letters to the editor

Please submit letters for the Editor's consideration within three weeks of receipt of the Journal. Letters should ideally be limited to 350 words, and sent by email to: Clinicalmedicine@rcplondon.ac.uk

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.

DANIEL M SADO
Specialist Registrar in Cardiology
KIM GREAVES
Consultant in Cardiology (Poole Hospital) and Honorary Senior Lecturer (Southampton General Hospital)
Department of Cardiology, Poole Hospital NHS Foundation Trust

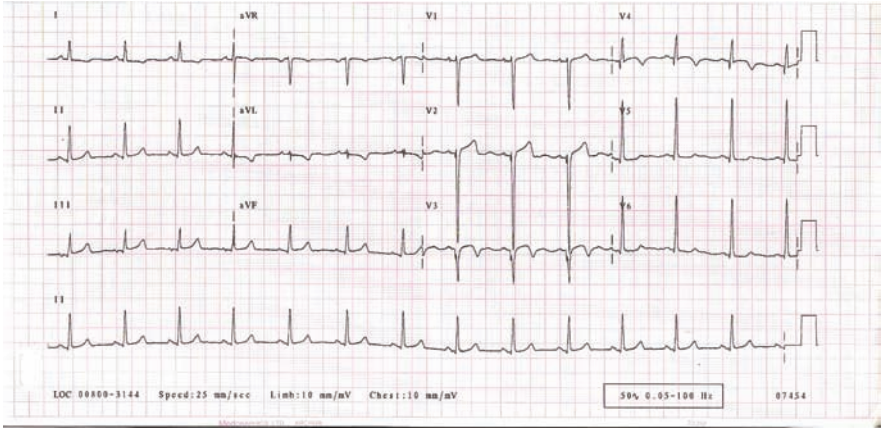

Fig 1. The most recent ECG before published event (three months prior).

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

with the history of left ventricular (LV) failure and angioplasty and would explain the negative enzymes. Although hypercalcaemia has, on rare occasions, been described as mimicking ST elevation due to its effect on the ST segment, there is no reason that it would cause the pathologic Q waves seen in leads V1 to V5. In contrast, pathologic Q waves with persistent ST elevation are typical of LV aneurysm.

The case was described as a 'lesson of the month'. I believe that the lesson here is not the one that was presented but just the opposite. From the presented data one can conclude that common things occur most commonly, incomplete data (lack of prior ECG and no echocardiogram) may lead to incorrect diagnosis, and that even reputable journals allow information to be published that is inaccurate and misleading.

RODNEY H FALK
Associate Clinical Professor of Medicine, Harvard Medical School and Adjunct Professor of Medicine, Boston University School of Medicine

Response to both letters

Sado and Greaves have raised the possibility of left ventricular (LV) aneurysm, hypertrophic cardiomyopathy (HOCM) and pericarditis as a differential diagnosis. Falk suggests that LV aneurysm was the most likely diagnosis. We agree that the points raised are valid. The patient certainly had a significant history of ischaemic heart disease. Her past cardiac history included non-ST elevation myocardial infarctions (NSTEMIs) with left ventricular failure (LVF). Seven months before the published event, she was admitted with NSTEMI and LVF. The angiogram showed normal left main stem, significant stenosis of left anterior descending (LAD) artery, moderate stenosis of distal right coronary artery, and obtuse marginal. She subsequently had elective percutaneous coronary intervention (PCI) to her LAD, with three bare metal stents, as there would have been difficulties to insert drug-eluting stents due to calcification and tortuosity. She had a further admission with NSTEMI and LVF three months before the published event, due to instent restenosis, which was treated with balloon dilatation only.

On both occasions the LV systolic function was well preserved with mild hypokinesia of the anteroapical and inferoapical walls; no evidence of LV aneurysm on either occasion. An echocardiogram (ECHO) at the time of the first PCI did not show any evidence of LVH or HOCM. Therefore we felt LV aneurysm or cardiomyopathy were unlikely based on previous investigations. Moreover the patient did not have any significant cardiac event between the last PCI and the published event. We agree that a repeat ECHO during the published admission (unfortunately not undertaken) or post-mortem (declined by family) would have clinched or refuted the differential diagnosis of LV aneurysm.

Supporting our theory, the most recent ECG before this published admission showed Q waves in V3 and V4 with ST coving and T wave inversion. Therefore we felt that the profound ST segment elevation during published admission was secondary to severe hypercalcaemia.

The chest pain history was not suggestive of pericarditis. As saddle-shaped ST elevation is only seen in V5, the ST segment elevation in other leads does not suggest pericarditis. Moreover the PQ/PR segment appears normal. Therefore we felt pericarditis to be an unlikely diagnosis. To conclude we feel that the ECG changes are secondary to profound hypercalcaemia rather than LV aneurysm or hypertrophic cardiomyopathy.

LAURA WESSON
Eldercare Specialist Registrar, Royal Cornwall Hospital
VENK SURESH
Locum Cardiology Consultant, Derriford Hospital, Plymouth

The story of Axel Munthe

Robert Allan comments that 'the true sequence of [Axel Munthe's] life is unravell[ing] and it becomes evident that many events are either imagined or at least embroidered', but surely this was already evident to the averagely intelligent reader from Munthe's own text even before the research by Alex Paton and Bengt Jangfeldt (*Clin Med* June 2009 pp 204–5). Munthe even asks the reader's forbearance for having perhaps described the man he would have liked to have been, rather than the man he actually was. The following examples drawn from the 1932 impression of *The story of San Michele* confirm that Munthe never intended us to take him too seriously[1]:

> *I am aware that some of the scenes in this book are laid on the dangerous borderland between the real and the unreal, the dangerous No Man's Land between fact and fancy…I do not ask for better than not to be believed.*
>
> Pre-preface, 'To those who have read this book', p xiii

> *But I could see quite distinctly a little man as big as the palm of my hand sitting cross-legged on the table carefully pulling at my watch chain and bending his grey old beard on one side to listen to the ticking of my repeater.*
>
> Chapter VII, 'The tallow candle and the goblin', p 139

> *'[Axel Munthe] was an unbeliever', St Ignatius went on. 'A blasphemous scoffer, a liar, an impostor, an enchanter full of black magic, a fornicator…'. 'He was fond of children', said St John. 'He was fond of their mothers too', growled a Patriarch in his beard.*
>
> Chapter XXXII(b), 'In the old tower', p 515

Unless they believed that Munthe really did spend an evening in the company of a goblin, it is hard to understand the sense of betrayal felt by some readers on subsequently discovering that Villa San Michele was designed by an architect or that the Sphinx was bought in an antique shop, rather than dug up from Nero's villa under supernatural guidance. Weaving fact and fiction beautifully together, Munthe told a story that still remains particularly inspirational to those of us in the medical profession.

However, his greatest legacy may be in the area of wildlife conservation, eg Chapter VII 'The bird sanctuary. The wings of the angels', p 448:

> *The mountain of Barbarossa is now a bird sanctuary. Thousands of tired birds of passage are resting on its slopes every spring and autumns, safe from man and beast.*

Several years ago, I chanced upon a copy of an Italian weekly magazine from around