated with a significant risk of subsequent myocardial infarction or death. The diagnosis is often made on clinical grounds. A subgroup of patients with unstable angina were found to have raised concentrations of troponin T. Detection of this cardiac structural protein indicated myocardial injury and this finding was associated with an excess of adverse cardiac events similar to patients with acute myocardial infarction.

Case History

Mr. P.V., a 46-year-old Indian man, presented with sudden onset of retrosternal chest pain typical of ischaemic pain which occurred at rest and lasted for three hours prior to admission. He had an alcoholic bout prior to the onset of the chest pain. There was a history of chronic alcoholism for ten years. He was a diabetic and his elder brother died of ischaemic heart disease. On admission, he appeared drunk with alcohol in his breath. He was pink and anicteric with no liver flap. There were no signs of hyperlipidaemia. He had bilateral parotid swellings with no other stigmata of chronic liver disease. His blood pressure was 110/70 mmHg and regular pulse rate of 70 beats per minute. Jugular venous pressure was not raised. Examination of the praecordium and lungs was normal. The abdomen was soft with mild epigastric tenderness. There was no pedal oedema.

His ECG on admission showed sinus tachycardia with T-wave inversion in L_{II}, L_{III}, AVF and V_4-V_6 (Fig. I). A subsequent ECG at 12 hours following admission showed ST-segment depression in the same leads (Fig. II). Based on Braunwald's classification, this patient presented with class $IIIB_1$ unstable angina in which the acute chest pain occurred at rest within 48 hours of admission and it occurred in the absence of antianginal therapy. Intravenous (IV) morphine 2.5 mg and IV nitroglycerin 5 µg/min were given which

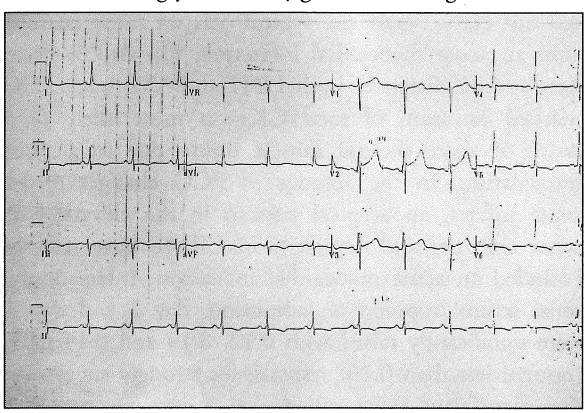

Fig. 1

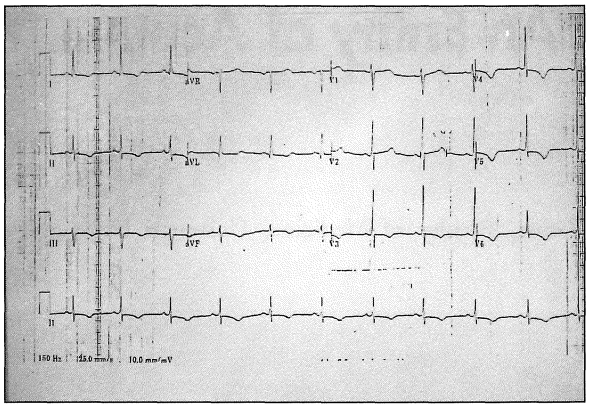

Fig. 2

reduced his chest pain. He was also started on oral aspirin 75 mg daily and metoprolol 50 mg twice daily, IV ranitidine 50 mg 8 hourly, oral thiamine 50 mg thrice daily and oral glibenclamide 5 mg twice daily.

His haemoglobin was 16.3 g/dL with macrocytosis, total white cell count 10.4 x 10^9/L and platelet 229 x 10^9/L. Random blood sugar was 7.9 mmol/L and liver function test was normal except for a mild elevation of alanine transaminase of 40 U/L (normal up to 32). On admission, his total creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate transaminase (AST) were 1715, 410 and 69 U/L respectively (normal range in male: CK 24-195, LDH 200-480, AST 0-37). On day 2 and 3, total CK levels dropped to 865 and 200 U/L respectively. However, CK-MB(using catalytic method) was not detected in any of the three blood samples. There were also concomitant decreased levels of LDH and AST which did not demonstrate the typical delayed rising pattern after an acute myocardial infarction. On day 5, there were normalization of both LDH and AST levels. The marked elevation of total CK was most likely as a result of acute skeletal muscle injury due to alcohol intoxication. In the absence of ECG changes of an acute infarct, an atypical pattern in the elevation of other enzymes and negativity of CKMB have reliably excluded an acute myocardial infarction. Interestingly, serial serum troponin on admission, day 2 and day 3 were significantly raised with 0.12, 0.10 and 0.14 µg/L (normal less than 0.10) respectively, strongly suggesting that significant acute myocardial injury has occurred in this patient. He was also found to have hypertriglyceridaemia (TG 6.4 mmol/L) with normal total cholesterol (4.4 mmol/L). His serum amylase was normal and HBs antigen was not detected.

On day 3 of admission, he was asymptomatic and his ECG showed improvement of the inferolateral ischaemia. Predischarge symptom-limited exercise tolerance test using modified Bruce protocol was done on day 8 and this showed significant ST segment depression in the inferolateral leads at low workload (3.0 METS). This normalized at 7 minutes of recovery (Fig. III). He was discharged with isosorbide dinitrate 10 mg three times daily, aspirin 75 mg daily, metoprolol 50 mg twice daily and glibenclamide 5 mg twice daily. The patient refused to give consent for coronary angiography and defaulted follow up.

Discussion

The clinico-pathological features of the two principal manifestations of ischaemic heart disease - acute myocardial infarction and chronic stable angina - have already been well described. A syndrome which is an intermediate between these two entities, unstable angina, is often difficult to define and diagnose. It is a change in the usual pattern of angina resulting in an increase in the number, severity or duration of anginal episodes; angina occurs at a decreasing level of exercise or at rest. The episodes of chest pain may also be less responsive to nitroglycerin, often with attacks recurring shortly after initial relief. It is characterized by severe transient myocardial ischaemia without significant elevation of cardiac enzymes.

With the availability of more sensitive and specific immunoassays for biochemical markers such as troponin T, it is being recognised that occasionally in cases of unstable angina, 'acute myocardial injury' may occur. Troponin T is only available in the cardiac and skeletal striated muscles in man. It is the structural protein that binds the troponin complex to tropomyosin molecules. After loss of integrity of myocardial cell membranes following a severe ischaemia, troponin T proteins are released into the circulation. Cardiac troponin T can be differentiated from its isoforms in the skeletal muscle by a one-step enzyme immunoassay, and it is not detectable in the serum of healthy people. However, there are reported

cases of raised troponin T in patients with chronic renal failure and polymyositis (Peter Stubbs, Charing Cross Hospital, London personal communication), but not in patients with chronic liver disease. It has a sensitivity of 96% and specificity of 98% in detecting ischaemic myocardial injury. Even in the presence of 30-40% severe skeletal muscle injuries, it still maintains a high specificity (94%)[1]. Serum troponin T is highly useful not only in the diagnosis of acute myocardial infarction as defined by the accepted conventional criteria, but also helpful especially in detectinig small ischaemic myocardial injuries[1].

Gerhardt *et al* proposed that acute myocardial injury occurs in patients with severe ischaemic heart disease when there is increased serum troponin T (> 0.10 μg/L) with concomitant changes of serum CKMB below the discrimination limit <10 μg/L5. It is important for one to be able to recognize this subgroup of patients as they constitute about 26-35% of cases of unstable angina. Surprisingly, their prognosis in terms of subsequent cardiac events (cardiac death and non-fatal acute myocardial infarction) within 28 months of follow up is similar to those who suffered a confirmed acute myocardial infarction[2]. Recently, Van der Warf postulated that elevated levels of cardiac troponins in patients with acute coronary syndromes may be associated with unstable coronary plaques which affect their prognosis[3].

Therefore, it awaits to be seen whether the identification of a raised troponin levels could complement a risk stratification scheme for selection of patients for coronary angiography and more aggressive revascularization procedures.

References

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