Is this an acute coronary syndrome?

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A 46-year-old man whom you have never met before is waiting at your surgery at 8.00 a.m. on a Monday morning. He tells you he has had 'chest pains' overnight and that he had a big heart attack four years ago, after which he had a coronary stent inserted. He didn't go to hospital this time because he doesn't think it is his heart causing these symptoms, but he wants your reassurance.

You take a brief history. He is quite stable clinically and the examination is normal. He looks relaxed and the symptoms on questioning do not sound at all cardiac-related. His 'pain' is fluttering and uncomfortable in nature and does not change significantly with position; it is not a pressure or true pain but more an irritation that lasts minutes to hours at a time. He says he has had these symptoms intermittently for 24 hours and has had them several times before but did not tell his usual doctor or his cardiologist. The cardiologist and usual GP are both uncontactable.

You do an ECG and the patient says the 'pain' was minimal or absent at the time the trace is done.

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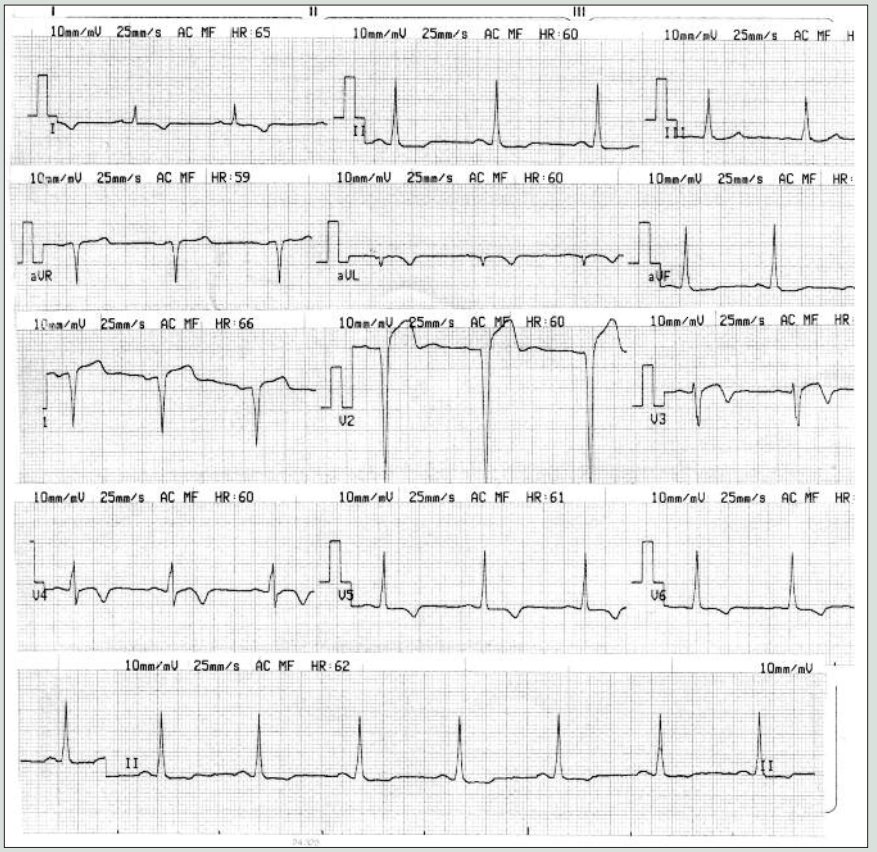

Q1. What abnormalities are shown on this ECG?
This ECG shows deep pathological Q-waves in leads V1, V2 and deep T-wave inversion in leads I, aVL, V3, V4, V5 and V6. There is ST elevation in leads V1 and V2 (over Q-waves) and in V3.

Q2. Given the information from the clinical picture and the ECG, what can be concluded about whether this patient's chest pain could be cardiac?
The history does not support cardiac pain but the patient is at high risk of recurrent myocardial ischaemia. The ECG is limited in the information it provides without comparison to previous ECGs and serum troponin results. It shows either a recent or an old anteroseptal myocardial infarction. The ST elevation could be old or new (further infarction in that region). If it is old, it suggests the patient has a ventricular aneurysm.

Q3. What clinical signs might suggest a patient is having an acute coronary syndrome?
A patient having an acute coronary syndrome often looks very unwell (pale or grey, sweaty, short of breath and anxious). They might be hypertensive (or hypotensive if they have poor cardiac output) and tachycardic (if no bradycardias or conduction defects are present). Auscultation often reveals a third heart sound and a new murmur may be present (from increased blood flow, heart failure or valvular ischaemia with resulting incompetence). If heart failure is developing, there may be crepitations at the lung bases, the apex will be displaced to the left and the jugulovenous pressure may be raised. Peripheral perfusion may be reduced

and the patient may be cyanosed. However, none of these signs are very sensitive for an acute coronary syndrome.

Q4. How can the ECG signs indicate the type of myocardial infarction?

Acute coronary syndromes are now categorised as either ST-elevation myocardial infarction (STEMI) or non-ST elevation acute coronary syndromes (NSTEACS) on the basis of ECG findings. NSTEACS will subdivide into non-STEMI (NSTEMI) or unstable angina according to cardiac enzyme test results.

Complete coronary artery occlusion results in more generalised myocardial damage and the development of ST elevation and then evolving Q-waves. Incomplete coronary artery occlusion or the presence of collateral flow from surrounding territories results more often in unstable angina or non-Q-wave myocardial infarction. NSTEMIs are more common than STEMIs. The greater the number of leads of the ECG that have acute ischaemic changes (especially Q-waves and ST elevation), the more the likelihood of a larger infarction and a correspondingly worse prognosis.

The area of the heart affected is reflected in the ischaemic changes seen on the ECG. Inferior changes involve the right coronary artery perfusion and this is reflected in leads II, III and aVF; the Q-waves are usually largest in lead III and smallest in lead II. Anterior and anteroseptal changes involve the left coronary artery and ECG changes are seen in leads V1 to V3 (V4 if septal). Anterolateral ischaemia is reflected on the ECG by the involvement of leads V4 to V6. Critical stenosis of the left main coronary artery is suggested by ST elevation in leads aVR and V1, with ST depression in multiple other leads. Note, however, that the ECG is relatively insensitive to inferolateral ischaemia, which is often associated with left circumflex artery stenosis/occlusion.

Q5. How might a series of ECGs change over time when a patient has an acute myocardial infarction?

Assuming the ECG is initially normal, the first changes seen in a typical transmural Q-wave infarction are hyperacute T-wave changes (increased amplitude and widening of the T-wave) followed by ST elevation, which is often planar or domed ('tombstone') in shape.

When there is necrosis of myocardial tissue, this is indicated by terminal T-wave inversion and the development of pathological Q-waves (these by definition have a duration longer than 0.04 s or more than 25% of the R-wave amplitude). ST elevation then decreases. Finally, and often weeks later, T-waves become upright again. Pathological Q-waves generally do not resolve with time or healing of the infarct.

In patients who have non-Q-wave infarctions, the ST-T-wave changes are more helpful to the diagnosis, especially when combined with serial serum troponin levels. Isolated symmetrical T-wave inversion is common. Any of these changes may coexist.

Q6. Which conditions can mimic acute coronary syndromes on ECG?

Conditions that can show ECG changes that may mimic acute coronary syndrome include those listed below.

- Wolff-Parkinson-White syndrome and other pre-excitation syndromes have delta waves that can mimic Q-waves, and may have ST-segment abnormalities.
- Left bundle branch blocks (LBBB) may have QS-waves and poor R-wave progression in the right precordial leads (it is difficult to be sure of an acute coronary syndrome by the ECG alone in patients with pre-existing LBBB as the typical changes are obscured by the conduction defect). If it is new, however, LBBB itself is a sign of infarction in patients with ischaemic-type chest pain.
- Acute pericarditis often develops raised, typically concave-up 'scooped out' ST segments in most leads, but ST depression in lead aVR.
- Severe chronic obstructive airways disease, cor pulmonale or pneumothorax may all be associated with inferior Q-waves, right axis deviation and loss of right precordial R-waves.
- Left ventricular hypertrophy can cause ST-segment depression or elevation in the absence of ischaemia.
- Hypertrophic cardiomyopathy with septal

Key points

- **Cardiac symptoms may be atypical so it is wise to always remember this.**
- **A normal or unchanged ECG does not exclude an acute cardiac syndrome.**
- **An acute coronary syndrome requires a serum troponin test for diagnosis if the patient has a left bundle branch block.**
- **If you think it is possible that the patient could have an acute coronary syndrome, do not waste time doing an urgent serum troponin test. The patient needs referral to a hospital centre that can do angiography.**
- **It follows that performing a serum troponin test in general practice is useful only to reassure the doctor and patient that the presentation was definitely not an acute coronary syndrome.**
- **If you request an urgent serum troponin test, ensure you are contactable for notification of the result and to take appropriate action.**

hypertrophy may have Q-waves that mimic pathological Q-waves from ischaemia.

Outcome

The cardiologist returns your call and agrees that this patient is, from the history and examination, unlikely to be an acute coronary presentation. He suggests seeing the patient early that afternoon in his rooms.

You discuss with the patient the options of his going by ambulance for hospital review now or seeing the cardiologist in his rooms in three hours' time. You tell the patient he must not drive and that you would prefer he stay in the general practice for observation until it is time to see the cardiologist. You explain that his symptoms are probably not a heart problem but this should be confirmed. The patient is surprised that you suggest hospital review because, he says, this is nothing like his previous heart attack. He wants to have his wife drive him home and she will stay with him and take him to the cardiologist that afternoon. You agree, and give him appropriate instructions should he feel worse during that time.

After review, the cardiologist thinks these symptoms are not cardiac. The serum troponin results come back negative. The echocardiogram suggests no change compared with the ultrasound performed two years ago. The sestamibi stress test performed the following week suggests no change in exercise capacity and no ischaemia compared with the test of two years ago.

The patient is educated about his condition and how to best manage it with lifestyle and medication. He is instructed to ring triple zero in future within 20 minutes of chest discomfort even if he is unsure whether the symptoms could be cardiac, as early diagnosis and treatment can improve the outcome if it were to be the beginning of a heart attack. **CT**