CASE REPORT

Chest pain with raised troponin, ECG changes but normal coronary arteries

Ayesha Amjad,¹ Amjad Ali,² Ahmed Bashir,³ Muhammed Ali,³ Muhammad Najeeb Azam⁴

¹Department of Cardiology, Princess Royal Hospital Telford, Telford, Shropshire, UK

²Department of General Surgery, Shrewsbury and Telford NHS Trust, Princess Royal Hospital, Telford, UK

³Department of Cardiology, Princess Royal Hospital Telford, Birmingham, West Midlands, UK

⁴Department of Cardiology/Medicine, Princess Royal Hospital, Telford, UK

Correspondence to
Dr Ayesha Amjad,
ayesha.amjad@nhs.net

Accepted 24 February 2014

SUMMARY

A 65-year-old woman presented to A&E department, with acute onset central chest pain and dyspnoea. ECG showed dynamic T wave changes while 12 h troponin was elevated. A diagnosis of acute coronary syndrome was made and she underwent an inpatient coronary angiogram. Although her coronary arteries were normal, symptoms persisted and D-dimers were found to be elevated. This led to a CT pulmonary angiogram, which ruled out pulmonary embolism, but uncovered a large ascending aortic aneurysm with a contained leak. She was immediately transferred to regional cardiothoracic unit for urgent surgical intervention. This case report illustrates the importance of a good clinical history, physical examination and timely investigations. It also emphasises that not all chest pain events with elevated troponin level are due to acute coronary syndrome and that alternative diagnoses should still be considered.

BACKGROUND

Aortic dissection is relatively uncommon, but it is a potentially lethal condition which typically presents with an acute sharp chest pain which radiates to the back. However, it can also manifest in a variety of different ways including congestive heart failure, inferior myocardial infarction, stroke, abdominal pain or acute renal failure. Early and accurate diagnosis is of critical importance as mortality rate surges briskly with time. For example, when left untreated, around 33% of patients die within the first 24 h, and 50% die within 48 h. Many patients with an eventual diagnosis of aortic dissection are often suspected and treated initially for other conditions such as acute coronary syndrome, pericarditis, pulmonary embolism or even cholecystitis.¹ In this article, we report a case of a middle-aged woman who presented with cardiac sounding chest pain and acute rise in cardiac enzymes suggestive of acute coronary syndrome but was found to have acute aortic dissection. This particular case illustrates that raised troponin is not always a consequence of myocardial infarction even in patients with cardiac sounding chest pain. Elevated troponin should not deter one from considering alternative causes of chest pain other than acute coronary syndrome.

CASE PRESENTATION

A 65-year-old Caucasian woman presented with a 2 h history of sudden onset sharp central chest pain. The pain was severe, episodic and non-radiating, accompanied by shortness of breath. She

is an ex-smoker, hypertensive and has a family history of ischaemic heart disease. All observations were stable with no evidence of acute haemodynamic compromise. (Clinical examination was otherwise unremarkable.) Her pain settled after administration of sublingual nitrates and intravenous morphine.

INVESTIGATIONS

Her ECG demonstrated dynamic T wave changes in V4–V6 (figure 1). Cardiac troponin I showed an acute rise (baseline troponin I, ie, 3 h post chest pain was <40 ng/L; 12 h troponin I was 301 ng/L), while her renal functions were normal (estimated-glomerular filtration rate >90 mL/min).

Initial diagnosis of acute coronary syndrome was made and standard treatment followed the diagnosis. Her chest radiograph was considered to be normal by the admitting team. An inpatient diagnostic coronary angiography was carried out on day 3 via radial approach. This revealed normal coronary arteries.

In view of raised D-dimers (>1000 µg/L) and elevated troponin I with normal coronary arteries, CT pulmonary angiogram (CTPA) was organised to rule out pulmonary embolism. CTPA revealed extensive thoracic aortic aneurysm (figure 2), extending from aortic root (5.4 cm) to diaphragmatic hiatus (4.5 cm) with a contained leak and a pericardial effusion (probable haemopericardium). There was no evidence of pulmonary embolism.

DIFFERENTIAL DIAGNOSIS

- ▶ Acute coronary syndrome
- ▶ Pulmonary embolism
- ▶ Thoracic aortic aneurysm

TREATMENT

The patient was immediately transferred to the regional cardiothoracic unit where she underwent hemiarch and ascending aorta replacement with reimplantation of innominate artery.

OUTCOME AND FOLLOW-UP

The patient was discharged on eighth postoperative day and made an uneventful recovery. She is currently followed up in clinic with no significant complications as yet.

DISCUSSION

The European Society of Cardiology defines acute myocardial infarction as ‘detection of rise and/or fall of cardiac biomarkers values (preferably

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To cite: Amjad A, Ali A, Bashir A, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-201975

Figure 1 ECGs showing dynamic changes, that is, on presentation showing T wave inversion in lateral leads (A) which normalised as chest pain settled (B).

troponin) with at least one value above the 99th percentile of the upper reference limit with at least one of the following;

Symptoms of ischemia, new or presumably new significant ST-T changes or new LBBB, development of pathological Q waves, imaging evidence of new loss of viable myocardium or new regional wall motion abnormality and identification of an intracoronary thrombus by angiography or autopsy.² In our case, the provisional diagnosis of acute coronary syndrome was supported by the clinical presentation, ECG changes and the serial rise in the cardiac troponin. Although studies have shown that raised troponin is not always a consequence of myocardial infarction, even in cases associated with cardiac chest pain. Aortic dissection is among one of the other non-ACS-related causes of elevated troponin such as pulmonary embolism, end-stage renal disease, pericarditis/myocarditis, heart failure and sepsis.³ About 20% of patients with type A dissection have ECG evidence of acute ischaemia or acute myocardial infarction.⁴

ESC recommends imaging, especially routine use of echocardiography, to confirm the diagnosis of acute myocardial infarction. The role of imaging is vital as it helps in excluding or confirming other alternative diagnoses, for example, aortic dissection, aortic aneurysm and pulmonary embolism, etc.⁵ Triple-rule-out coronary CT angiography, which looks for abnormalities of aorta, coronary arteries and evidence of pulmonary embolism, has been found useful in diagnosing non-cardiac conditions causing chest

pain.⁶ The role of imaging is of immense importance in cases where chest pain is secondary to aortic dissection as thrombolysis in aortic dissection can prove to be fatal.^{7 8} Elevated D-dimer level is also suggested to have a role in diagnosis and prognosis of aortic aneurysm.⁹ Yuan *et al*¹⁰ found that the levels of D-dimer and high sensitivity-C reactive protein were highly elevated in cases of aortic aneurysms and dissections when compared with coronary artery diseases.

In our case, retrospectively, careful comparison of current chest radiograph to the older one (figure 3) showed the inner movement of the calcification in the aortic arch—a known sign of dissection. An early echocardiogram may have reduced the time to the diagnosis, and possibly the chances of survival.

There are different systems of aortic dissection classification. According to more commonly used Stanford system, aortic dissection is of two types: type A involves ascending aorta (De-Bakey types I and II) and type B does not involve the ascending aorta (DeBakey type III).

CTPA is the first-line diagnostic investigation. Transoesophageal echocardiography follows CT aortogram; MR angiography is preferred for surveillance.

Medical treatment is reserved for uncomplicated aortic dissection (especially type B) or those awaiting surgical intervention. Various drugs are used to maintain the systolic blood pressure to 100–120 mm Hg and pulse <60 bpm. It includes intravenous

Figure 2 A CT scan showing thoracic aneurysm with a contained leak; arrows pointing towards the penetrating atheromatous ulcer which has ruptured (A, transverse section; B, coronal section; and C, sagittal section) and (D) A CT scan showing mild pericardial effusion (narrow arrow) and pleural effusion (bold arrow).

β -blockers (initial management), nitroprusside and calcium channel blockers. Proximal type A are managed surgically wherever possible. Endovascular stenting does have a role in

management of both types of dissection, particularly type B.¹¹ Patients need lifelong antihypertensive treatment and surveillance imaging.¹²

Figure 3 Chest X-ray showing aortic arch calcifications (A) when compared with previous chest X-ray performed few years ago (B).

Learning points

- ▶ The threshold of suspicion for alternative diagnoses in case of chest pain with elevated troponin should be low, especially with refractory symptoms, not responding to treatment to consider alternative diagnoses.
- ▶ Results of investigations, including imaging, should be carefully reviewed not to miss any clues. Relevant well-timed imaging helps in identifying the non-cardiac causes of chest pain.
- ▶ Appropriate early diagnosis is crucial as mortality rates are very high with every hour delay in aortic dissection. Thrombolysis in aortic dissection can be catastrophic.

Contributors AyA was involved in conception and analysis of the data, drafting the article, revision and approved the final version. AmA helped in conception and design and conducted literature research, composing and writing of the case report, revision and approved the final version. AB provided guidance during conception and design, helped to retrieve relevant images and approved the final version. MA was involved in acquisition of the data, drafting the article, revision and approved the final version. MNA provided support during conception and revision of the case report and approved the final version.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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