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Parathyroid adenoma masquerading as acute pancreatitis

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Abstract
Primary Hyperparathyroidism can be challenging to diagnose, often asymptomatic or only presents with non-specific symptoms. It can affect the skeletal, renal, and neurological systems. Therefore, it is common to diagnose the disease only after it has resulted in complications. One of the key components of the disease is hypercalcemia, which is a known cause of acute pancreatitis. Our case highlights this relatively rare association.
A 24-year-old male presented to the emergency room (ER) with severe epigastric pain and vomiting. Pancreatic enzymes were elevated, confirming acute pancreatitis. After the common causes of acute pancreatitis had been ruled out, the patient was found to have hypercalcemia. Primary Hyperparathyroidism due to a parathyroid adenoma was found to be the underlying pathology. He underwent a minimally invasive parathyroidectomy, following which he made an uneventful recovery.
Acute pancreatitis is associated with significant morbidity and mortality. It also has a high recurrence rate unless the underlying etiology is identified and eliminated. Calcium levels should always be measured in cases of acute pancreatitis. Primary Hyperparathyroidism is the most common cause of hypercalcemia, and parathyroid adenoma is the most common cause of primary Hyperparathyroidism. Parathyroid adenoma can be effectively managed with surgical excision. This will not only prevent recurrence of acute pancreatitis but will also prevent further complications of primary Hyperparathyroidism.

Keywords: Primary hyperparathyroidism, hypercalcemia, acute pancreatitis, minimally invasive surgery

Introduction
Primary Hyperparathyroidism is most commonly caused by parathyroid adenoma. It is often asymptomatic or presents with a variety of non-specific symptoms. It is usually only diagnosed after complications have occurred, which is why it is often so difficult to make a diagnosis. The disease affects mainly the renal, skeletal, and neurological systems [1].
One of its key features is hypercalcemia, which is in addition to raised or inappropriately normal parathyroid hormone levels. Hypercalcemia is one of the known causes of acute pancreatitis. It can be easily overlooked because acute pancreatitis has many other, more common causes [2]. Acute pancreatitis is a disease with considerable morbidity and mortality, as well as a high risk of recurrence, unless the underlying cause is identified and eliminated [3].
Here, we present a case of a young gentleman with acute pancreatitis. Management for acute pancreatitis was initiated. However, a definite etiology could only be confirmed later. Hypercalcemia secondary to primary Hyperparathyroidism was finally determined to be the underlying pathology. Our case highlights this relatively rare complication of the disease and the need to look for it in cases of acute pancreatitis.

Case Presentation
A 24-year-old male presented to the emergency room (ER) with severe epigastric pain for three days. The pain radiated to the back and was exacerbated by movement of any kind. It was associated with vomiting, which was non-bloody and non-bilious and contained food particles. On examination, he was tachycardiac, with a heart rate of 120 beats per minute. His blood pressure was 110/70 mmHg, his respiratory rate was 26 per minute, and he maintained oxygen saturation of 98% at room air. His epigastrium was markedly tender.

Investigations were done, which revealed a raised serum amylase level of 1,140 units/liter. He was admitted to the surgical unit, and management was started for acute pancreatitis.

Initial attempts to determine the etiology were unsuccessful. The patient did not have any history of alcohol intake, and an ultrasound scan ruled out gallstone disease. He did not have any other comorbidities and had not been taking any medications. In the meantime, the patient began to deteriorate. A computed tomography (CT) scan was done, which showed acute necrotizing pancreatitis involving 30-40% of the pancreatic parenchyma (Figure 1), bilateral pleural effusions with basal atelectasis, and a right-sided renal calculus.

Conservative management was continued, and further workup was done, which revealed serum calcium levels to be high at 13.32 mg/dL. The cause of the hypercalcemia was identified as raised parathyroid hormone levels of 232 pg/mL. Serum phosphate levels were 2.45 mg/dL, and vitamin D levels were severely deficient at 8 ng/mL. Based on these biochemical findings, a diagnosis of primary Hyperparathyroidism was made. Ultrasound of the neck showed a single enlarged parathyroid gland to the right of the thyroid gland, measuring 15 mm × 9 mm. A contrast-enhanced computed tomography (CT) scan of the neck and chest also confirmed a right-sided parathyroid adenoma (Figure 2,3) and also ruled out any other pathology in the region.

Sestamibi scan also showed a right inferior parathyroid adenoma (Figures 4A-4D). The patient began to respond to conservative management, and once he had recovered from pancreatitis, he was prepared for surgery. A minimally invasive parathyroidectomy was done, and postoperatively, he recovered well. His postoperative parathyroid hormone levels came down to 51 pg/mL, and calcium levels similarly improved to 10.1 mg/dL. The histopathology report confirmed the lesion to be a parathyroid adenoma. The patient was doing well at the latest follow-up and had no disease recurrence.

Discussion

Acute pancreatitis is a disease with significant morbidity and mortality and is the leading cause of gastrointestinal-related hospitalization. It affects both men and women alike, predominantly those in the middle-aged or older age groups. It has an annual incidence of 34 cases per 100,000 general population per year globally, although this varies in different parts of the world [4].

While most cases follow a relatively mild course, a significant proportion develop a more severe form of the disease. While the mortality of mild edematous pancreatitis is only about 3%, this can go up to 28% in the severe form of the disease [2]. Necrotizing pancreatitis is associated with a 13% mortality if it involves sterile necrosis. However, in the case of infected necrosis, not only is there a higher risk of organ failure and need for admission to the intensive care unit, but the mortality is also higher at 28% [5].

The most common causes of acute pancreatitis worldwide include gallstones and alcohol intake. There are also several other, less common, causes. These can be classified into mechanical, toxic, metabolic, and miscellaneous. Mechanical causes include, first and foremost, gallstones, as well as pancreatic duct obstruction, dysfunction of the sphincter of Oddi, ampullary obstruction, congenital malformations, and trauma. The toxins and metabolic causes include alcohol as the most common factor, as well as drugs, scorpion venom, organophosphate poisoning, hyperlipidemia, and hypercalcemia. Other causes include ischemia, iatrogenic injury, infection, autoimmune pathology, hereditary factors, and cystic fibrosis. The frequency of each etiology varies depending on the region and socioeconomic factors [2]. After ruling out the more common etiologies, we identified hypercalcemia as the cause of pancreatitis.

Hypercalcemia is a known cause of acute pancreatitis. The exact pathology is still unclear. However, persistently raised calcium levels in the extracellular compartment can result in persistently raised calcium levels within the cell. As a result of this increased cytosolic calcium, vacuoles form, and trypsinogen is activated, leading to pancreatitis. The persistently elevated cytosolic calcium levels are also seen in pancreatitis due to other causes, such as gallstones and alcohol. They are implicated in the destruction of cellular defense mechanisms, aiding pancreatic inflammation [6]. The role of calcium in the subsequent disease process is different, with hypocalcemia indicating a worse prognosis. Calcium levels are included in various pancreatitis severity scoring systems [3].

Calcium levels in the extracellular compartment are tightly regulated. This homeostasis depends on the complex interplay among three chemical mediators: Vitamin D, calcitonin, and parathyroid hormone. Any interference can result in the serum calcium concentration rising above 10.5 mg/dL, resulting in hypercalcemia. While most such patients will remain asymptomatic, a small fraction with even higher serum calcium levels will experience symptoms relating to the neurological, muscular, renal, gastrointestinal, or cardiovascular systems. Primary Hyperparathyroidism is the most common cause of hypercalcemia, along with malignancy. Other causes include genetics.

Disorders such as familial hypocalciuric hypercalcemia, endocrine disorders such as adrenal insufficiency, granulomatous diseases such as sarcoidosis, and the effects of drugs such as thiazide diuretics and antacids. Hypercalcemia is managed mainly with hydration. There is a role of bisphosphonates in chronic cases, and hemodialysis is only required in severe cases [7].

Primary Hyperparathyroidism (PHPT) occurs when one or more parathyroid glands begin to secrete increased amounts of parathyroid hormone. Most cases involve a benign pathology, mainly adenoma of a single gland (80%) or multi-gland hyperplasia (20%). Malignancy is exceedingly rare, accounting for less than 1% of cases. The parathyroid hormone levels and calcium levels are both usually elevated. In some cases, only the calcium levels are raised, along with inappropriately normal parathyroid hormone levels. While the disease is usually sporadic, there are some genetic forms of PHPT and some germline mutations [8].

PHPT is usually asymptomatic, especially in developed countries (85%), with only the remainder presenting with renal or skeletal complications. In contrast, in developing countries, where routine biochemical testing is not done, patients present more often with the complications of the disease. These include kidney stones, nephrocalcinosis, bone pain, pathological fractures (especially vertebral fractures), and osteitis fibrosa cystica. There is also an association with depression, neurocognitive changes, and an

increased risk of cardiovascular diseases [1]. Once we had found the calcium levels to be elevated in our case, we also checked serum phosphate, vitamin D, and PTH levels, all of which supported the diagnosis of PHPT.

The earliest association between hypercalcemia and pancreatitis was made in 1903 [6]. Since then, hypercalcemia secondary to PHPT has been associated with acute pancreatitis. However, it is relatively uncommon for PHPT to present as the initial manifestation of the disease [9,10]. The majority of the literature suggests that it is the symptomatic, diagnosed cases of PHPT who are at an increased risk of developing acute pancreatitis, often with concomitant causes. Up to 65% of cases of PHPT-associated pancreatitis had at least one other associated etiology, such as gallstones or alcohol intake. While this has led some to conclude that PHPT is merely a coincidental association, it could also mean multiple factors may cause pancreatitis. This multi-hit theory is also supported by the fact that specific gene mutations associated with pancreatitis were found to be more common in PHPT patients who develop pancreatitis than in PHPT patients who did not develop pancreatitis [11].

The management of acute pancreatitis is generally supportive, based on intravenous hydration, analgesics, and nutrition (enteral or parenteral). In severe cases, patients may need intensive care with organ support, antibiotics for superimposed infection, and pancreatic replacement therapy. Referral to tertiary care centers is required for complicated cases. Once the initial episode of pancreatitis has resolved, one-fifth of patients will develop recurrence of the disease, and a third will develop chronic pancreatitis. Therefore, risk reduction and preventing recurrence are of the utmost importance [12]. This is why it is necessary to determine the etiology of the disease so that it can be eliminated if possible or controlled as an alternative. The initial and subsequent management includes treating the underlying cause. In the case of biliary pancreatitis, cholecystectomy must be performed. In the case of alcohol-induced pancreatitis, alcohol intake must be stopped [3]. In most cases of pancreatitis due to Hyperparathyroidism, parathyroidectomy results in the cessation of recurrent episodes of pancreatitis [6].

The diagnosis of PHPT is a biochemical one. Imaging is done not to confirm the diagnosis but to guide surgical management with various modalities available. 99mTc-sestamibi scintigraphy detects the increased uptake in parathyroid adenoma due to the increased quantity of mitochondria in the tissue, with an accuracy of 97.2% for PHPT. 99mTc-sestamibi scintigraphy was used in our case to identify the diseased gland. Variations of this technique include washout scintigraphy and subtraction scintigraphy, the latter being better for suspected single or multiple-gland disease cases. Single-photon emission computed tomography (SPECT) helps identify the location more precisely. CT scan with contrast may also be used, especially if the parathyroid gland is suspected to be in the mediastinum. Ultrasound is usually used in combination with other imaging studies. MRI is uncommon except in specific situations such as pregnancy [1].

PHPT is best managed by surgery. While there is some role in medical management, surgery (parathyroidectomy) is the only definite management, especially in symptomatic cases, with non-surgical therapies usually reserved for cases where surgery is impossible [1]. Medical management can be considered in mild, asymptomatic cases, especially where surgery is either contraindicated or has failed. This includes dietary modifications and medications, mainly calcimimetics such as cinacalcet for hypercalcemia and antiresorptive therapy for skeletal protection. However, these patients must be carefully monitored with regular biochemical and bone mineral density evaluation [13].

The traditional surgery for PHPT, the standard parathyroidectomy, is slowly but surely being replaced by minimally invasive procedures. While there is no consensus on the exact definition, most experts agree that it involves a small wound with minimal dissection. Endoscopic and video-assisted parathyroidectomy, as well as robotic-assisted parathyroidectomy, represent the recent advances in this field. Good preoperative imaging must be emphasized more regarding minimally invasive approaches. Another option is radio-guided parathyroidectomy, in which a preoperative dose of 99mTc-sestamibi is given, and then a gamma probe is used for intraoperative localization. To increase the accuracy of the surgical procedure, adjuncts such as intraoperative PTH levels and frozen sections, especially in the case of suspected malignancy, may be utilized [14].

Conclusion

Primary Hyperparathyroidism has an insidious course. It is usually only diagnosed once the disease has caused significant complications. The skeletal and renal systems are commonly involved in this pathology, especially in places where routine biochemical testing is uncommon.

One relatively rare complication is acute pancreatitis. Acute pancreatitis is associated with significant morbidity and mortality. It also has a high recurrence rate unless the underlying etiology is identified and eliminated. Therefore, the etiology should always be sought in patients with acute pancreatitis, and serum calcium levels should be measured. If the underlying cause is determined to be Hyperparathyroidism, that should be addressed to prevent further complications and the recurrence of acute pancreatitis.

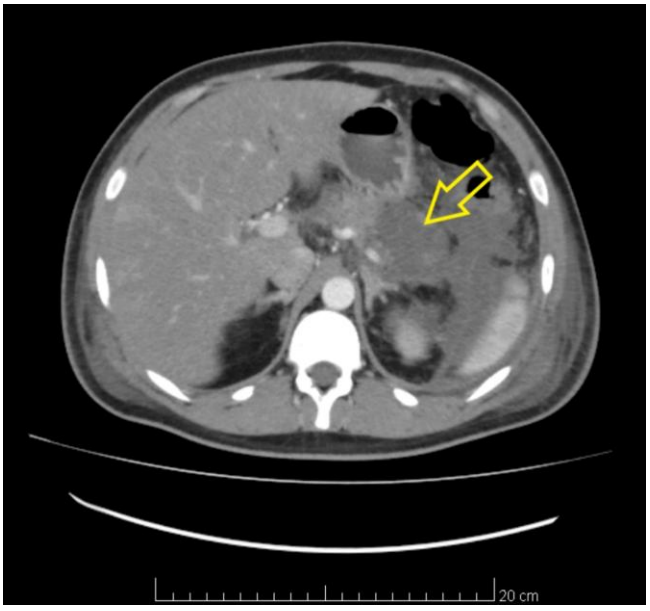


Fig 1: Computed Tomography Scan Abdomen (axial view) showing acute necrotizing pancreatitis (yellow arrow)

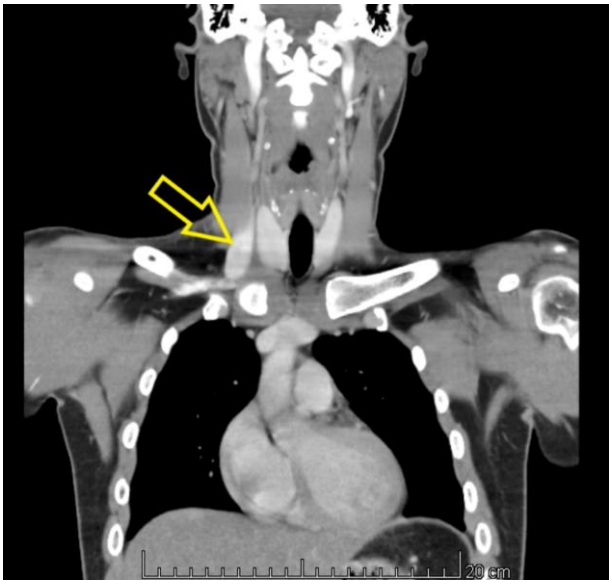


Fig 2: Computed Tomography Scan Neck and Chest (coronal view) showing the parathyroid adenoma (yellow arrow)

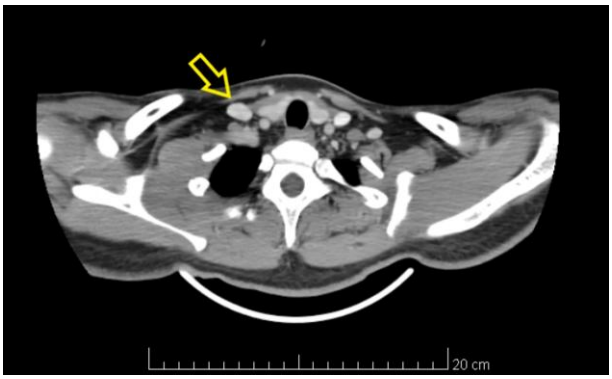
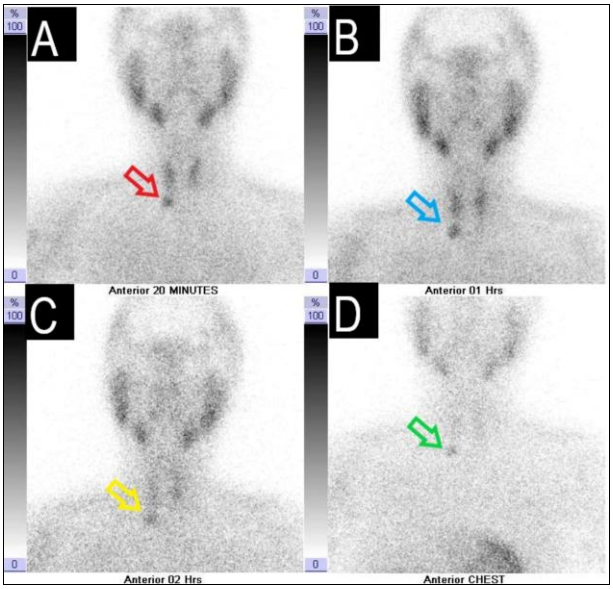


Fig 3: Computed Tomography Scan Neck and Chest (axial view) showing the parathyroid adenoma (yellow arrow)



(A) Early image at 20 minutes, with parathyroid adenoma (red arrow), as well as uptake in thyroid and salivary glands (B) Image at 1 hour, with parathyroid adenoma (blue arrow), (C) Late image at 2 hours, with persistent uptake in the parathyroid adenoma (yellow arrow), and washout from the other glands (D) Parathyroid adenoma (green arrow) in the neck, and no such lesion in the chest

Fig 4: Sestamibi Scan

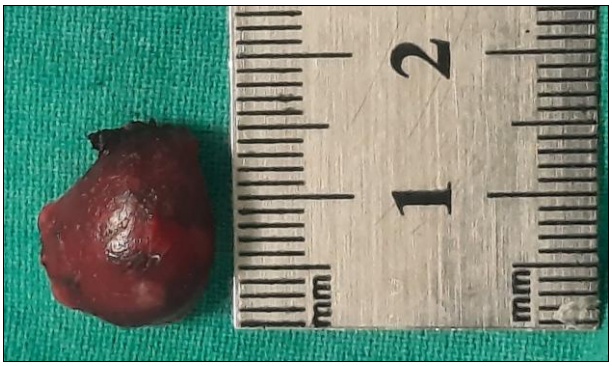


Fig 5: Parathyroid adenoma specimen (with measuring ruler for scale)

Conflict of Interest
Not available.

Financial Support
Not available.

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