letters to the editor

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Severe hypercalcaemia mimicking acute myocardial infarction

Editor – We read with interest the lesson of the month by Wesson and colleagues (*Clin Med* April 2009 pp 186–7) which describes a patient admitted with vague chest pain. The patient was found to have severe hypercalcaemia (5.09 mmol/l) and an abnormal electrocardiogram (ECG). A past medical history of ischaemic heart disease (IHD), coronary angioplasty, hypertension and left ventricular failure was documented. The authors explain that the ECG in Fig 1 showed ST segment elevation in leads V1 to V5 with Q waves and poor R wave progression in the inferior leads. They conclude that these changes were due to hypercalcaemia, noting that this has only been described in two previous cases.

Having studied the ECG shown in Fig 1, we find that it also shows voltage criteria for left ventricular hypertrophy (LVH) and poor R wave progression from leads V1 to V4. We do not agree that there was poor R wave progression in the inferior leads. We would therefore suggest there are other more common diagnoses that might explain the abnormalities shown on this ECG.

Firstly, it is possible that the patient had previously developed a left ventricular aneurysm secondary to IHD, resulting in persistent anterior ST elevation. The poor R wave progression seen in leads V1 to V4 would suggest that there has been significant infarction in the anterior territory that would potentially predispose to this diagnosis. Another explanation is that some patients with voltage criteria for LVH on their ECG develop repolarisation changes that can include anterior ST segment elevation.[1] Cardiomyopathies, such as hypertrophic cardiomyopathy (which can present in older patients), can also cause voltage criteria for LVH and ST elevation.[2] A last possibility which would tie in with the chest pain, would be a diagnosis of pericarditis. The ST elevation in V5 is saddle shaped which would be in keeping with this, although the other elevated ST segments do not have this morphology. There is also no PR depression seen.

The authors do not comment on previous ECGs or any current or previous echocardiographic findings. Without considering these, and the more common causes of ST elevation, we do not feel that the authors are justified in making this very rare association.

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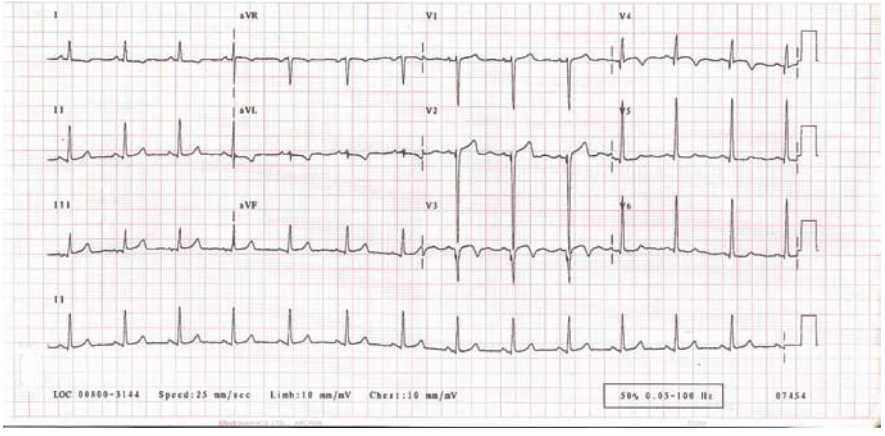

Fig 1. The most recent ECG before published event (three months prior).

References

1 Mirvis D, Goldberger A. Electrocardiography. In: Libby P, Bonow R, Mann D *et al. Braunwald's heart disease. A textbook of cardiovascular medicine*, 8th edn. Philadelphia: Saunders Elsevier, 2008.
2 Di Bella G, Bramanti O, Russo M *et al.* Hypertrophic cardiomyopathy mimicking acute myocardial infarction: diagnostic role of cardiac magnetic resonance. *Int J Cardiol* 2008;125:e34–6.

Severe hypercalcaemia mimicking acute myocardial infarction

Editor – The case report by Wesson and colleagues (*Clin Med* April 2009 pp 186–7) purports to show an electrocardiographic pattern of a myocardial infarct produced by hypercalcaemia. The authors base this supposition on the absence of elevation in cardiac enzymes and the lack of ST elevation over the 36 hours before death. However they presented no previous electrocardiogram (ECG) for comparison nor an echocardiogram at the time of the abnormality, and they noted a history of coronary artery disease and left-sided heart failure.

I would suggest that, rather than postulate a pseudo-infarct due to elevated serum calcium, the correct diagnosis is unrelated to hypercalcaemia and simply represents the presence of a left ventricular aneurysm, with anterior wall Q waves and persistent ST elevation. This would fit