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Acute Pancreatitis Due To Hypercalcemia Revealing A Parathyroid Adenoma: A Case Report

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Abstract:
Introduction: *Gallstones and alcohol are the two main causes of acute pancreatitis (AP). Primary hyperparathyroidism (PHPT) is a rare etiology, responsible for less than 5% of cases, through hypercalcemia. Hypercalcemia induces pancreatic inflammation via premature activation of trypsinogen and precipitation of calcium salts in the pancreatic ducts. Consequently, AP can reveal an underlying PHPT.*
Case Presentation: *We report the case of a 78-year-old female, hypertensive and with a prior cholecystectomy, admitted for epigastric pain of pancreatic type. Laboratory tests showed elevated lipase, repeated significant hypercalcemia, and elevated parathyroid hormone (PTH) levels. Imaging revealed Balthazar grade C pancreatitis with 30–50% pancreatic necrosis, without biliary obstruction. Cervical investigations identified a paratracheal parathyroid adenoma associated with a mandibular brown tumor. Initial management included supportive care and medical treatment. The patient subsequently underwent parathyroidectomy, confirming the parathyroid adenoma.*
Conclusion: *PHPT is a rare but important cause of acute pancreatitis. Systematic measurement of serum calcium in cases of non-biliary, non-alcoholic pancreatitis allows early identification of this etiology. Surgical treatment is curative and prevents recurrence. This case highlights the necessity of considering PHPT in the differential diagnosis of unexplained pancreatitis.*

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I. Introduction

Acute pancreatitis (AP) is a sudden inflammation of the pancreas resulting from premature intraglandular activation of pancreatic enzymes, leading to autodigestion of pancreatic tissue. It is a common condition, with an estimated incidence of 13–45 cases per 100000 inhabitants per year, and a variable severity spectrum from mild self-limiting forms to severe necrotizing pancreatitis associated with significant morbidity and mortality.

In approximately 80% of cases, AP is caused by gallstones or alcohol. Less common causes include trauma, infections, medications, anatomical abnormalities, and metabolic disorders. Among the latter, hypercalcemia accounts for less than 5% of cases.

Hypercalcemia may result from primary hyperparathyroidism (PHPT), usually due to a parathyroid adenoma. The pathophysiological mechanisms involve premature activation of trypsinogen in acinar cells and precipitation of calcium salts in the pancreatic ducts.

We report a case of necrotizing acute pancreatitis revealing PHPT due to a parathyroid adenoma.

II. Case Presentation

Mrs. K.H., a 78-year-old hypertensive patient on amlodipine with a history of cholecystectomy 12 years prior, was admitted for sudden epigastric pancreatic pain associated to bilious vomiting.

On admission: GCS 15/15, normotensive, fever 38 °C, asthenia, tachycardia, without tachypnea. Abdominal, cardiopulmonary, and cervical examinations were normal.

Laboratory tests:
- Lipase 5× normal
- Normal triglycerides
- Preserved renal function.

Imaging: Abdominal CT (on day 3) showed Balthazar grade C pancreatitis with 30–50% pancreatic necrosis, without biliary obstruction.

Initial management: Intravenous hydration, prophylactic anticoagulation, analgesics, and antipyretics.

Etiological workup:
- History: No alcohol, no drug use, no medications or abdominal trauma.
- Abdominal ultrasound: No gallstones, bile ducts free.
- Corrected serum calcium: 141 mg/L → 169 mg/L → 177 mg/L (reference: 84– 102 mg/L).
- Phosphorus: 19 mg/L.
- PTH: 392 pg/mL (~10× normal).

Additional investigations:
- Cervical ultrasound: Parathyroid adenoma with mediastinal extension.
- Cervicothoracic MRI: Well-defined right paratracheal lesion 17×15×23 mm consistent with parathyroid tissue, associated with a right mandibular brown tumor.

Specific management: Single administration of bisphosphonate (4 mg/day). No acute complications occurred.
Systemic evaluation: Echocardiography, bone densitometry, and renal function were normal.
The patient was referred to endocrine surgery for parathyroidectomy.

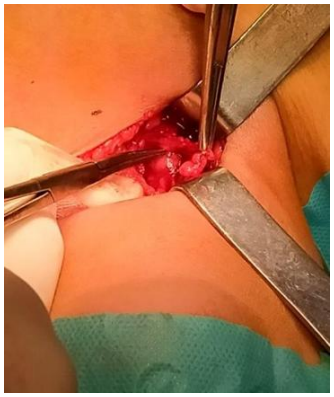

Figure 1: Intraoperative view of the parathyroid adenoma during parathyroidectomy.

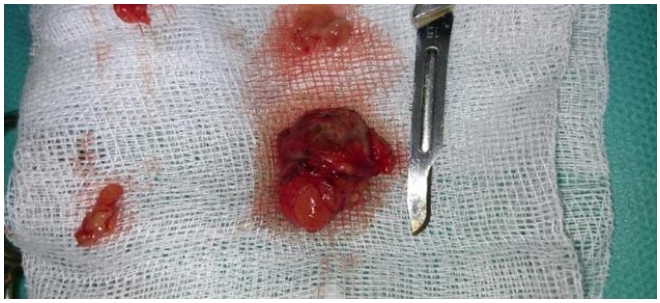

Figure 2: Surgical specimen of the parathyroid adenoma after parathyroidectomy.
Histopathology: Confirmed parathyroid adenoma.

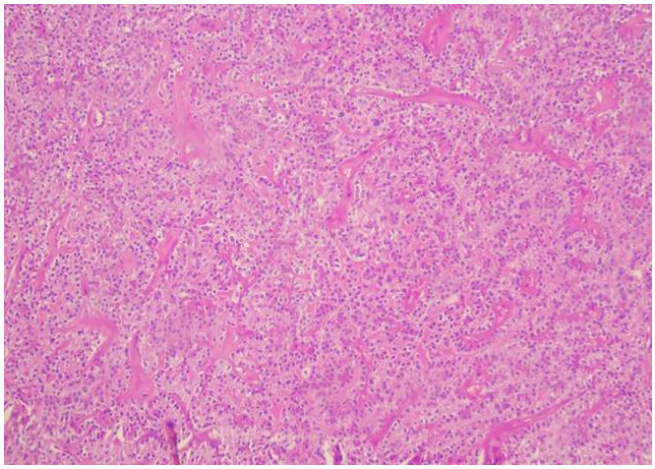

Figure 3: Histological appearance of the parathyroid adenoma (H&E stain, ×40).

III. Discussion

The association between acute pancreatitis (AP) and PHPT is rare but well documented, with prevalence between 3–15%.[1] The pathophysiology is related to the deleterious effects of hypercalcemia: premature activation of trypsinogen in acinar cells and calcium salt precipitation in pancreatic ducts, causing obstruction and inflammation.[2]

Clinical reports support this association. Biondi et al. (2011) reported a case of AP revealing PHPT, resolved after parathyroidectomy.[2] Desmedt et al. (2021) described a patient with hypercalcemia-induced pancreatitis, highlighting that elderly patients without classic risk factors can present similarly.[3]

On a larger scale, Bai et al. (2012) demonstrated in a meta-analysis that the risk of pancreatitis was significantly increased in patients with PHPT, reinforcing the hypothesis of a direct causal link.[1] In an Indian series, Rashmi et al. (2022) found that nearly 10% of patients with PHPT initially presented with acute pancreatitis, and this figure could reach 23% when including chronic forms. These data emphasize the need to investigate hypercalcemia in cases of non-biliary, non-alcoholic acute pancreatitis.[4]

In an African context, Diallo et al. (2016) reported five cases of Senegalese female patients presenting with acute or chronic pancreatitis revealing PHPT. Three patients had favorable outcomes after surgery, but one death occurred in a case of infected necrotizing pancreatitis, illustrating the potential severity of this association and the importance of early diagnosis, particularly in resource-limited settings.[5]

In our case, quick measurement of serum calcium allowed the identification of PHPT as the underlying etiology, thereby avoiding prolonged investigations. Surgical management provided a curative treatment, preventing the risk of recurrent pancreatitis.

IV. Conclusion

PHPT is a rare but important cause of acute pancreatitis, especially in patients without biliary or alcoholic risk factors. Systematic measurement of serum calcium allows early identification and curative management via parathyroidectomy. Early diagnosis prevents severe complications and recurrence, highlighting the need for vigilance even in elderly, endocrinologically asymptomatic patients.

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