CASE REPORT

Acute Pancreatitis predating the Diagnosis of Parathyroid Adenoma

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Abstract

Background and aim: Parathyroid adenoma-induced hypercalcemia and acute pancreatitis are known but rare.

Case description: We report a case of 30 years male patient, nonalcoholic with acute pancreatitis associated with hypercalcemia as the first manifestation of primary hyperparathyroidism (PHPT). A parathyroid nuclear scan suggested parathyroid adenoma of the left inferior parathyroid gland. Initially, acute pancreatitis was treated conservatively. The patient subsequently underwent surgical resection of the parathyroid adenoma. Postoperatively, his clinical symptoms of pancreatitis did not subside with no improvement in ultrasonography. He was readmitted and a surgical cystogastrostomy was performed.

Conclusion: Some mechanisms have been proposed for its pathophysiology. We aim to treat the causative factor.

Clinical significance: Therefore, the cause of hypercalcemia should be identified at the earliest followed by appropriate treatment.

Keywords: Acute pancreatitis, Hypercalcemia, Parathyroid adenoma.

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Background

Acute pancreatitis is most commonly diagnosed in a patient meeting two of three criteria—epigastric abdominal pain radiating to the back, increased serum lipase or amylase by three folds, and findings on cross-sectional abdominal imaging.[1] Acute pancreatitis is largely associated with gallstones (30–60%) and alcohol (15–30%).[1,2] Primary hyperparathyroidism-induced hypercalcemia causing acute pancreatitis is very rare and was first described in 1957 by Cope et al.[3] The prevalence of acute pancreatitis in patients with PHPT is estimated up to 7%[3–7] with a female predilection.[8,9] In India it is mostly seen in young adults, whereas it is diagnosed at an older age in US and France.[4] We present a case report of a young male patient with hypercalcemia-induced acute pancreatitis as the first manifestation of a benign parathyroid adenoma.

Case Description

A 30 years old male patient, nonalcoholic with no relevant past medical and drug history presented with a sudden attack of severe epigastric pain and vomiting. The pain was 8/10 in severity, radiating to the back with no exacerbating or relieving factors. The patient was afebrile with a blood pressure of 120/80 mm Hg, a heart rate of 80 beats per minute, and oxygen saturation of 98% on room air. On clinical examination, the abdomen was distended with severe tenderness noted in the epigastric region. On percussion resonant note was heard.

In blood analysis, his leucocyte count was in the normal range of 9290 cells/cm. Serum calcium was elevated at 16.44 mg/L. Pancreatic enzymes, serum amylase was 465 U/L and serum lipase 3450 U/L, which was more than three folds of normal values. On ultrasonography visualized, the head and body of the pancreas appeared mildly bulky and the pancreatic duct was mildly dilated 4–5 mm.

With clinical, blood analysis, and ultrasonographic findings, a diagnosis of acute pancreatitis was given. He was kept nil per oral, started on intravenous isolate M fluid 100 mL/hr, analgesics, and regular monitoring. However considering his high serum calcium, ultrasonography of the neck was advised which was reported as a 19 × 12 mm round to oval lesion inferior to the lower pole of the left lobe of the thyroid gland, serum level of intact parathyroid hormone (iPTH) was 750.3 pg/mL. The parathyroid nuclear scan was performed using Tc99m which was suggestive of functioning parathyroid adenoma below the region of the lower pole of the left lobe of the thyroid gland (Fig. 1). Considering above mentioned details a diagnosis of parathyroid adenoma of the left inferior parathyroid gland was given.

The patient underwent surgical resection of the tumor of the left inferior parathyroid gland and histopathological examination confirmed the diagnosis of parathyroid adenoma (Fig. 2). Immediate postoperative iPTH was 59.4 pg/mL, which was repeated after 8 hours and was reported as 10.2 pg/mL. Intact parathyroid hormone was restored to normal by the 4th postoperative day to 78.5 pg/mL. Clinically he developed symptoms of tetany from the 1st postoperative day for which intravenous calcium gluconate was started and was withdrawn by the 5th postoperative day after the symptoms subsided. In the care of pancreatitis signs and symptoms, he was started on conservative management. He was discharged and was called for regular follow-up.

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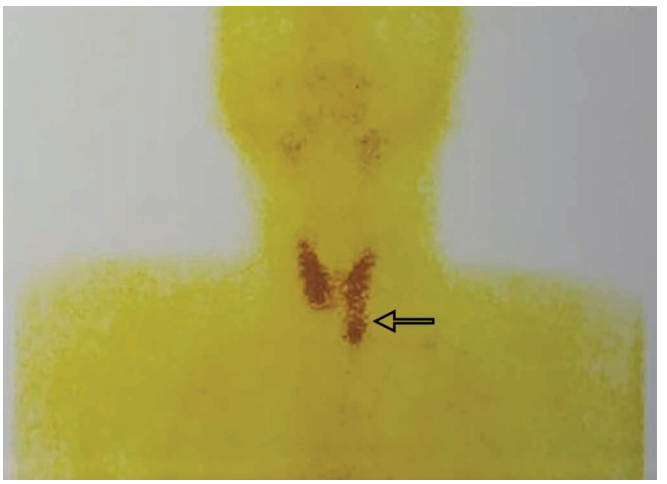

Fig. 1: Nuclear scan using Tc99m image showing functioning parathyroid adenoma below the region of the lower pole of the left lobe of the thyroid gland

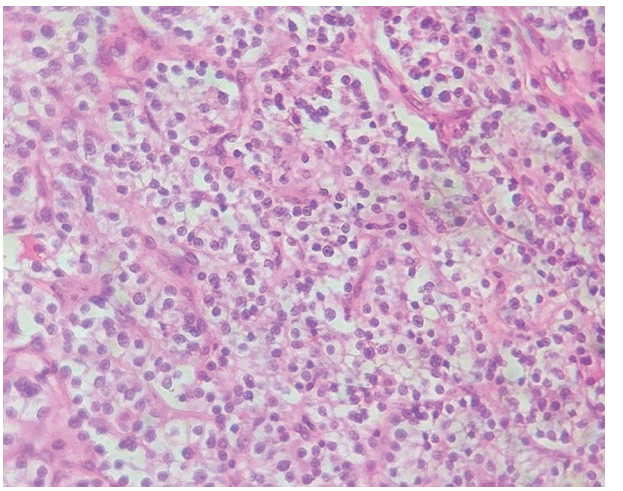

Fig. 2: The histopathologic section showing sheets of tumor cells separated by a fibrovascular stroma. Cells are round and monomorphically arranged in the acinar pattern—parathyroid adenoma

After 3 months postoperative his clinical symptoms of pancreatitis did not subside with no improvement in ultrasonography. He was readmitted and a surgical cystogastrostomy was performed. He recovered well and remained free of symptoms.

Discussion

Primary hyperparathyroidism occurs in 1% of the adult population and about 2% in the population older than 55 years.[8] The main etiology of PTHP is single or double parathyroid adenoma in 80%, hyperplasia of all parathyroid glands in 15–20%, and 2% due to malignancy of the parathyroid gland.[10] Most of the adenomas are located in the neck with about 6% in an ectopic location. Identification of adenomas can be done with ultrasonography (sensitivity of 27–89%) and parathyroid nuclear scan (sensitivity of 80–90%).[10] Surgical resection of the parathyroid gland tumor, histopathological confirmation, and regular follow-up are the ultimate therapy.[3]

Pancreatitis is generally related to hypocalcemia, with a decrease in serum calcium paralleling the severity of the acute process.[3,11,12] If pancreatitis is present along with hypercalcemia then one should be alerted to the presence of hyperparathyroidism or malignancy for which iPTH, ultrasonography of the neck becomes important. Serum calcium rises to more than 1.3 times the normal value which was also noted in our case.[4] The risk factor of pancreatitis with the evidence of PHPT multiplies by the factor of 10–30,[4,7,12] however many studies have found that there was no relation between PHPT and pancreatitis when compared to the general population.[3,6]

Acute pancreatitis in PHPT could be attributed to vascular thrombi.[11] As a result of hypercalcemia, there are few pathophysiological mechanisms suggested. First, pancreatic duct obstruction is a result of the deposition of calcium.[3–5,9,11] Second, calcium is an essential cofactor inactivation of trypsinogen within the pancreatic parenchyma, it increases the stability and activity of trypsin by binding to the N terminal aspartyl residues of trypsinogen activation peptide and thus eliminating their inhibitory effect on autocatalytic activation causing autodigestion of pancreas.[3–5,13,14] Third, hypercalcemia induces the release of cholecystokines through a cholinergic pathway which causes stimulation of pancreatic acinar cells to cause the activation of pancreatic proteases.[13] Hypercalcemia in combination with N34S SPINK1 (Serine Protease Inhibitor Kazal type I) and CFTR (Cystic Fibrosis Transmembrane conductance Regulator) mutation is one of the explanations which can be considered a disease-modifying factor in PHPT.[3,12,14]

We presented this rare case of nonalcoholic young adult here with acute pancreatitis as the first clinical manifestation of parathyroid adenoma hence clinician must consider for evaluation PHPT presents with hypercalcemia.

Clinical Significance

Pancreatitis as a consequence of the PHPT can be managed by surgical removal of adenoma followed by regular follow-up. If the symptoms persist surgical management of pancreatitis is advisable.

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