Letters to the editor

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Non-uraemic calciphylaxis – an unexpected differential diagnosis for a necrotic ulcer

Editor - Calciphylaxis, first described by Selye in 1962, is also referred to as calcific arteriolopathy or calcification-cutaneous necrosis syndrome. 1,2 However, the latter term is not accurate as systemic calcification may affect internal organs such as the lungs, stomach, kidneys and adrenals.3 Though the cause of calciphylaxis is multifactorial, sustained, relative or absolute, hypercalcaemia is thought to be the initiating facor.³ In patients with calciphylaxis a Technetium (99mTc-MDP) radioisotope scan may show extensive increased activity throughout both lungs and lower extremities suggestive of hypercalcaemia or metastatic calcification. In the case described by Bataillard et al (Clin Med 2015;15:594-6), the corrected serum calcium is within normal limits but it may be artificially low because of the very low serum albumin (20 g/L). The patient was bed bound. I believe that the patient may have developed hypercalcaemia due to her prolonged immobilisation triggering the calciphylaxis. Immobilisation hypercalcaemia and hypercalciuria occur due to bone resorption and may respond to intravenous bisphosphonates. 4 However, more than 60% of cases with calciphylaxis have a fatal outcome.3

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- 3 Essary LR, Wick MR. Cutaneous calciphylaxis: an underrecognized clinicopathologic entity. *Am J Clin Pathol* 2000;113:280–7.
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Response

We thank the correspondents for their interest in our case and comments. Our patient had been transferred to our care following a period in a rehabilitation ward which she had been admitted to following an unrelated inpatient hospital stay. It was during this time, when she was mobile (albeit not fully), that the ulcer originally developed. During this period she persistently had normal adjusted calcium and albumin levels (adjusted Ca²⁺ range, 2.14–2.40 mmol/L; albumin 36–39 g/L). When the ulcer failed to heal, progressed and became increasingly painful despite prolonged antibiotic courses, she then became immobile and was referred back to the acute hospital for further assessment. Importantly there was no prior history of primary hyperparathyroidism and at the time of admission, parathyroid hormone concentration was normal.

Serum ionized (free) calcium is maintained within a narrow range through parathyroid hormone secretion, which in turn is regulated by serum ionized calcium acting via calcium-sensing receptors on the surface of the parathyroid cells. Therefore normal parathyroid hormone levels would indicate that our patient was normocalcaemic even in her hypoalbuminaemic state. Although both immobility and hypoalbuminaemia can lead to an increase in ionized calcium, this would consequently lead to a reduction in parathyroid hormone secretion which was not the case. Thus, although her immobility may potentially have contributed to her overall condition, we did not feel that it was the trigger to the calciphylaxis.

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Sticks and stones: investigating rude, dismissive and aggressive communication between doctors

Editor – Bradley and colleagues make an important contribution to understanding the cultural issues that underpin patient safety in the NHS by describing the problem of rude, dismissive and aggressive (RDA) communication between doctors. I would like to emphasise that this problem is not just confined to communications within hospitals but also across the primary–secondary care interface. As a general practitioner (GP) I have encountered RDA communication when trying to discuss patients with on-call hospital doctors. Often the tone of the conversation is patronising and not focused on solutions