

Presenting with Hypercalcaemia: 'Chicken' or 'Egg' effect?

G Keogh¹, GA Ribaroff¹, A Kawsar¹, T Vakilgilani¹, N Vanderpant¹, V Bravis^{1,2}

¹Department of Diabetes and Endocrinology, St Marys Hospital, Imperial College Healthcare NHS Trust, London

²Division of Diabetes, Endocrinology and Metabolism, Imperial College London

Email: vassiliki.bravis@nhs.net

Introduction

Hypercalcaemia is an electrolyte disturbance seen in emergency departments¹ and patients admitted to hospital². It is caused by primary hyperparathyroidism or malignancy in 90% of patients.³ We present four cases, that demonstrate how some of the causes and sequelae of hypercalcaemia can often overlap.

Case 1

- A 36-year old man presented with severe epigastric pain after completing a marathon. He was diagnosed with acute pancreatitis and admitted under the care of the surgical team.
- Blood tests were performed to assess the severity, revealing: Adjusted calcium (cCa) 3.16mmol/L (NR 2.2 – 2.7 mmol/L), phosphate 0.50mmol/L (NR 0.8-1.5 mmol/L). PTH was elevated at 24.2pmol/L (NR 1.1-6.8 pmol/L).
- Hypercalcaemia due to primary hyperparathyroidism is a rare cause of acute pancreatitis. It can be unmasked, in the context of significant dehydration, as you might expect during a marathon.

Case 3

- A 66-year old woman presented having been found by her family collapsed on the floor. She had previously had a total thyroidectomy, that resulted in permanent iatrogenic hypoparathyroidism, for which she was taking 1-alpha-calcidol and sandocal supplements.
- Over the preceding 6 days she had become increasingly confused and weak.
- Bloods revealed cCa 3.37mmol/L, PTH<0.3pmol/L.
- The hypercalcaemia was presumed to be caused by over-supplementation. The supplements were stopped and IV rehydration commenced.
- After further discussion, it became clear that the patient had become increasingly disorientated over a period of many months, with a normal calcium level at the time. This triggered further investigation into her confusion and brain imaging revealed brain lymphoma (Figure 2). **She had overdosed on her calcium medications secondary to the confusion caused by her CNS malignancy.**
- Steroid therapy led to resolution of the lesions.

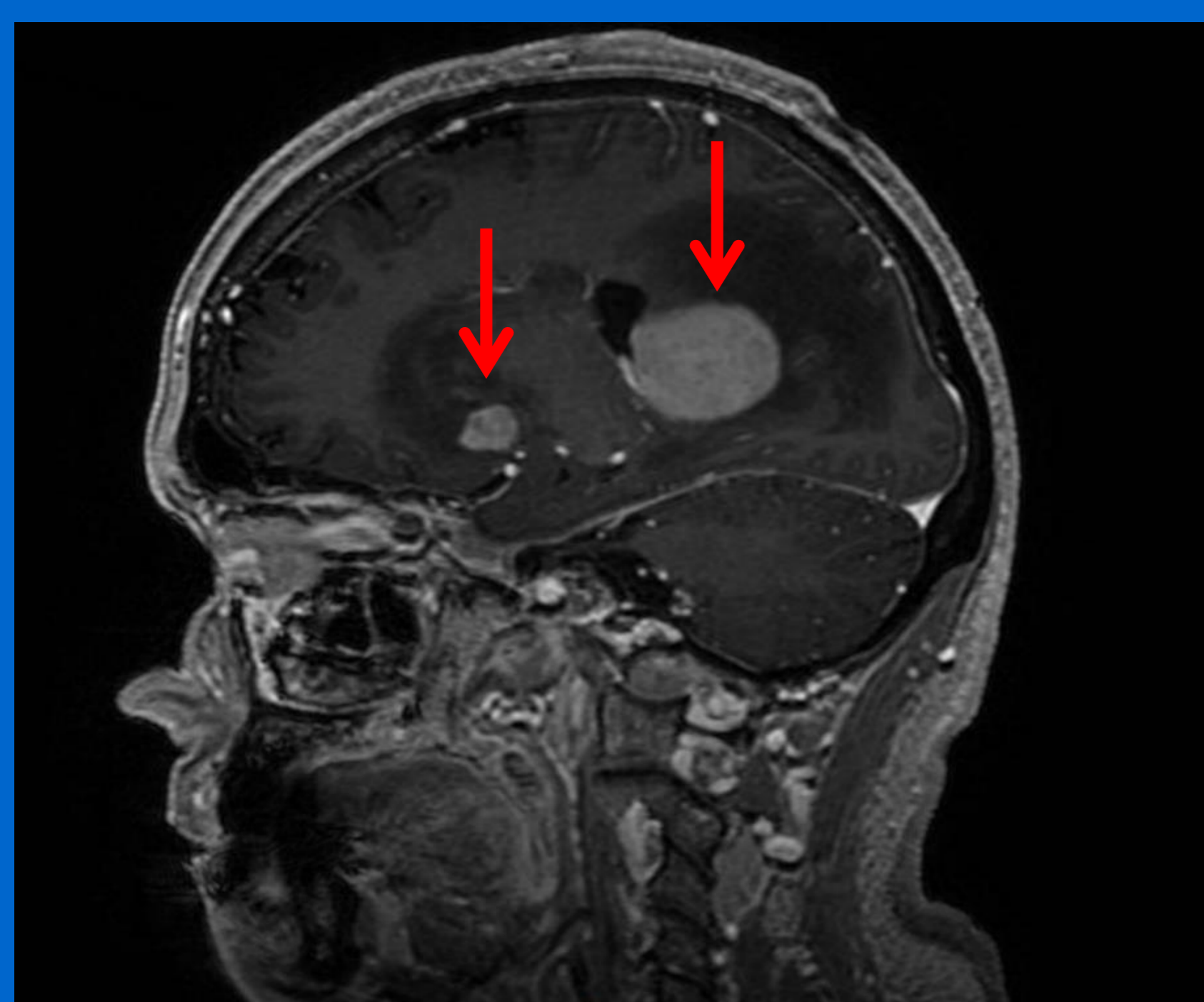


Figure 2. MRI head demonstrating marked thickening of the corpus callosum posteriorly, area of abnormality on the right caudate head, and smaller lesion in the left cerebellar hemisphere; all consistent with CNS Lymphoma seen in Case 3.

Case 2

- An 81-year old woman presented with confusion. Admission bloods showed cCa 3.3mmol/L, PTH 0.5pmol/L.
- She had been receiving annual Zoledronate infusions for osteoporosis and cCa had fluctuated between 2.6 and 2.8mmol/L. She had missed her most recent infusion.
- Her diagnosis was PTHrP-driven hypercalcaemia secondary to a pancreatic neuroendocrine tumour (Figure 1).
- **Missing her bisphosphonate allowed for osteoclastic activity and unmasked hypercalcaemia.**

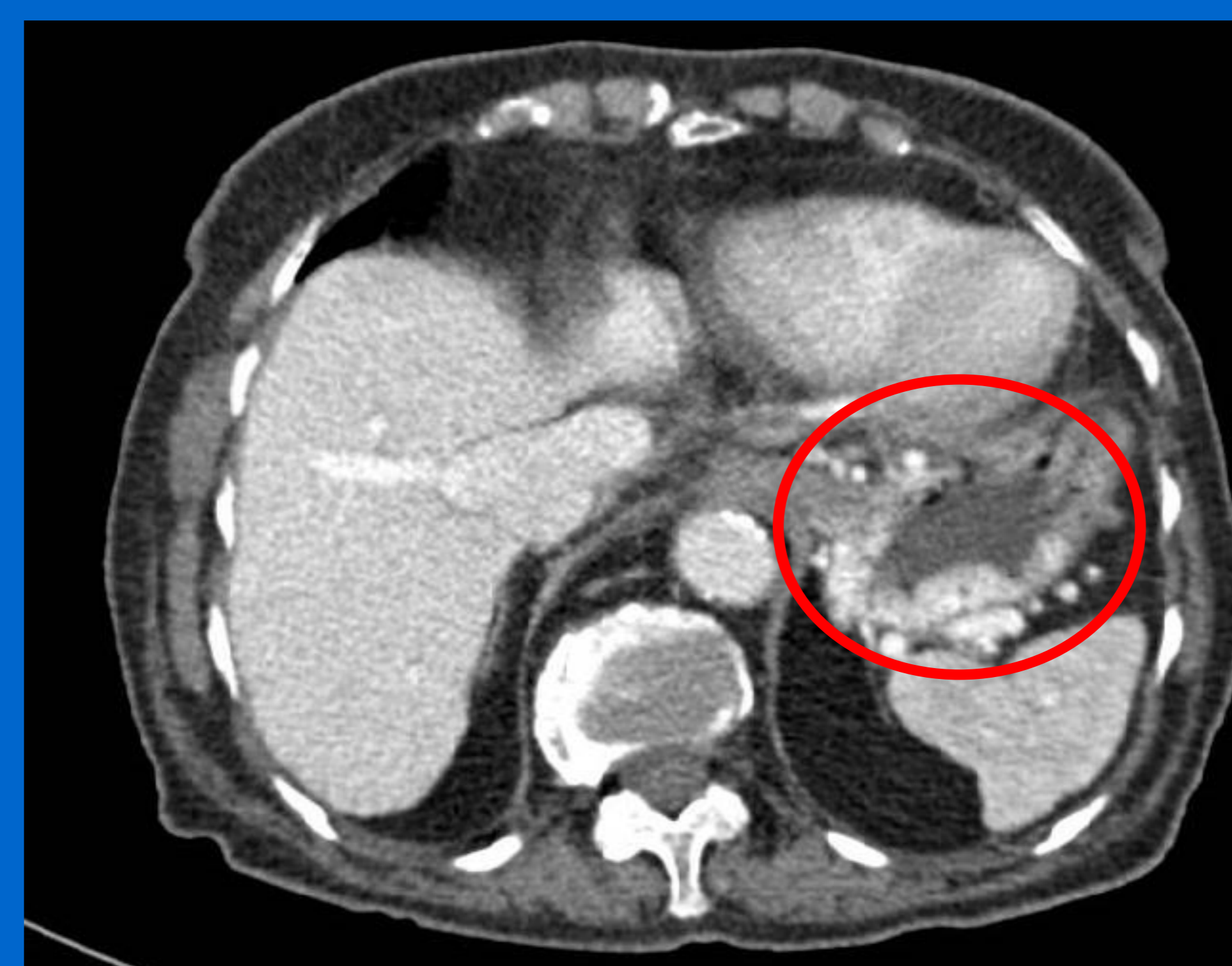


Figure 1. CT Scan demonstrating a 6.4cm*5.4cm*6.2 cm neuroendocrine tumour at the tail of the pancreas, with central necrosis, which led to PTHrP-driven hypercalcaemia in Case 2.

Case 4

- A 50-year old woman presented with a 3 week history of severe constipation, abdominal pain and nausea. She had previously been an active and healthy physiotherapist. More recently she had developed depression and had very poor oral intake.
- Bloods revealed cCa 2.83mmol/L, PTH 8.3pmol/L.
- **Dehydration unmasked primary hyperparathyroidism**, in an otherwise well individual, who had previously managed such adequate water intake that hypercalcaemia had not occurred.
- Her depression was treated with sertraline and her oral intake improved. Her calcium normalised and repeat blood tests demonstrated normocalcaemic hyperparathyroidism.
- Thereafter, she was managed conservatively, with maintenance of hydration. Surgery will be considered if clinically indicated, depending on her progress with mood management and end organ sequelae.

Conclusion

- During acute presentations, hypercalcaemia is a common finding and bone profile should always be part of acute presentations with delirium, change in behaviour or in the context of sepsis and dehydration.
- Hypercalcaemia can be the cause of the presentation or an effect of underlying pathology and needs to be approached in a thoughtful manner.
- Differentiating between cause and effect leads to appropriate investigation and management of patients.

References

1. Lindner G, Felber R, Schwarz C, et al. Hypercalcaemia in the ED: prevalence, etiology, and outcome. Am J Emerg Med 2013;31:657-60
2. National Cancer Institute. PDQ® Hypercalcaemia. 2013. www.meb.uni-bonn.de/Cancernet/ CDR0000062737.html
3. Potts JT Jr, Jüppner H. Disorders of the parathyroid gland and calcium homeostasis. In: Longo DL, Fauci AS, Kasper DL, Hauser S, eds. Harrison's principles of internal medicine. 18th ed. McGraw-Hill, 2012:3096-120.