Cardiac troponins and the risk stratification of chest pain

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Abstract

We present three cases of patients who had chest pain with abnormal but non-diagnostic ECGs and negative troponin I, carried out in the appropriate time frame. All three went on to have extensive coronary artery disease demonstrated on coronary angiogram. These cases illustrate that use of troponin I alone as a marker for risk stratification of cardiac chest pain is not adequate: above all, a high index of clinical suspicion is of paramount importance.

Key words: chest pain, cardiac troponins, risk stratification.

Introduction

There are three troponins that form part of the troponin-tropomyosin complex, which regulates muscle contraction. Of these, troponin T and troponin I exist in cardiospecific isoforms, cardiac troponin T (cTnT) and cardiac troponin I (cTnI).

There has been a great surge in the use of cardiac troponins in the risk stratification and subsequent management of chest pain. These tests are the gold standard in the preliminary assessment of chest pain and acute coronary syndromes. There has also been particular interest, in light of the NICE guidelines, which recommend that patients with suspected acute coronary syndrome and unstable angina who are troponin positive, should be both regarded as high risk and considered for the use of a glycoprotein (GP) IIb/IIIa antagonist.

Case 1

A 59 year old man, who had been previously healthy, presented with a history of chest pain whilst cycling. He described a three-week history of increasingly frequent episodes of heavy left-sided chest pain. His only risk factor was a cholesterol of 6.07 mmol/L. His clinical examination was unremarkable.

On investigation, he was troponin I negative at 12 hours from admission. His electrocardiogram (ECG) can be seen in figure 1a. An exercise tolerance test was positive at three minutes, with infero-lateral ST segment depression.

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Figure 1. **a** illustrates an ECG for case 1, showing sinus rhythm with T wave inversion in leads III and AVF and ST segment high take-off in V1 and V2 **b** illustrates a coronary angiogram for case 1, showing almost complete stenosis of the proximal third of the left anterior descending artery, plus other abnormalities
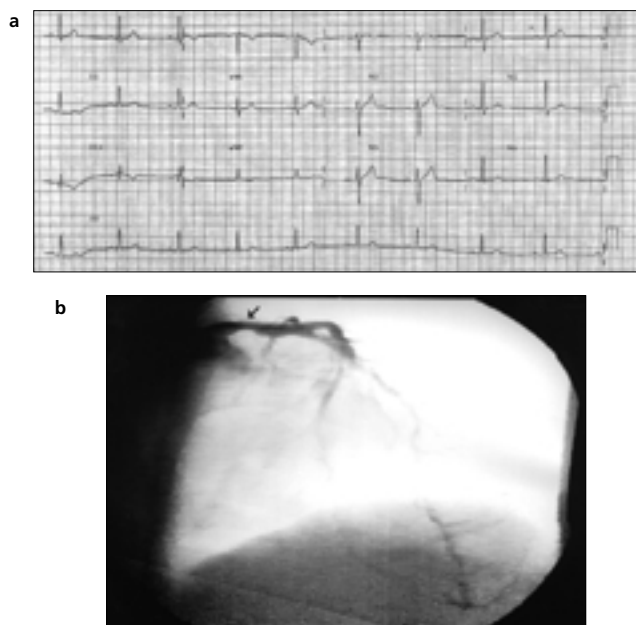
Coronary angiography showed 99% stenosis of the proximal third of the left anterior descending coronary artery, 90% stenosis of the mid-third of the circumflex artery and proximal stenosis of the right coronary artery (see figure 1b).

Case 2

A 51 year old Asian man presented with a history of chest pain at rest, which appeared to be cardiac in origin. He had strong cardiac risk factors, ie. he had type 2 diabetes mellitus, he was a smoker of 40 pack years and he had a serum cholesterol level of 6.18 mmol/L. Clinical examination was unremarkable.

On investigation, he was troponin I negative at 12 hours from admission. His ECG can be seen in figure 2a.

Coronary angiography showed: 60-80% stenosis of the left anterior descending coronary artery, 50% stenosis of the middle third of the left circumflex artery, 90% proximal stenosis of the second obtuse marginal branch of the circumflex artery, 99% stenosis of the middle third of the right coronary artery and 80%

Figure 2. **a** illustrates an ECG for case 2, showing sinus rhythm with saddle shaped 1 mm ST elevation in leads II, aVF, V5 and V6 with ST elevation of 2 mm in V2 and V3 **b** illustrates a coronary angiogram for case 2, showing almost complete stenosis of the middle third of the right coronary artery, and other abnormalities

a

b

Figure 3. **a** illustrates an ECG for case 3, showing atrial fibrillation with T wave inversion in lead III and downsloping of the ST segments in lateral leads, possibly secondary to digoxin **b** illustrates a coronary angiogram for case 3, showing almost complete stenosis of the left anterior descending and left circumflex arteries

a

b
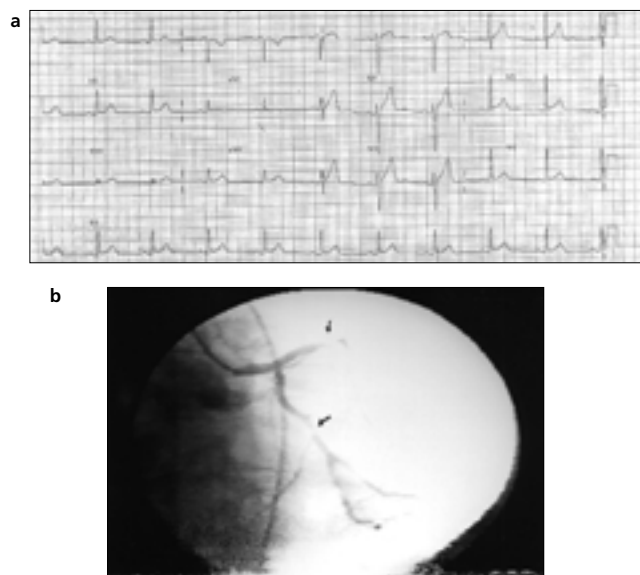
Table 1. The sensitivity and specificity of troponin I as a serum markers in chest pain

Time (hours) from onset of symptoms	Sensitivity (%)	Specificity (%)
1	13	95
4	67	95
6	90	95

Adapted from: Ebell MH *et al.*[1]

Key messages

- Troponin I alone as a marker for risk stratification of cardiac chest pain is not adequate
- Troponin levels should be evaluated in conjunction with other aspects of the case
- We do not yet have an ideal biochemical test for diagnosis and risk stratification

stenosis of the proximal third of the posterior descending artery (see figure 2b).

Case 3

A 73 year old women with known atrial fibrillation, taking digoxin, was admitted with symptoms of angina at rest. Her risk factors were hypercholesterolaemia and hypertension. Clinical examination was unremarkable.
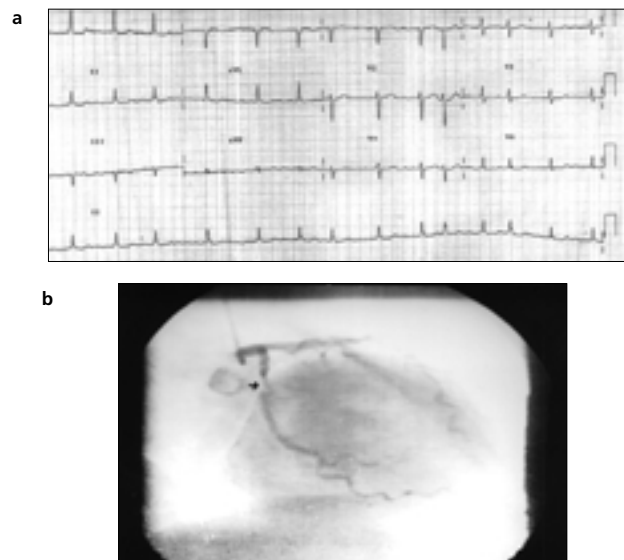
On investigation she was troponin I negative at 12 hours from admission. Her ECG can be seen in figure 3a.

Coronary angiography showed: 99% stenosis of the middle third of the left anterior descending artery, 99% stenosis of the middle third of the left circumflex artery and 50% stenosis of the proximal third of the first diagonal artery (see figure 3b).

Discussion

There is no one biochemical test that is an ideal marker for the

diagnosis and risk stratification of chest pain. Troponins are cardiospecific but their use is time dependent (see table 1).[1]

Troponin levels should be evaluated in conjunction with the presentation, the patient's risk factors and the clinical impression of the case.

Conclusion

All three patients were referred on to a tertiary centre for coronary artery bypass grafting and are subsequently well.

Editors' note

An accompanying editorial can be found on pages 195–7 of this issue.

References

1. Ebell MH, Flewelling D, Flynn CA. Systematic review of troponin T and I for the diagnosis of acute myocardial infarction. *J Fam Pract* 2000;**49**:550-6.