Gastric Outlet Obstruction Following Recurrent Pancreatitis Uncovers a Giant Parathyroid Adenoma: A Case Report

Brijesh Kumar Singh,1 Toshib GA,1 Yashwant Singh Rathore,1 Shipra Agarwal,2 Sunil Chumber,1 Nishikant Damle3

1Department of Surgical Disciplines, All India Institute of Medical Sciences, New Delhi, India
2Department of Pathology, All India Institute of Medical Sciences, New Delhi, India
3Department of Nuclear Medicine, All India Institute of Medical Sciences, New Delhi, India

Abstract

A 35-year-old female presented with abdominal pain, fever, projectile vomiting, and a diffuse tender epigastric mass. She was diagnosed to have acute persistent pancreatitis with a pancreatic pseudocyst. Elevated serum calcium levels provided an etiologic link between hypercalcemia and pancreatitis. On examination, a nodule was found in the left side of her neck which was later diagnosed as a giant left inferior parathyroid adenoma. This report highlights the critical analysis of history, examination, and investigations to reach an ultimate diagnosis. Pseudocyst drainage and parathyroidectomy resolved her symptoms.

Key words: giant parathyroid adenoma, pancreatitis, gastric outlet obstruction, primary hyperparathyroidism

INTRODUCTION

Acute pancreatitis, an inflammation of the pancreas, can result in life-long morbidity and even mortality if not treated appropriately. Pancreatitis most commonly results from gallstone disease, alcohol intake, and following procedures like endoscopic retrograde cholangiopancreatography. Primary hyperparathyroidism (PHPT) mediated hypercalcemia is comparatively a less common cause of pancreatitis (1.5-13%).

Although PHPT is a common endocrine disorder, in Western literature, approximately 75-80% of patients are asymptomatic and are detected incidentally by routine calcium screening. However, in our setup, due to lack of awareness and routine screening, patients are diagnosed in the symptomatic stage with the most common presenting features being bone pain and metabolic myopathy. Seven percent of PHPT cases develop pancreatitis.

In this case report, we describe a young woman presenting with abdominal pain and non-bilious projectile vomiting. The features were suggestive of gastric outlet obstruction due to recurrent pancreatitis and pancreatic pseudocyst. An incidental finding of hypercalcemia was the only clue to an underlying PHPT leading to a whole gamut of signs and symptoms.

CASE

A 35-year-old female presented to the emergency department complaining of intermittent abdominal pain over 3 months which became severe over the past two days. It was associated with fever and persistent postprandial non-bilious vomiting for seven days. She had a history of intermittent generalized body ache and epigastric pain which subsided after pain relievers. She also sustained blunt trauma to the abdomen after a fall, following which she had severe pain in the epigastric region.

She consulted in the Gastroenterology department where she was diagnosed to have acute pancreatitis based on blood investigations showing raised serum amylase (112 U/L; normal range: 28 - 100 U/L) and lipase levels (138 U/L; normal range: 13 - 60 U/L). A contrast-enhanced computed tomography (CT) of the abdomen showed a bulky pancreas...
with peripancreatic fluid collection in the head, body and tail.

During this episode, her vital signs were stable. There was no pallor, icterus, cyanosis, or edema. Examination of her abdomen revealed a diffuse, firm, and tender epigastric mass which did not move with respiration. Given the present findings and clinical background, recurrent pancreatitis with pancreatic pseudocyst was considered.

**Investigations**

Ultrasound (USG) of her abdomen showed necrotic areas in the head of the pancreas with intra- and peripancreatic fluid collection. Contrast-enhanced CT (CECT) abdomen showed peripherally enhancing collection in the pancreatic bed replacing the entire pancreas with extension into the lesser sac and gastro-hepatic region with few air foci within suggestive of walled-off necrosis, likely a pseudocyst (Figure 1). These features were consistent with residual pancreatitis with a pseudocyst compressing the stomach leading to gastric outlet obstruction.

Hematologic parameters were normal except for leukocytosis (total count – 18000/mm³). Blood chemistry showed normal serum amylase (38 U/L), lipase (25 U/L), and renal parameters. However, a remarkable finding was hypercalcemia (blood ionized calcium, iCa²⁺ - 1.9 mmol/L; normal range: 1.1 – 1.4 mmol/L). Further evaluation of the cause of this hypercalcemia revealed raised total serum calcium (12.3 mg/dL; normal range: 8.6 – 10 mg/dL), and markedly raised parathormone (PTH) levels (PTH - 759 pg/mL; normal range: 15 – 65 pg/mL) with mild hypophosphatemia (serum phosphorus - 2.1 mg/dL; normal range: 2.5 – 4.5 mg/dL).

After noting the hypercalcemia, her neck was re-examined and a firm palpable nodule was discovered in the lower left side. Neck USG revealed a 2.3 x 1.1 x 1.8 cm hypoechoic lesion inferior to the left lobe of the thyroid. Further 4-dimensional CECT revealed a 1.5 x 1.5 cm lesion, posterolateral to the left lobe of the thyroid, with arterial enhancement and washout, suggesting a left inferior parathyroid adenoma. These findings were concordant with the results of the ⁹⁹mTc-Flurorocholine PET/CT scan of the patient (Figure 2).

**Differential diagnosis**

The possible causes of PHPT were parathyroid adenoma, parathyroid cyst, cystic adenoma and parathyroid carcinoma. USG, 4-dimensional CT, and ⁹⁹mTc-Flurorocholine PET/CT imaging studies ruled out the possibility of a thyroid nodule or ectopic parathyroid.

**Treatment**

The patient was finally diagnosed to have a left inferior parathyroid adenoma and hypercalcemia-induced recurrent pancreatitis with a pancreatic pseudocyst manifesting as gastric outlet obstruction. She was stabilized, a Freka’s nasojejunal tube was inserted for feeding and a CT-guided abdominal external drain was placed to decompress the pseudocyst. The patient was prepared for focused parathyroidectomy in an elective setting with preoperative endocrine and anesthesia consults.

Intraoperatively, a parathyroid nodule was identified at the inferior pole of the left lobe of the thyroid gland and was excised (Figure 3). Gross examination of the surgically excised parathyroid specimen revealed a 2.8 cm x 3 cm globular and well-circumscribed tumor weighing 4.8 grams (Figure 4). Histological examination of the specimen was compatible with a benign adenoma (Figure 5).

Intact parathormone (iPTH) levels declined drastically in the immediate postoperative period (from 759 pg/mL to 67.25 pg/mL). Serial monitoring of serum calcium revealed hypocalcemia on the second postoperative day (serum total calcium - 8.0 mg/dL) and third postoperative day (serum total calcium - 7.8 mg/dL) accompanied by tingling and
numbness of the perioral region and extremities. These features were suggestive of hungry bone syndrome and managed with intravenous and oral calcium with vitamin D supplements.

The patient improved over 2 weeks with the resolution of abdominal pain. Postoperative abdominal USG revealed resolving pancreatitis. The nasojejunal tube was removed on the eighth postoperative day. The abdominal drain was removed on the tenth postoperative day. Oral feeding was gradually increased and was tolerated. She was discharged on the twelfth postoperative day. At 8 months follow-up, she was doing well and her serum calcium and parathormone levels were normal.

Figure 2. $^{18}$F-Fluorocholine PET/CT scan showing transaxial image of intense tracer uptake behind left lobe of thyroid suggesting parathyroid hyperactivity (arrow).

Figure 3. Intraoperative findings of focused parathyroidectomy showing enlarged left parathyroid.

Figure 4. Schematic diagram showing the size and location of the parathyroid specimen with a cut section depicting gross features of adenoma.
The incidence of PHPT-associated acute and chronic pancreatitis is almost similar in the Indian population, but acute pancreatitis predominates in the Western population. Among patients with acute pancreatitis, 7% develop pancreatic pseudocysts, while among those with chronic pancreatitis, 30-40% develop pancreatic pseudocysts. Approximately 50% of these may remain asymptomatic or regress spontaneously; the rest may develop acute or chronic complications in the form of infection, bleeding, rupture, bile duct dilatation, gastric outlet obstruction or thrombosis of the portal or splenic vein. Gastric outlet obstruction due to pressure effect has been reported in 8% of acute pancreatitis (due to any cause) related pseudocysts and 15% of chronic pancreatitis (due to any cause) related pseudocysts. There is no consensus on whether surgical drainage is required for acute pancreatitis-induced pseudocyst without infection. However, persistent vomiting from gastric outlet obstruction may predispose to malnutrition. Therefore, most clinicians agree that surgical drainage is an effective treatment for pancreatic pseudocyst with features of gastric outlet obstruction. Symptoms of bone pain, recurrent nephrolithiasis, neuromuscular weakness and psychiatric disorders must raise the suspicion of parathyroid disease and prompt further evaluation. Elevated levels of serum calcium and PTH confirm the diagnosis. Localization of parathyroid adenoma is done by imaging studies like cervical USG, sestamibi scintigraphy, and
magnetic resonance imaging (MRI). A Tc99m Sestamibi scan of the parathyroid glands has a sensitivity of 92% while neck USG has a sensitivity of 80% for the preoperative localization of parathyroid adenomas. Newer modalities like PET-CT imaging with 18F-fluorocholine (18F-FCH) tracer carry advantages in terms of detection of smaller adenomas and reduced scanning time with a high sensitivity of 92% and specificity of 100%. Moreover, PET-CT 18F-FCH has an advantage in identifying smaller lesions very close to the thyroid, low gland mass, and multiple gland involvement.

In this case, the diagnosis was made based on the elevated serum calcium and PTH levels. Initial screening of the neck was conducted using USG, while localization of the lesion was done by 4D-CT. We also performed a PET-CT 18F-FCH to rule out ectopic parathyroid tissue.

Surgical excision of the parathyroid adenoma is the preferred treatment. Documenting intraoperative or immediate postoperative decline in IPTH levels is important to confirm successful removal of the adenoma. Close monitoring of serum calcium levels in the postoperative period is crucial for the diagnosis and management of life-threatening hypocalcemia. Long-term follow-up is equally important as the postoperative course can be complicated by episodes of acute or chronic pancreatitis. However, various studies have shown significant improvement in abdominal symptoms and a low recurrence of pancreatitis when normocalcemia is achieved following removal of the parathyroid adenoma. Our patient’s symptoms also improved without any recurrence of pancreatitis over 8 months of follow-up.

CONCLUSION

The present case report emphasizes the importance of thorough history taking and clinical examination to reach a final diagnosis. It is equally important to analyze laboratory investigations and their correlation with clinical signs and symptoms because PHPT may have myriad manifestations, and pancreatitis in a young, non-alcoholic patient warrants evaluation of endocrinologic causes.

Ethical Consideration

Patient consent was obtained before the submission of the manuscript.

Statement of Authorship

All the authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

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