A curious case of hypercalcaemia

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Introduction

- Hypercalcaemia is an endocrine emergency requiring prompt investigation and careful management.
- Determining the precise cause of hypercalcemia is essential to commence the right treatment as well as plan further management.
- This case will alert clinicians managing acutely ill patients to consider this in the differential diagnosis while managing patients with hypercalcemia.

Case Presentation

Case history

- Routine investigation of an 88-year-old Caucasian man admitted with acute on chronic kidney disease with normal calcium levels on admission showed incidental severe hypercalcaemia which was confirmed on repeat testing

Past Medical History

- He was previously known to have hypertension, single functioning kidney, Vitamin D insufficiency and had undergone transurethral resection of prostate for benign prostatic hypertrophy.

Examination

- On examination, patient was no more confused than his usual self, euvolemic with no significant clinical signs or symptoms

Investigations

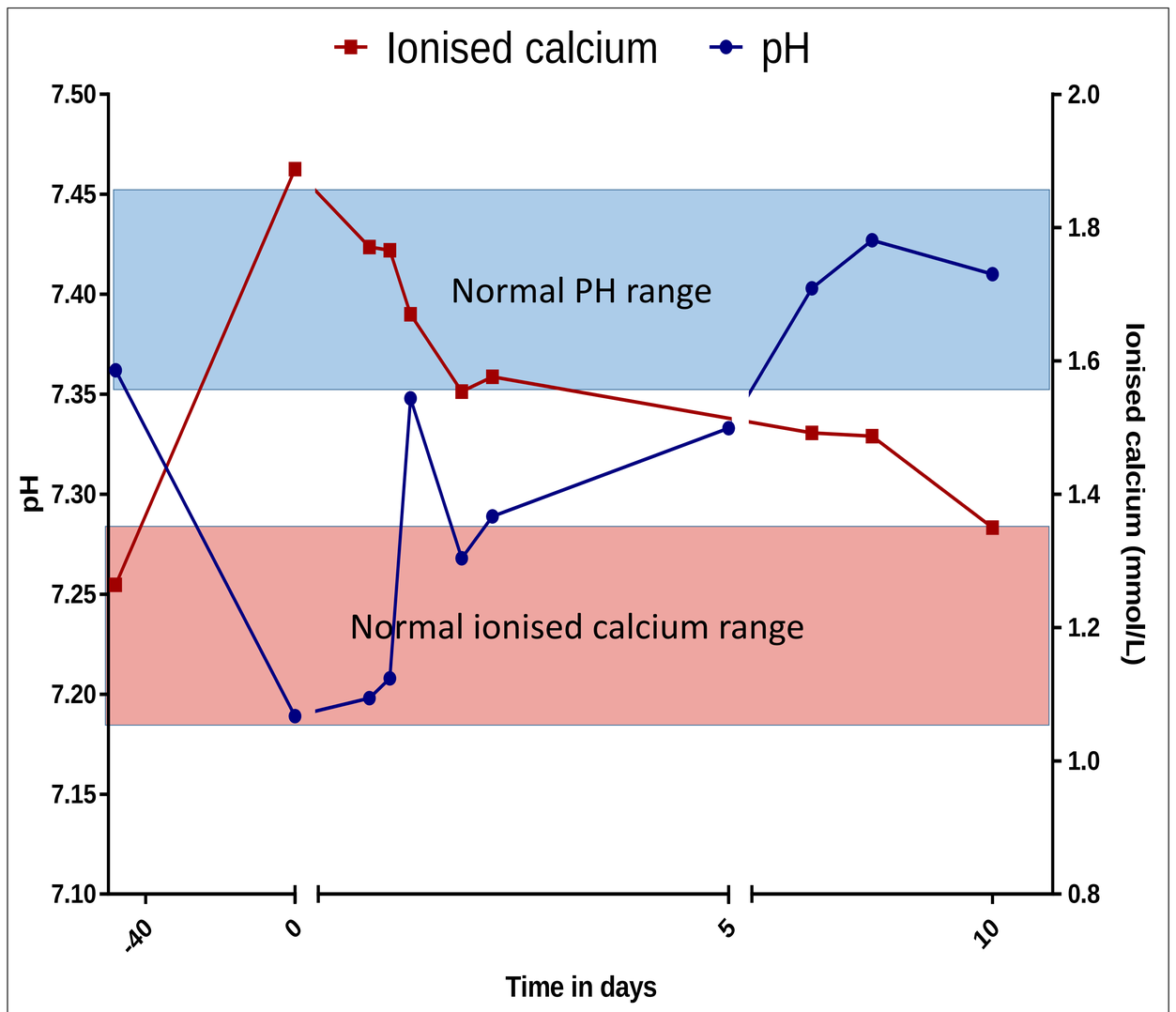
- Blood gas analysis revealed hyperchloremic metabolic acidosis (pH- 7.19, bicarbonate 17.7 mmol/l ,chloride- 114.1 mmol/l).
- Investigations for common aetiologies of hypercalcaemia were negative (Parathyroid hormone 1.1pmol/L, vitamin D- 69.1 nmol/L, Immunoglobulin (Ig) G- 12.55, IgM- 0.80, IgA- 5.07, Magnesium- 0.86 mmol/l, Phosphate-0.64 mmol/L, Alkaline phosphatase- 134 IU/l).

Management

- In the absence of an alternative explanation for his hypercalcaemia, a provisional diagnosis of proximal renal tubular metabolic acidosis was made and treatment with intravenous fluids and sodium bicarbonate was commenced.

Outcome

- The patient responded well to intravenous fluids and bicarbonate, and over the next few days, his hypercalcemia improved as his acidosis resolved. He was discharged once his calcium levels normalised.



Graph depicting pH and ionised calcium for our patient during this stay. Calcium acutely increased with simultaneous decrease in pH which resolved following hydration.

Discussion

- Sporadic cases of hypercalcaemia associated with distal RTA has been reported in new-borns and infants but has not been described in adults.
- Acidosis is thought to increase osteoclastic activity resulting in an increase in serum calcium. Alternatively, severe dehydration resulting in extracellular volume depletion, can lead to a reduction in eGFR with subsequent increased calcium reabsorption in the proximal convoluted tubules.
- To our knowledge, this is the first case of acute onset idiopathic hypercalcaemia associated with proximal renal tubular acidosis. Although rare, we feel it is important to recognise such an association as it would prompt early treatment of acidosis and fluid therapy to facilitate speedy recovery.

Learning points

1. It is important to follow a systematic approach to investigate the aetiology for acute onset hypercalcaemia.
2. Severe metabolic acidosis as a result of proximal renal tubular acidosis is a rare but easily reversible cause of acute onset hypercalcaemia.
3. Early initiation of fluid and bicarbonate therapy in such situations results in prompt resolution of hypercalcemia.

References

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