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General Medicine

PARATHYROID ADENOMA AND ITS UNUSUAL PRESENTATION: A CASE OF NECROTISING PANCREATITIS

KEY WORDS: Parathyroid Adenoma, Hypercalcemia, Primary Hyperparathyroidism (phpt), Necrotizing Pancreatitis

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ABSTRACT

Primary hyperparathyroidism (PHPT), often caused by a parathyroid adenoma, is a rare but important cause of hypercalcemia. Though PHPT typically presents with symptoms of bone pain, kidney stones, and gastrointestinal disturbances, it can occasionally lead to acute pancreatitis. Hypercalcemia-induced pancreatitis, while uncommon, presents a diagnostic challenge due to its non-specific symptoms and overlapping causes. We report the case of a 60-year-old female who presented with persistent epigastric pain ,vomiting and loss of apetite for over two months .She was diagnosed to have acute on chronic necrotising pancreatitis with persistent and worsening hypercalcemia ,with elevated serum intact parathyroid hormone levels (iPTH) which was diagnosed to be secondary to Parathyroid adenoma with the help of ultrasound neck and Sestamibi scan. Our objective is to highlight how crucial it is to take PHPT into account in patients with pancreatitis, without additional risk factors ,especially with persistent or worsening hypercalcemia and the effective role of parathyroidectomy in clinical resolution of such patients.

INTRODUCTION

Approximately 85% of instances of primary hyper-parathyroidism (PHPT) are caused by parathyroid adenomas [1]. The hallmark of PHPT is elevated parathyroid hormone (PTH) release. Increased calcium resorption from bones, improved absorption in the intestines, and decreased calcium excretion by the kidneys are the three main ways that elevated PTH levels cause hypercalcemia [2]. While many individuals with PHPT are asymptomatic, others present with symptoms related to hypercalcemia, such as fatigue, muscle weakness, renal stones, or bone disease. The condition can also manifest with gastrointestinal disturbances, including nausea, constipation, and in severe cases, acute pancreatitis [3]. This variable clinical presentation of PHPT often making diagnosis challenging.

Although the correlation between hypercalcemia and acute pancreatitis is well-established, it only manifests in a minor portion of PHPT patients. Multiple hypotheses have been attributed to the pathophysiology of pancreatitis secondary to hypercalcemia [5,6].

The treatment of pancreatitis in hypercalcemia involves not only addressing the pancreatic inflammation but also correcting the underlying metabolic disturbance by reducing serum calcium levels [8].

The management of PHPT typically involves surgical excision of the adenoma to restore normal PTH and calcium levels. Preoperative localization of the adenoma is achieved through imaging techniques such as ultrasound or sestamibi scans, which guide the surgeon during parathyroidectomy. Following surgery, patients generally experience a rapid decline in calcium levels and resolution of symptoms [9,10].

This case report is presented to highlight a rare and complex presentation of PHPT with hypercalcemia leading to necrotizing pancreatitis in a 60-year-old female. Given the rarity of this association, this example highlights the need of taking hypercalcemia into account as a possible cause of pancreatitis, highlighting the necessity of an extensive evaluation to ensure early diagnosis and intervention.

Case Presentation

A 60-year-old woman presented with complaints of vomiting and appetite loss that had persisted for the previous two months. The vomiting episodes, described as yellowish green in color, were non-blood-stained and non-foul smelling. Along with nausea, she experienced significant epigastric pain, which was exacerbated by food intake. She also noticed an unquantified weight loss during this period. However, there was no history of fever, diarrhea, jaundice, cough, or any other respiratory symptoms.

The patient's past medical history revealed that she had recently been diagnosed with type 2 diabetes mellitus and was started on oral metformin (500 mg daily). She had no history of alcohol consumption or prior surgeries, nor was there any family history of similar symptoms.

The patient was found to be aware and cognizant of time, location, and people during the physical examination. There was pallor. All of her vital signs were stable.

There was no palpable organomegaly during the abdominal examination, however there was some slight discomfort in the epigastric area. The results of the cardiovascular examination, respiratory examination and neurological examination were grossly normal.

Baseline laboratory investigations were done (Table 1) which revealed anemia, hypokalemia, with elevated lipase levels, elevated ESR and borderline hypercalcemia.

Parameter	Value (normal range)	Parameter	Value (normal range)
Hemoglobin	9 g/dl (12-15)	Total bilirubin	0.72 mg/dl (0.1-1.2)
Total count	8900/cu.mm (4000-11000)	SGOT	20 U/L (0-35)
Platelet	4.23 lakhs/ cu.mm (1.5-4.5 lakhs)	SGPT	10 U/L (0-41)
BUN	4 mg/dl (7-18)	ALP	99 U/L (45-129)
Creatinine	0.5mg/dl (0.6-1.3)	Albumin	2.5 g/dl (3.2-4.8)
Sodium	145 mmol/L (134-144)	Globulin	3.6 g/dl (2-3.5)
Potassium	2.6 mmol/L (3.5-5)	Amylase	59 U/L (22-80)
Bicarbonate	23 mmol/L (21-29)	Lipase	136 U/L (<60)
Corrected calcium	10.2 mg/dl (8.5-10.1)	ESR	55 mm (4-19)

An abdominal ultrasound was conducted, the results showed heterogenous head of pancreas with dilated main pancreatic duct of 5mm, with distal dilated CBD of 10mm.

Then, we proceeded with CECT abdomen which showed necrotising type of acute on chronic pancreatitis- Modified CT severity score 10/10, with multiple hypodense hypoenhancing areas seen throughout the pancreatic parenchyma, largest measuring 2.4 x 1.5 cm in the neck of pancreas, with features suggestive of walled off necrosis (Figure 1). Also showed complete splenic vein thrombosis with mild splenomegaly. Common bile duct appeared dilated measuring 11-12mm with abrupt tapering within the pancreas.

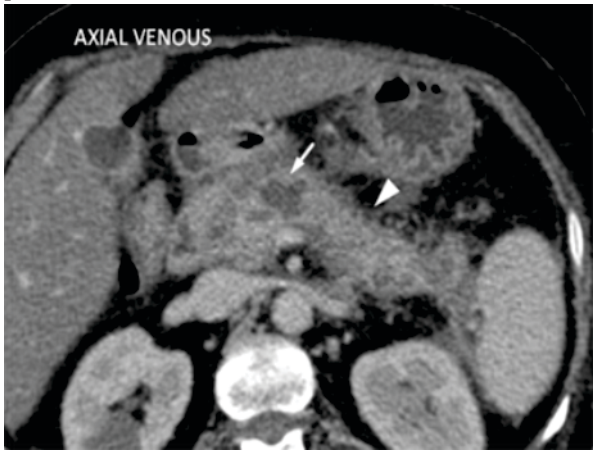

Figure 1: CT abdomen showing hypodense hypoenchancing areas within the pancreatic parenchyma (white arrow) and peripancreatic fat stranding (white arrowhead) suggestive of acute on chronic pancreatitis (necrotising type).

MRCP abdomen was done to rule out malignancy which confirmed the findings on CECT abdomen with a short segment stricture (measuring ~ 4.7 mm) noted in the mid common and thus causing upstream dilatation of the common hepatic duct measuring 9.0 mm and proximal portion of common bile duct measuring ~ 10.0 mm.Distal to the stricture the common bile duct appears normal in caliber (Figure 2).

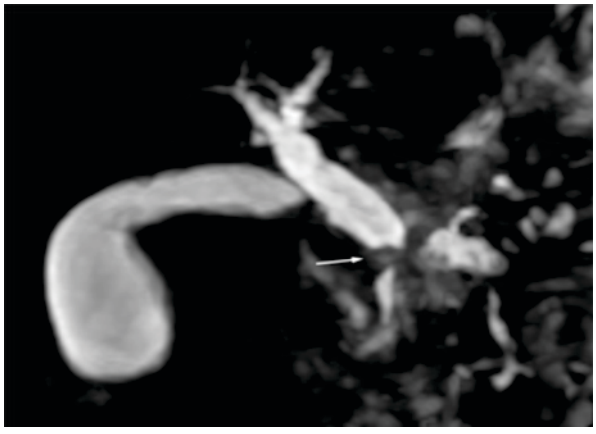

Figure 2: MRCP showing short segment stricture of 4.7 mm in the mid common bile duct with upstream dilatation of the common hepatic duct and proximal portion of the common bile duct.

CA 19-9 levels were normal.

Serial monitoring of calcium levels was done (Table 2).

DATE	26/4/24	1/5/24	5/5/24	10/5/24
Corrected Calcium (mg/dl)	10.2	10.5	11.4	11.7

The presence of persistent and worsening hypercalcemia raised suspicion of a possible endocrine disorder. Hypercalcemia workup revealed an elevated parathyroid hormone (iPTH) level of 172 pg/ml, prompting further evaluation.

A neck ultrasound revealed a well defined ovoid homogeneous hypoechoic lesion, measuring 2.0 x 1.0 x 2.5 cm noted caudal to the echogenic thyroid capsule of the inferior pole of right lobe of thyroid gland with internal vascularity on colour doppler, suggestive of a parathyroid adenoma (Figure 3).

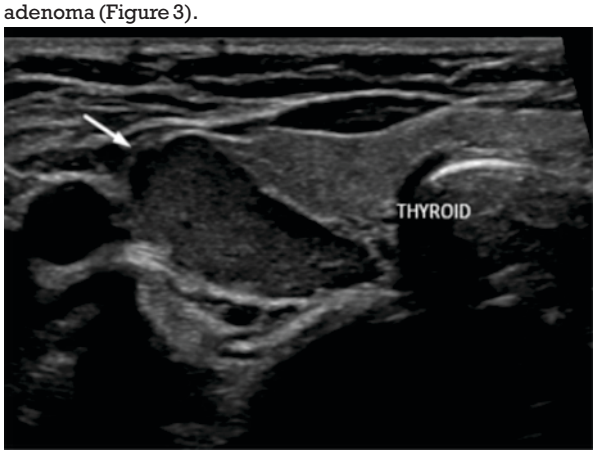

Figure 3: Ultrasound neck showing a well defined ovoid homogenous hypoechoic lesion (white arrow) caudal to the echogenic thyroid capsule of the inferior pole of the right lobe of the thyroid gland- suggestive of right inferior parathyroid adenoma.

This diagnosis was confirmed by a Sestamibi scan, which showed abnormal uptake in the right inferior parathyroid gland.

The patient was taken up for right inferior parathyroidectomy, with pre gland excision PTH-423 and post gland excision PTH-142 and the histopathological examination confirming the diagnosis of parathyroid adenoma.

Repeat calcium post surgery normalised- 8.2 mg/dl.

She made a full recovery and was discharged in stable condition.

DISCUSSION

This case highlights a complex interplay between hypercalcemia, parathyroid adenoma, and necrotizing pancreatitis. Hypercalcemia, particularly when persistent, often raises suspicion of an underlying parathyroid pathology, as was the case in this 60-year-old female. In the context of her clinical presentation, the symptoms of vomiting, loss of appetite, and epigastric pain with weight loss initially pointed towards local cause of pancreatitis, but the elevated serum calcium levels and subsequent investigations directed attention to an endocrine etiology, ultimately leading to the diagnosis of PHPT secondary to a parathyroid adenoma.

Primary hyperparathyroidism is one of the most common causes of hypercalcemia, with parathyroid adenomas accounting for 80-85% of cases [1]. The classic presentation includes "stones, bones, abdominal groans, and psychic moans, referring to kidney stones, bone pain, gastrointestinal disturbances, and neuropsychiatric symptoms, respectively. In this patient, gastrointestinal symptoms were prominent, consistent with the abdominal manifestations of hypercalcemia, such as nausea, vomiting, and epigastric pain. Hypercalcemia results from increased bone resorption, renal reabsorption of calcium, and increased gastrointestinal calcium absorption due to elevated parathyroid hormone (PTH)" [2].

This patient's hypercalcemia was initially moderate, but serial monitoring revealed a growing trend, which is indicative of primary hyperparathyroidism. High calcium levels can impact several organ systems, resulting in symptoms such as exhaustion, weakened muscles, and digestive issues. However,

in certain instances, acute pancreatitis—a well-known but uncommon side effect of primary hyper-parathyroidism—can also be brought on by hypercalcemia [4].

Acute pancreatitis has multiple etiologies, with gallstones and alcohol being the most common causes. However, hypercalcemia is a known, though less frequent, precipitant of pancreatitis. Although the precise process by which hypercalcemia causes pancreatitis is unclear, it is believed to be related to the impact of calcium on the cells that line the pancreatic acinar lumen. Calcium is believed to activate pancreatic enzymes within the acinar cells, leading to autodigestion and inflammation of the pancreas [5]. Hypercalcemia may also lead to the formation of calcium stones in the pancreatic ducts, contributing to ductal obstruction and subsequent pancreatic inflammation [6].

A severe variant of the illness with a high morbidity and death rate is necrotizing pancreatitis [7]. This patient's Modified CT Severity Index (MCTSI) score of 10/10 indicated severe pancreatitis, requiring intensive treatment that included electrolyte replacement, hydration, and monitoring for consequences [8]. The association between the patient's hypercalcemia and pancreatitis further supported the diagnosis of primary hyperparathyroidism as the underlying cause.

The definitive treatment for primary hyperparathyroidism caused by a parathyroid adenoma is surgical removal of the adenoma [9]. This patient underwent a successful right inferior parathyroidectomy, as confirmed by intraoperative findings and the subsequent normalization of her PTH and calcium levels [10].

Histopathological examination of the excised lesion confirmed the diagnosis of parathyroid adenoma, as it demonstrated typical features of a benign neoplastic process without evidence of malignancy or capsular invasion. The patient showed symptomatic improvement following surgery, with resolution of her gastrointestinal symptoms and normalization of her biochemical abnormalities.

Our case report aligns with previous literature, such as Behera et al. (2024), "who emphasized the critical need for evaluating calcium and PTH levels in cases of non-biliary, non-alcoholic acute pancreatitis. This approach led to the identification of the patient's parathyroid adenoma, reinforcing the importance of biochemical workups in pancreatitis patients with hypercalcemia. The timely identification and management of the underlying cause—primary hyper-parathyroidism—allowed for successful surgical intervention and the patient's full recovery, which is consistent with findings from similar case reports" [11].

Elmaaz et al. (2021) "further support the association between hyperparathyroidism and acute pancreatitis. In their case study, a 63-year-old male presented with hypercalcemia and pancreatitis, much like our patient. They highlight how the resolution of hypercalcemia post-parathyroidectomy was key to the patient's recovery, mirroring our case's outcome. This reinforces the notion that parathyroidectomy is the definitive treatment for hypercalcemia-induced pancreatitis secondary to a parathyroid adenoma. However, as Elmaaz points out, this condition remains exceedingly rare, with an incidence of hypercalcemia-induced pancreatitis of only about 1.5%" [12].

CONCLUSION

This case emphasizes how crucial it is to rule out primary hyperparathyroidism in individuals who arrive with hypercalcemia that does not seem to be explained, especially if they also have acute pancreatitis or gastrointestinal symptoms. Although pancreatitis in the setting of hypercalcemia is an uncommon complication, physicians should be aware of its existence. Patient outcomes can be enhanced by identifying and treating parathyroid adenomas surgically as soon as possible to avoid complications. This patient was successfully managed, demonstrating the importance of a multidisciplinary strategy that included surgical, gastroenterological, and endocrine competence to diagnose and treat this difficult case.

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