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Case Report

Acute Pancreatitis as a Manifestation of Primary Hyperparathyroidism: An Uncommon Association

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Abstract

The manifestation of primary hyperparathyroidism (PHPT) as acute pancreatitis (AP) is a rare entity. We report a case of a 55-year-old female, admitted to the intensive care unit of the general surgery department presenting with features of acute abdomen with a clinical and radiological diagnosis of AP. Further evaluation revealed hypercalcemia and PHPT due to parathyroid adenoma. Hypercalcemia due to PHPT plays a vital role in the pathogenesis of pancreatitis. The patient underwent parathyroidectomy and pathological examination of the parathyroid gland confirmed a parathyroid adenoma. After aggressive medical management of AP and hypercalcemia, parathyroidectomy has been shown to improve clinical outcomes and prevent further recurrences of pancreatitis.

Key words: Acute or chronic pancreatitis, Hypercalcemia, Parathyroid adenoma, Primary hyperparathyroidism

INTRODUCTION

Acute pancreatitis (AP) induced by hypercalcemia due to primary hyperparathyroidism (PHPT) is a rare event and the reported incidence range from 0.23% to 6.8%.[1] The causes of pancreatitis are largely dominated by gallstones and alcohol. PHPT is also considered a metabolic cause of acute or chronic pancreatitis (CP). Hypercalcemia secondary to the secretion of parathyroid hormone (PTH), is postulated to induce pancreatic injury through the activation of trypsinogen to trypsin and intraductal precipitation of calcium, leading to ductal obstruction and subsequent attacks of pancreatitis. Furthermore, genetic defects in (serine protease inhibitor Kazal Type 1 (SPINK 1) and cystic fibrosis transmembrane conductance regulator genes have been suggested as possible mechanisms of AP in the setting of hypercalcemia.[2] Approximately 1.5–13% of patients with PHPT develop AP. Normal or elevated calcium levels during acute or CP should always get attention.

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CASE REPORT

A 55-year-old hypertensive woman, presented to the casualty with complaints of upper abdominal pain, radiating to the back, for the past 2 days. She did not report any recent medication use or alcohol intake. On physical examination, she was hemodynamically stable and per abdominal palpation revealed severe epigastric tenderness with guarding. Initial laboratory studies revealed leukocytosis of 12 thousand/μL, hemoglobin 10.6 g/dL, creatinine 1.05 mg/dL, amylase, and lipase were elevated at 220 IU/L and 542 IU/L, respectively. Serum calcium level was found to be 15.5 mg/dL. Her alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and total bilirubin were normal. Contrast-Enhanced computed tomography of the abdomen and pelvis [Figure 1] showed features suggestive of AP with mildly enlarged pancreas with peripancreatic inflammation and no evidence of cholelithiasis or common bile duct dilatation. Soft tissue density nodules in the momentum, transverse mesocolon,

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and hepatorenal recess suggestive of fat necrosis and degenerative changes and end-plate sclerosis noted in the spine and bilateral sacroiliac joints with lysis of pars interarticularis of L4 and L5 vertebrae. Patient was initially managed conservatively with aggressive fluid therapy, I.V. Antibiotics, pain management, and three doses of Injection. zoledronic acid. After adequate resuscitation and stabilization of the patient, patient was discharged and readmitted 3 weeks later for further evaluation. On examination of the neck [Figure 2], she was found to have a swelling in the anterior aspect on the left side of size 3 cm × 3 cm, which moved with deglutition. Ultrasonography of the neck [Figure 3] showed complex mixed cystic solid lesion of size 5.1 cm × 3.2 cm × 3.4 cm (Volume 30 cc) arising from the inferior pole of the left lobe of the thyroid: TIRADS IV (moderately suspicious). About 5 ml of colloid-like fluid was aspirated and FNAC results showed the possibility of cystic colloid goiter in view of abundant colloid and occasional thyroid follicular cells. Given the absence of biliary, alcohol-induced, or medication-associated pancreatitis, hypercalcemia was considered to be the probable cause. Serum intact PTH level was found to be significantly elevated at 1002.8 pg/mL (12–88 pg/mL). 99mTc - Sestamibi parathyroid single-photon emission computerized tomography-computed tomography scan [Figure 4a and b] showed features suggestive of left inferior parathyroid adenoma. It also revealed altered trabeculae and lytic areas showing lace-like pattern and mildly increased 99mTc - Sestamibi uptake in the skull, bilateral scapulae, bilateral few ribs, multiple visualized cervical, and dorsal vertebrae likely Brown's tumor. Hence, proceeded with left parathyroidectomy with left thyroid lobectomy under general anesthesia. Procedure started with a transverse collar incision in the neck, followed by raising of superior and inferior flaps. Deep fascia and strap muscles were opened and retracted. Cystic mass [Figure 5a] of size 4 cm × 3 cm × 3 cm involving the left lower pole of thyroid extending from tracheoesophageal groove upto the suprasternal notch. The interior part of the cystic lesion had solid components of size 2 cm × 2 cm. The left lobe of the thyroid was mobilized medially and cystic mass was completely excised [Figure 5b] after delineating from the recurrent laryngeal nerve and inferior pole of the thyroid. The left superior parathyroid gland was identified and excised along with the left lobe of the thyroid. Hemostasis was secured and wound closed in layers. Wound closed in layers. Postoperatively, serial monitoring of serum calcium levels and PTH assays were done. Serum intact PTH level was found to be 0.02 pg/mL (12–88 pg/mL), on postoperative day 1. Patient showed a gradual drop in the serum calcium levels from postoperative day 1 (8.8 mg/dL) and was managed with intravenous calcium accordingly. Serum calcium levels were found to be within normal limits and

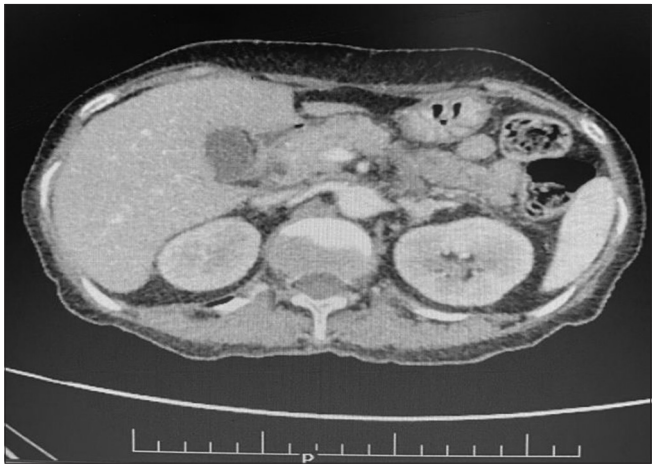

Figure 1: Contrast-Enhanced computed tomography of the abdomen showing features sug-gestive of acute pancreatitis with mildly enlarged pancreas with peri-pancreatic inflammation

Figure 2: Clinical image of swelling over the left lower anterior aspect of the neck

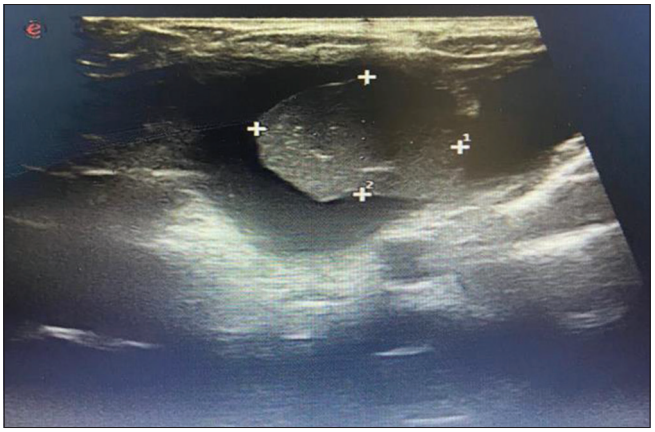

Figure 3: Ultrasonography of the neck showing Complex mixed cystic solid lesion arising from the inferior pole of the left lobe of the thyroid

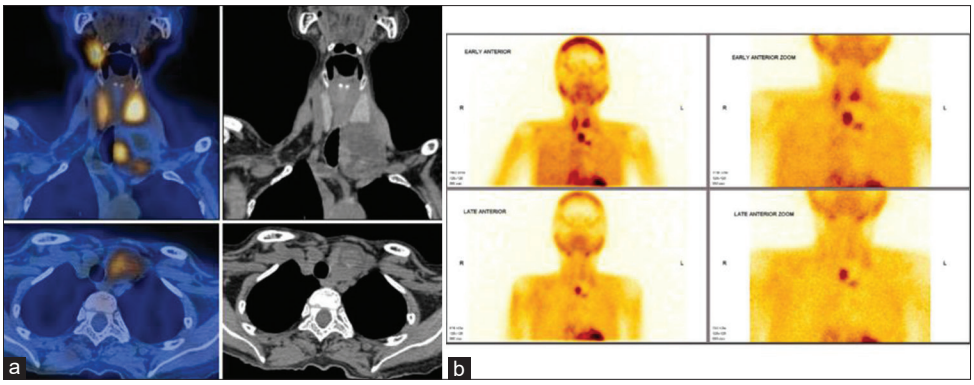

Figure 4: (a and b) 99mTc - Sestamibi parathyroid single-photon emission computerized to-mography-computed tomography scan showing features suggestive of left inferior parathy-roid adenoma

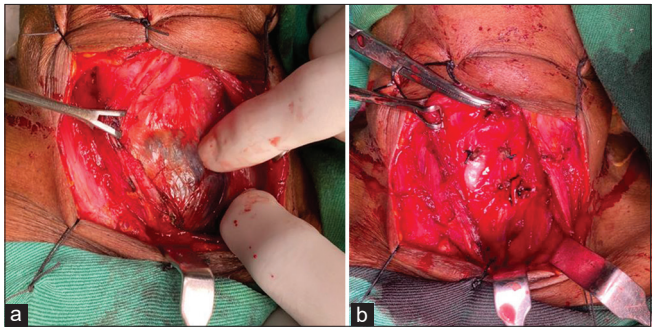

Figure 5: Intra-operative images of (a) Left parathyroid adenoma and (b) Post excision of the adenoma

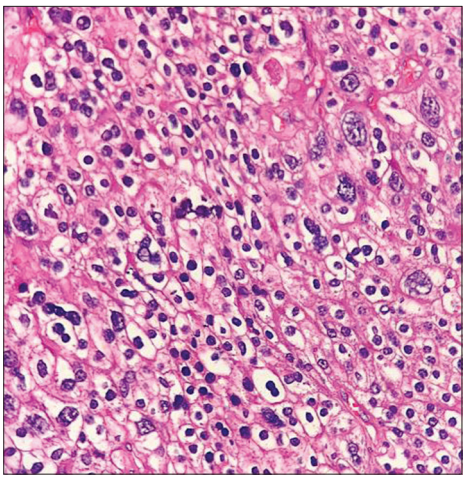

Figure 6: Microscopic image shows chief cells arranged in sheets with a foci showing tumor cells exhibiting nuclear atypia with bizarre nuclei, prominent nucleoli, and some spindle cells interspersed in fibro collagenous tissue

were managed with oral calcium supplementation upon discharge. Histopathological reports were consistent with features of atypical parathyroid adenoma. Thus, patient had a favorable outcome and did not develop any further attacks of pancreatitis or other complications of the procedure, on any of the follow-up visits [Figure 6].

DISCUSSION

The incidence of AP in PHPT was found to be no different than in the general population in recent studies, although the pancreatitis risk is known to be 10 fold elevated in PHPT. Biliary stones and alcohol are responsible for 80% of AP cases whereas the remaining 20% can be related to various etiologies such as infection, ductal obstruction, hypercalcemia of any cause, hyperlipidemia, impaired pancreatic perfusion, or idiopathic. Patients with PHPT have shown to have an increased incidence of pancreatitis, peptic ulcer disease, cholelithiasis, probably due to an increase in biliary calcium, which leads to the formation of calcium bilirubinate stones. Hypocalcemia is a poor prognostic factor in AP.[3] However, when hypercalcemia is detected in AP it should compel clinicians to search for alternative explanations such as malignancy or hyperparathyroidism. In this case, the presence of hypercalcemia in AP guided us to detect undiagnosed PHPT and a parathyroid adenoma. The absence of any other explanation for AP and clinical improvement after parathyroidectomy supports a likely relationship between PHPT and AP in this case.

CONCLUSION

The occurrence of acute or CP is rarely associated with PHPT. The pointer to find the parathyroid adenoma was the hypercalcaemia. This case report highlights the importance of fully investigating for PHPT in a pancreatitis patient with high normal or raised serum calcium level, especially in the absence of other common causes of pancreatitis. The return of serum calcium levels to normal values and remission of AP episodes after surgical intervention, thus suggests a positive cause and effect relationship between hyperparathyroidism and AP.

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