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Case Report

Internal Medicine Section

Hyperparathyroidism: A Hidden Trigger for Recurrent Pancreatitis

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ABSTRACT

It is uncommon to find an association between pancreatitis and Primary Hyperparathyroidism (PHPT), but not unknown. Hypercalcaemia causes acute pancreatitis by initiation of an inflammatory cascade and formation of pancreatic calculi leading to ductal obstruction, causing acute or chronic pancreatitis. We report a unique case of a 51-year-old male who presented to us with recurrent episodes of acute pancreatitis. Biochemical investigations were suggestive of hypercalcaemia. Further evaluation of hypercalcaemia, not only confirmed PHPT by biochemical investigations but also localised a left inferior parathyroid adenoma by nuclear imaging. There were no additional risk factors for acute pancreatitis. The patient was initially managed medically, once the patient was normocalcaemic, he underwent left inferior parathyroidectomy, which led to resolution of recurrent acute pancreatitis. The patient was followed-up for one year and was found to be biochemically normocalcaemic and there was no recurrence of symptoms.

Keywords: Hypercalcaemia, Parathyroidectomy, Parathyroid adenoma, Parathyroid surgery

CASE REPORT

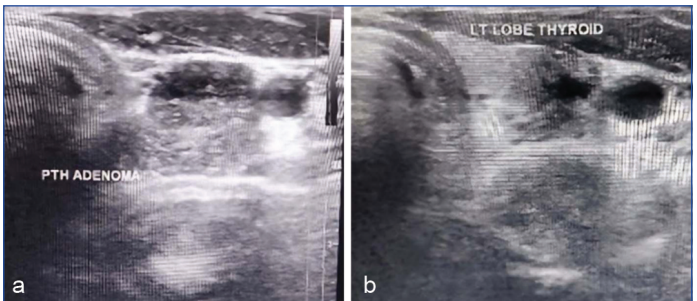
A 51-year-old male, presented to the General Medicine Out-Patient Department (OPD) with acute pancreatitis. The patient had been admitted with multiple episodes of acute pancreatitis in the last 1.5 years and this was his fourth episode. He presented this time with complaints of abdominal pain and nausea for four days. The pain abdomen was located in the epigastric region, severe in intensity, radiating to the back, alleviated by bending forward and was progressive in nature. The patient had no history of alcohol intake, gallstone disease, illicit drug intake, blunt trauma to the abdomen, any history of addictions or abdominal surgery in the past. On examination, the blood pressure was 128/84 mmHg, measured in the right upper limb, pulse rate was 110/minute, respiratory rate of 19/minute and the oxygen saturation was 98% at room air. On general examination, there was no pallor, icterus, cyanosis, clubbing or lymphadenopathy. On per abdomen examination, there was epigastric tenderness, while other systemic examination was normal.

The patient was provisionally diagnosed as a case of acute pancreatitis and underwent biochemical [Table/Fig-1] and radiological investigations for diagnostic confirmation.

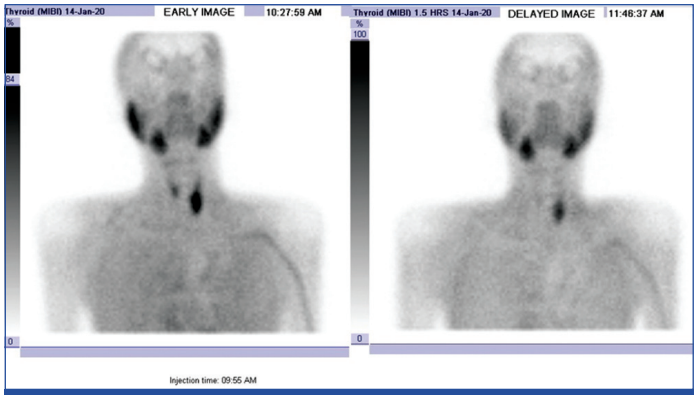
Investigations	Value	Normal range
Haemoglobin (g/dL)	13.2	13-16
Total leukocyte count (/cumm)	14,200	4000-11,000
Platelet count (Lac/cumm)	2.6	1.5-3
Total bilirubin (mg/dL)	0.6	0.3-1.2
Direct bilirubin (mg/dL)	0.2	0.1-0.4
AST (U/L)	25	8-48
ALT (U/L)	21	7-55
ALP (U/L)	76	40-129
Blood urea (mg/dL)	15	14-45
Serum creatinine (mg/dL)	0.6	0.3-1.2
Random blood glucose (mg/dL)	119	70-140
Serum cholesterol (mg/dL)	123	<200
Serum triglycerides (mg/dL)	145	<150
HDL (mg/dL)	43	>50
Serum amylase (U/L)	1400	30-110
Serum lipase (U/L)	1250	0-160
Serum calcium (mg/dL)	13.2	9.1-10.8
Serum phosphorus (mg/dL)	1.9	2.5-4.5
Serum 25 hydroxy vitamin D (ng/dL)	13	30-optimum
ipTH (pg/dL)	310	10-65
Serum albumin (g/dL)	4.1	4.0
Serum lactate (mmol/L)	1.1	

[Table/Fig-1]: Biochemical investigations of the patient at admission.
AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; HDL: High density lipoprotein; ipTH: Intact parathyroid hormone

On the basis of the biochemical investigations, the diagnosis of acute pancreatitis was confirmed. The radiological investigations were carried out to find out the aetiology of PHPT. The Electrocardiogram (ECG) showed short QT interval and short ST segment with flattening of T waves, suggestive of changes secondary to hypercalcaemia. Ultrasound whole abdomen was suggestive of acute pancreatitis along with bilateral medullary nephrocalcinosis. Ultrasound neck revealed a hypoechoic lesion of 3.1×1.5×1.4 cm at the lower pole of the left lobe of the thyroid [Table/Fig-2]. To confirm our diagnosis and to identify the aetiology, Technetium 99m sestamibi scintigraphy was done, which revealed a parathyroid adenoma located posteromedially to the inferior pole of the left lobe of thyroid [Table/Fig-3]. Hence, a confirmatory diagnosis of acute pancreatitis due to PHPT was made. The patient was kept nil per mouth, hypercalcaemia was aggressively managed with continuous intravenous (i.v.) normal saline 0.9%, one



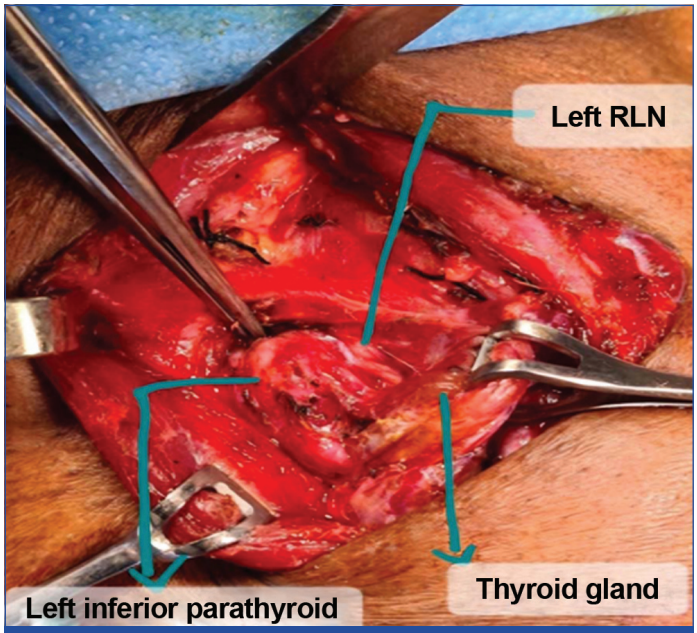
[Table/Fig-2]: Ultrasound neck showing a well defined, predominantly hypoechoic solid lesion measuring 3.1×1.5×1.4 cm at the inferior pole of the left lobe of the thyroid gland at the parathyroid location.



[Table/Fig-3]: Technetium 99 m scan showing focal tracer uptake in a soft-tissue nodule measuring 1.6×1.3×3 cm located posteriomedially to the inferior pole of the left lobe of thyroid.

dose of injection zoledronic acid 5 mg in the form of an i.v. infusion along with calcitonin nasal spray, one spray (200 IU) in alternate nostril was given twice a day. For acute pancreatitis, the patient was managed conservatively with i.v. tramadol 100 mg twice a day along with i.v. fluids. The serum calcium was monitored every 12 hourly, after three days the patient achieved normocalcaemia (serum calcium 10.4 mg/dL), was then transferred to the general surgery department for left inferior parathyroidectomy.

Patient underwent left inferior parathyroidectomy under general anaesthesia. Intraoperatively, an enlarged inferior parathyroid gland of 1.8×1.5×3.4 cm was noted and excised, while the recurrent laryngeal nerve was preserved during the procedure [Table/Fig-4].



[Table/Fig-4]: Intraoperative image of left inferior parathyroidectomy, the parathyroid adenoma measures 1.8×1.5×3.4 cm.
RLN: Recurrent laryngeal nerve

Postoperative biopsy showed a well circumscribed encapsulated tumour arranged in nests and insular pattern divided by thin fibro vascular septae. Individual cells had clear cytoplasm, round centrally placed nuclei with fine stippled chromatin. Focal areas showed scattered inflammatory cells. Adjacent normal compressed parathyroid tissue identified, features were suggestive of a parathyroid adenoma. The postoperative period was uneventful.Two months postsurgery the patient was followed-up with repeat biochemical parameters, serum calcium was 9.8 mg/dL, serum phosphorus 3.3 mg/dL, serum ipTH 26.1 pg/mL and serum 25- hydroxy vitamin D was 24.5 pg/mL. The patient had no further episodes of pancreatitis. The patient was followed-up on several occasions postsurgery, all biochemical markers were repeated on three monthly intervals for one year postsurgery, and were found to be within normal limits.

DISCUSSION

The association between pancreatitis and PHPT is rare and controversial. The incidence of PHPT is 1% in the adult population, which increases to almost 2% after the age of 55 years [1]. The prevalence of acute pancreatitis occurring secondary to primary hyperparathyroididm is nearly 1.5 to 15% [2]. Conventionally, 80% of acute pancreatitis is related to alcohol abuse and biliary stone disease and <10% have a metabolic cause as the aetiology [3]. Two mechanisms have been suggested, hypercalcaemia can lead to de novo activation of trypsinogen to trypsin causing auto-digestion of pancreas, and can also lead to the formation of pancreatic calculi, ductal obstruction and subsequent attacks of acute on chronic pancreatitis [4,5].

In our case, the patient had presented to our OPD with the fourth episode of acute pancreatitis. In all the previous admissions, the patient had been evaluated for the common causes of acute pancreatitis like alcohol intake, gall bladder stone disease, and others, but the metabolic causes were not evaluated. In the fourth admission, the metabolic profile was done and hypercalcaemia was seen. In view of hypercalcaemia, the further workup was done and parathyroid adenoma was found to be the aetiology. Post parathyroidectomy, no recurrence of symptoms was seen. Misgar RA et al., reported a similar case of a 32-year-old male who had recurrent episodes of pancreatitis secondary to PHPT, there were no additional risk factors of acute pancreatitis. Resolution of symptoms and hypercalcaemia was seen post parathyroidectomy [6]. A retrospective, descriptive study was done by Diallo I et al., in which 61 patients with acute and chronic pancreatitis were enrolled, among them five patients had a pancreatitis revealing PHPT (8%) [7].

In a case series of 40 patients by Carnaille B et al., showed that not incidental but rather a causal association exists between pancreatitis and PHPT, mainly owing to hypercalcaemia [8]. They also showed that the cure of acute pancreatitis occurs after the PHPT has been treated, but it does not prevent the development of subacute or chronic pancreatitis. Also, Agarwal A et al., showed a resolution of pancreatitis after parathyroidectomy for PHPT in 80% cases while Bhadada SK et al., showed 100% resolution of pancreatitis in Indian patients [9,10].

Though PHPT is less commonly associated with pancreatitis, serum calcium levels should be assessed as a part of the usual evaluation of acute or chronic pancreatitis [8]. The usual distinguishing biochemical features of PHPT are high serum calcium levels along with low serum phosphate level in the setting of inappropriately high or normal Parathyroid Hormone (PTH) level. There are few other clinical clues, which might help diagnose long- standing PHPT, like calcium oxalate or calcium phosphate renal stones, renal dysfunction, bony defects such as pathological fractures or osteoporosis. These signs and symptoms may help diagnose PHPT earlier than usual [11]. Several genetic syndromes have been associated with hypercalcaemia like multiple endocrine neoplasia type 1 or type 2A [12,13], familial hyperparathyroidism and others. The patient should be clinically evaluated for these syndromic features, if present and genetic testing should be considered [14].

CONCLUSION(S)

The PHPT induced acute pancreatitis, though rare, has been described in the literature. PHPT can be associated with both acute and chronic pancreatitis. It is essential to look for hypercalcaemia while evaluating a case of pancreatitis, if present, then PHPT should be worked up for. Pancreatitis almost always resolves after the cure of PHPT. Genetic and environmental factors may also be contributory to the cause, hence if there are any clinical features pointing towards the same, then a genetic study should also be performed.

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