LETTER TO THE EDITOR

Acute kidney injury, necrotizing pancreatitis and nephrocalcinosis: A rare presentation of primary hyperparathyroidism

Dear Editor:

Primary hyperparathyroidism is a common hormonal disorder presenting with hypercalcemia and non-suppressed or high parathyroid hormone (PTH) levels. This is due to a solitary adenoma in 80–85% of cases.[1] Parathyroid adenomas are most common in patients aged 50-70 years, and women are three times more likely to be affected.[1] Hyperparathyroidism can lead to high levels of calcium, causing non-specific symptoms to life-threatening complications. Sequelae such as acute kidney injury (AKI) and pancreatitis can also occur, with the latter being seen in <1.5% of patients.[2]

Herein, we present the case of a patient with severe hypercalcemia causing necrotizing pancreatitis and AKI requiring continuous renal replacement therapy (CRRT) with resolution after parathyroidectomy and medical management.

A 45-year-old female with known parathyroid adenoma presented with vomiting and abdominal pain. She was found to have serum calcium level of 16.2 mg/dL (reference range: 8.4–10.5 mg/dL), serum creatinine of 2.7 mg/dL (baseline of 1.3 mg/dL) and lipase > 2250 U/L. She was resuscitated with 5 liters of normal saline intravenous fluid and received four doses of calcitonin 4 U/kg intramuscularly.

Given patient's acute kidney impairment, a non-contrasted computed tomography of the abdomen was done. It was notable for bilateral nephrocalcinosis and pancreatitis (Fig. 1), although the patient denied any history of kidney stones. Repeat labs revealed an improving serum calcium level of 9.0 mg/dL although serum creatinine continued to worsen to 4.66 mg/dL. Other labs were notable for a serum phosphorus of 2.9 mg/dL (reference range: 2.2–4.5 mg/dL), PTH of 787 pg/mL (reference range: 15–65 pg/mL), PTH-related peptide 4.2 pmol/L (reference range: 0.0–3.4 pmol/L) and 25-OH Vitamin D level of 25 ng/mL (reference range: 30–80 ng/mL). In addition, point-of-care ultrasound identified right superior parathyroid adenoma.

She continued to decompensate and was eventually transferred to the intensive care unit for necrotizing pancreatitis with anuric kidney failure and respiratory failure. Subsequently, hypocalcemia to 6.6 mg/dL was noted and was presumed to be due to saponification from severe pancreatitis. Patient was initiated on CRRT with 2.5 mEq/L calcium dialysate, however her calcium levels trended up to 12 mg/dL so she was transitioned over to 0 mEq/L calcium dialysate. Despite the change in CRRT as well as the use of cinacalcet 30 mg twice a day orally for 3 doses and subsequent use of etelcalcetide 5 mg intravenously once, the patient continued to decline clinically. She underwent parathyroidectomy with removal of right superior parathyroid adenoma on Day 13. Preoperative PTH was 1282 pg/mL with post operative normalization of PTH to 79 pg/mL, phosphorus of 4.3 mg/dL and resolution of hypercalcemia to 8.2 mg/dL. This was eventually followed by kidney recovery, and the patient was transitioned off CRRT on Day 17.

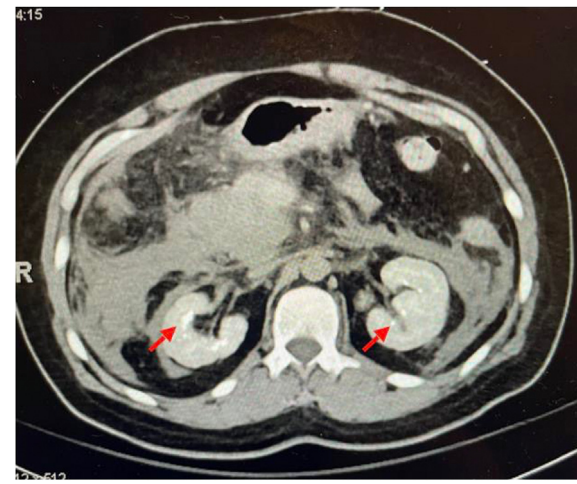

FIG. 1. CT Abdomen/Pelvis without contrast revealing nephrocalcinosis (red arrows) as well pancreatitis.

Primary hyperparathyroidism classically presents as 'bones, stones, abdominal moans and psychic groans'. Nephrocalcinosis and pancreatitis are rare complications of untreated hyperparathyroidism and the incidence has declined remarkably in the last few decades. The above case illustrates these infrequent sequelae and highlights the importance of appropriate investigation and timely treatment of primary hyperparathyroidism.

Pancreatitis as a diagnostic clue to otherwise undiagnosed hyperparathyroidism has been described as far back as 1957.[3] Patients may undergo recurrent episodes of pancreatitis prior to a diagnosis being made. This diagnosis can be elusive as hypercalcemia might not be present during the different stages of pancreatitis, as seen in our patient. Although pancreatitis in patients with

hyperparathyroidism has been a known entity for over half a century, it is seldom currently seen in practice due to readily available automated chemistry analyzers.[4]

Currently, the preferred treatment for symptomatic primary hyperparathyroidism is parathyroidectomy. In patients who are unable to have surgery, calcimimetics and bisphosphonates may be tried.

DECLARATION OF COMPETING INTEREST

None.

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