Rare ECG finding in a patient with severe hypercalcaemia

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DESCRIPTION
A man in his late 60s with a significant history of decompensated alcohol-related liver disease was admitted to hospital with a 2-week history of increasing jaundice and pruritus as well as confusion, drowsiness and a decreased exercise tolerance. Routine bloods revealed an exceptionally elevated corrected calcium level which peaked at 5.17 mmol/L. On admission, Alpha-fetoprotein (AFP) was also greatly increased (126 278 U/L). These two derangements were later discovered to be due to an underlying diagnosis of hepatocellular carcinoma on CT. Due to his severe hypercalcaemia, the patient was admitted to the coronary care unit for cardiac monitoring and treatment. This patient was treated with immediate fluid resuscitation, intravenous pamidronate and calcitonin in order to reduce his calcium levels. A 12-lead ECG was performed (figure 1) which showed Osborn waves (figure 2). This resolved after therapy (figure 3).

The Osborn (‘J’) wave is a deflection occurring at the J point (junctional point between the R wave and ST segment) on the ECG. The phenomenon was described first in the early 20th century by Kraus et al in patients with hypercalcaemia1 but was named decades later after Osborn, who detailed the characteristic deflection induced by experimental hypothermia in animals.2 Although most commonly associated with hypothermia, rare causes of the presence of Osborn waves on an ECG include hypercalcaemia, as in this case, and subarachnoid haemorrhage.3 This characteristic deflection is posited to be mediated by the increased prominence of the cardiac transient outward potassium current for cardiac monitoring and treatment. This patient was treated with immediate fluid resuscitation, intravenous pamidronate and calcitonin in order to reduce his calcium levels. A 12-lead ECG was performed (figure 1) which showed Osborn waves (figure 2). This resolved after therapy (figure 3).

Learning points
► Osborn waves are an important ECG feature to observe for in patients presenting with hallmark signs of hypercalcaemia; for example, abdominal or bone pain, lethargy, weakness or new confusion (remembered as ‘stones, bones, abdominal moans and psychic groans’).
► Hypercalcaemia is important to diagnose and treat in the acute setting due to the risk of cognitive impairment and life-threatening arrhythmias (for which patients should be on continuous cardiac monitoring).
► Hypercalcaemia is most commonly caused by primary hyperparathyroidism (for which a Parathyroid Hormone (PTH) level is required) or malignancy, but can also be seen in a variety of other conditions including Addison’s disease, drug toxicity (eg, with thiazide diuretics or lithium), thyrotoxicosis and multiple endocrine neoplasia.
► Acute treatment of hypercalcaemia consists first of immediate and extensive fluid therapy, followed by bisphosphonates (eg, pamidronate) or calcitonin (third-line agent) if calcium levels are unresponsive (alongside seeking specialist endocrinology advice).

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(L) and subsequently increased transmural gradient occurring in the epicardium, although direct evidence is lacking.4

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