Case report

Hypercalcaemic hyperparathyroid crisis

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Learning Point for Clinicians

Hypercalcaemic hyperparathyroid crisis is a rare but dangerous life-threatening manifestation of parathyroid disease. Initial management includes aggressive rehydration and intravenous bisphosphonate therapy. Definitive management involves parathyroidectomy. Accurate identification and prompt management is required to avert potentially life-threatening complications of this condition.

Case presentation

A 63-year-old woman presented to the medical assessment unit with a three week history of vomiting, polydipsia, polyuria and constipation. She had no history of renal stones, osteoporosis or low mood. The patient had a longstanding history of hypertension for which she was receiving propranolol and type 2 diabetes mellitus, treated with metformin. She was mildly confused being orientated to person and place but not to time. She had an otherwise normal physical examination. Her pulse was 84, regular in rhythm and normal in character. Her blood pressure was 132/73. Respiratory rate was 20 and oxygen saturation was 97% on room air.

On admission, serum calcium was elevated at 4.48 mmol/l (normal range: 2.17–2.51 mmol/l) and phosphate was low at 0.71 mmol/l (0.87–1.45 mmol/l). Her creatinine measured 70 umol/l (49–90 umol/l) and eGFR was 62 ml/min (60–160 ml/min). Parathyroid hormone (PTH) was elevated at 1084 ng/l (15–65 ng/l) suggesting a diagnosis of primary hyperparathyroidism. A raised 24-h urinary calcium at 21.78 mmol/24 h (2.5–7.5 mmol/24 h) supported the diagnosis.

The patient was treated with aggressive intravenous fluid resuscitation and cardiac monitoring over 24 h. Adjusted calcium remained elevated at 3.98 mmol/l and intravenous bisphosphonate therapy was administered. On day 5 post admission, the serum calcium had decreased to 2.73 mmol/l. Subsequent parathyroid ultrasound and scintigraphy failed to demonstrate an abnormality. These scans are of importance when planning surgical excision of one or more abnormal parathyroid glands as surgery remains the definitive treatment for primary hyperparathyroidism.

Failure of these tests to demonstrate an abnormality necessitated a surgical exploration of the neck which revealed a grossly enlarged right superior parathyroid gland which was excised. The false negative rate of combined ultrasound and sestimibi imaging for localizing primary hyperparathyroidism has been reported as 4%.³ Although the level of hypercalcaemia raised clinical suspicion for malignancy, the gland weighed 3.7 g and was consistent with an adenoma on pathological examination.

At 1-month post-surgery, corrected calcium was 2.28 mmol/l, phosphate 0.78 mmol/l and PTH 135.3 ng/l. The latter value is likely explained by an undetectable vitamin D level and intramuscular replacement has taken place.
Discussion

A hypercalcaemic hyperparathyroid crisis is a rare life threatening complication of primary hyperparathyroidism.² It is currently defined as a syndrome characterized by a serum calcium level 3.5 mmol/l (>14 mg/dl) resulting from a marked elevation of PTH with severe signs and symptoms of hypercalcaemia that are reversible with correction of the hypercalcaemia.³

Fewer than 350 cases have been described in the literature to date.³ It is an emergency that requires aggressive medical therapy and early surgical treatment.

The presentation of severe acute hypercalcaemia associated with a hyperparathyroid state involves profound dehydration and impaired renal function, gastrointestinal disturbance and psychological disturbances. Cardiac arrhythmias, pancreatitis and neuromuscular disturbance also represent serious complications associated with this condition.⁴

Although solitary adenomas account for 75–85% of cases of primary hyperparathyroidism⁴ our case demonstrates an unusual situation in which the markedly elevated calcium and PTH levels usually associated with malignant hyperparathyroid disease occurred as a consequence of a benign hyperparathyroid adenoma.⁵ A tumour mass of 3.7 g would also be more suggestive of a malignant rather than a benign lesion (Figure 1).⁶

Rapid reduction in serum calcium reduces the signs and symptoms of the condition and serves to optimize the patient for elective parathyroidectomy. Initial medical management includes intravascular volume expansion with IV isotonic saline, use of furosemide to encourage calciuresis and bisphosphonate therapy to inhibit osteoclast function.³ Definitive management involves parathyroidectomy.

Conflict of interest: None declared.

References