Case Report

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Hyperparathyroidism Masquerading as Acute Pancreatitis

Ashishjot Kaur[1], Avneet Singh Setia[2], Prabh Simranpal[3]

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Abstract

Background: Acute pancreatitis is a common condition, frequently attributed to gallstones or alcohol consumption. Interestingly, while hypocalcemia is typically associated with acute pancreatitis, hypercalcemia-induced pancreatitis is a rare clinical picture. Even more uncommon is the role of hyperparathyroidism as the underlying cause.This case challenges the conventional understanding of pancreatitis and its etiology, highlighting an atypical presentation that rose from hyperparathyroidism, through sustained hypercalcemia, emerged as the unexpected culprit for acute pancreatitis, shedding light on a rare interplay between endocrine dysfunction and pancreatic inflammation.

Case Description: We present a case of middle age patient, who developed acute pancreatitis with no history of alcohol use or gallstone disease. Lab Investigations revealed elevated serum calcium levels prompting further evaluation. Imaging studies confirmed the presence of parathyroid adenoma, implicating primary hyperparathyroidism as underlying cause of hypercalcaemia and subsequent pancreatitis. The patient underwent surgical excision of aroma with normal normalisation of calcium levels and resolution of symptoms thereafter.

Clinical relevance: This case highlights a rare, but critical association between hyperthyroidism and acute pancreatitis, emphasising the importance of investigating parathyroid pathology in patients presenting with hypercalcaemia and pancreatitis. Early diagnosis and targeted management can prevent recurrent episodes and associated complications.

Keywords: Hypercalcaemia, Pancreatitis, Hyperparathyroidism, Parathyroid adenoma, Endocrine dysfunction.

Author Affiliation: [1]Junior Resident, Department of Medicine, [2]Assistant Professor, [3]Junior Resident, Department of Surgery, Gian Sagar Medical College and Hospital, Rajpura, Jansla 140506, Punjab, India.

Corresponding Author: Avneet Singh Setia, Department of Surgery, Gian Sagar Medical College and Hospital, Rajpura, Jansla-140506, Punjab, India.

Email: jotghuman28@gmail.com

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INTRODUCTION

A serious global public health concern, pancreatitis, has become more common over the past 30 years, with South Asia experiencing the largest increase.[1]

The pancreas becomes inflamed and injured in acute pancreatitis, which can result in sepsis and multiple organ failure.It is among the most common reasons for hospitalizations across the globe. With a mortality rate of up to 20% overall, it can be a potentially deadly condition.[2]

Knowing the common and uncommon causes and unique appearance of acute pancreatitis is crucial given the risks and complications resulting from a delayed diagnosis.[3]

About two thirds of all cases of acute pancreatitis are caused by alcohol and gallstones, which together make up the main etiological component.[3,4]

Furthermore, calcium accumulation in the pancreatic duct and calcium-induced activation of trypsinogen are two rare pathologies for causing pancreatitis that are brought on by hypercalcemia.[2,3] This is well known to result from activation of pancreatic proteins and starting an inflammatory cascade which paves way to pancreatitis.**[3]

Hyperparathyroidism may also manifest as pancreatitis, particularly if serum calcium or phosphate levels are elevated.

About 80% to 85% of all cases of primary hyperparathyroidism are caused by a single parathyroid adenoma, which is a prevalent cause of hypercalcemia.[2] The majority of patients with primary hyperparathyroidism (PHPT) do not exhibit any symptoms. On the other hand, individuals who are symptomatic typically display signs of hypercalcemia, including painful bones, kidney stones, and mental and abdominal moans.[4] Additionally, a rare clinical Case report

A 58 year old female presented to the hospital with complaints of sudden onset dull pain in epigastric region radiating to back on and off for the past 3-4 days which increased in intensity and frequency on the day of presentation for the previous 7 to 8 hours. The pain was associated with nausea and 3 - 4 episodes of non-bilious vomiting.

Patient also compliant of bony pains and muscle weakness. No complaints of fever, constipation or loose stool, burning micturation, bony fractures, neuropsychiatric symptoms were found.

Her past surgical history revealed she has been operated for laparoscopic cholecystectomy in view of multiple gallstones 2 years back. Medical history was remarkable for hypertension (4 to 5 years, on regular tablets telmisartan 40 mg and amlodipine 5 mg) and recurrent renal calculi(treated with medications). She did not smoke or drink alcohol.

On admission, vitals were as follows

BP 160/100 mmHg

Pulse 100/min

RR 22/minute

and was Afebrile

Considering provisional diagnosis of acute pancreatitis, she was kept NPO and managed in ICU with IV fluids, antibiotics, antiemetics, antihypertensive and was closely monitored.

Investigations performed, were as follows:

TLC	8700
Hb	9
PLT	1.8
renal function tests	
urea	30
Creatinine	1
Uric acid	5.2
Calcium	22
Lipase	1250
Electrolytes	
Sodium	137
Potassium	4.3
Chloride	105
25-OH VIT D	46.3
Magnesium	3.5
Phosphorus	2
SGOT	15
SGPT	18
Bilirubin	1.3
ALP	172

Blood tests were unremarkable except for increase S. lipase and ECG changes showed short QT interval and increase in amplitude of QRS wave.

Additional surprise finding was for markedly elevated serum calcium (22 mg/dl). As a result, the patient was administered IV fluids at the rate of 120 ml per hour and IV bisphosphonates. Further investigations of serum Magnesium, phosphate, vitamin D levels, iPTH,repeat ECG and imaging of CECT abdomen with pelvis and screening of thorax and neck were ordered.

PTH levels were significantly raised (856pg/ml)

CECT chest revealed bilateral plural effusion, segmental collapse consolidation of left basal segment with bilateral fibrotic, trance and septal thickening.

CT scan of abdomen and pelvis signified bulky pancreas with extensive peri pancreatic fat stranding and left thickened perinephric fat pad suggestive of acute interstitial oedematous pancreatitis with ascites.

CECT scan of neck showed a well circumscribed hypodense nodule posterior to the right lobe of thyroid which was diagnosed **as right parathyroid adenoma.**

After the episode of pancreatitis subsided, investigations were repeated.

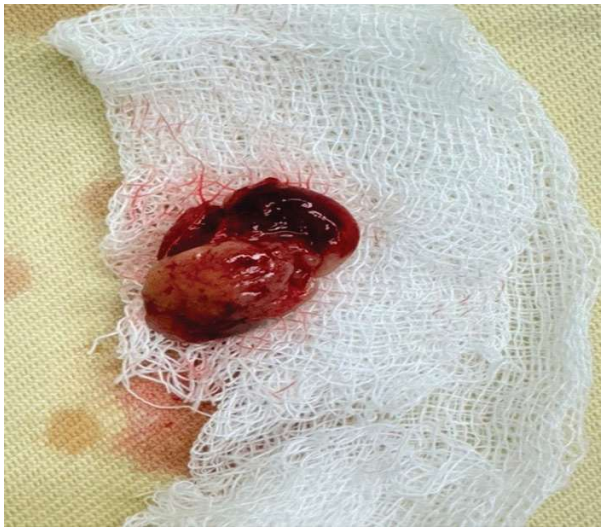
Patient was prepared for surgery, right parathyroidectomy.

Operative findings evidenced an enlarged right inferior parathyroid adenoma measuring approximately 4x3 cm.

Surgery was uneventful and post operative serum calcium (8.5) and serum iPTH(2.6pg/ml) repeated after 1 week were found to be within normal limits. Follow up planned at 3 months and 6 months confirmed no recurrence of abdominal pain in our case.

DISCUSSIONS

The severity of the disease in acute pancreatitis may correlate with a reduction in serum calcium, which is a typical picture in pancreatitis. The presence of pancreatitis and hypercalcemia should raise suspicions of hyperparathyroidism or cancer, for which iPTH and neck ultrasonography are recommended.[5,6]



As early as 1940, Smith and Cooke reported the first case of AP linked to hyperparathyroidism, and Pyrah et al tried to compile the conditions under which pancreatic illness took place in conjunction with PHPT.[7,8] In another study, Only 17 patients (1.5%) had concomitant pancreatitis out of the 1153 patients with hyperparathyroidism operated on at the Mayo Clinic between 1950 and 1975.[9] 0.4% cases of acute pancreatitis were shown to be caused by hyperparathyroidism in a research by Prinz et al in 1985.[9] Similarly, in 1998, Carnaille B et al. retrospectively assessed 1224 patients of hyperparathyroidism and discovered that 3.2% of them had acute pancreatitis.[10]

there has always been an ongoing debate in literature for direct causal relationship between hyperparathyroidism and parathyroid induced pancreatitis. In 1998, Sitges Sara rejected the Mayo Clinic's studies as untrue and proposed that hypercalcemia may cause pancreatitis with nonparathyroid reasons instead.[9,12]

While 64.7% of patients had at least one concurrent cause of pancreatitis, such as gallstones or alcohol abuse, 1.5% of patients with surgically confirmed PHPT had coexisting or prior pancreatitis, and symptoms of pancreatitis did not improve following treatment for hyperparathyroidism, according to Bess *et al*.[12]

Five out of 61 cases of pancreatitis had PHPT, according to Diallo *et al*.[13] Of these, two patients developed CP, and the prevalence of PHPT-associated CP was almost 3.27% of all hospitalized cases of pancreatitis. Additionally, three follow-up investigations from India revealed a low frequency

of CP linked to PHPT.

Jacob et al divided the pancreatic disease presentation in PHPT into four categories in 2006: PHPT presenting as AP, PHPT presenting as recurrent AP without CP, PHPT presenting as CP with or without pancreatic calcification, and PHPT complicated by AP in the postoperative phase.[11]

Therefore, different presentations of pancreatitis may be manifested as a result of parathyroid adenoma.

Patient's previous medical history, clinical presentation, lab investigations, and imaging results are all crucial to diagnose PHPT induced AP. Individuals might also exhibit recurring episodes of AP. Increased iPTH and calcium levels and decreased phosphate levels demand the need of further imaging to rule out parathyroid etiology.

Cervical ultrasonography is an inexpensive, safe, radiation-free, and extremely sensitive method, frequently utilized as the first localization examination in patients with PHPT.[14] Negative imaging results should raise a suspicion for ectopic parathyroid adenomas and further imaging be advised as per clinical judgement.

Tc-99m MIBI with SPECT of the neck can offer further functional information and is more sensitive for ectopic adenomas that are either undiscovered or overlooked by ultrasonography.[14]

Our patient refused further imaging due to affordability issues.

Primary treatment for parathyroid adenoma is removal of gland with simple parathyroidectomy as a favoured surgical treatment over medications or long-term monitoring.[15]

5282 patients from 14 studies were included in a systemic evaluation, which indicated that the long term (≥1 year) cure rate defined as normocalcemia remaining for more than 6 months following MIP was 96.9%. The most frequent consequence, temporary postoperative hypocalcemia, was seen in 4.4% of cases overall.[15]

Our patient was asymptomatic with normocalcemia, followed up at two occasions of 3 and 6 months post-operatively.Since there were no additional risk factors for pancreatitis, our case demonstrates a true positive causal connection between PHPT and pancreatitis.

CONCLUSION

A rare presentation of primary parathyroid adenoma is acute pancreatitis (AP).

It is highly recommended that imaging be done to check for any parathyroid proliferative disorders in patients with AP who have elevated serum calcium levels after excluding commonly recognized etiologies of pancreatitis. In addition to prevent AP problems, early identification and treatment can help stop AP from recurring.

For parathyroid adenoma, a parathyroidectomy is a safe, efficient, and permanent treatment.

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Conflict of interest: The authors declared no conflict of interest in relation to this case report.

Ethical consideration

Ethical approval was not required for this case report. However, informed consent was obtained from the patient and all measures were taken to protect their privacy and confidentiality.

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