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Recurrent Acute Pancreatitis as the First Presentation of Primary Hyperparathyroidism

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Abstract: ***Pancreatitis is a common non - bacterial inflammatory disease caused by activation, interstitial liberation and autodigestion of pancreas by its own enzymes. Common causes of acute pancreatitis are gall stones, alcohol, drugs, trauma, viral infections and hypertriglyceridemia. Much is known about the causes of pancreatitis but huge experimental data available about understanding of its pathogenesis is still incomplete. Hypercalcemia as a cause of pancreatitis is rarely reported. Hypercalcemia is usually the result of Primary hyperparathyroidism (PHPT) and the most common cause of PHPT is parathyroid adenoma. It is thought that the increased calcium concentration in pancreatic juice resulting from hypercalcemia may prematurely activate proteases. Mutations in different genes have been proposed as well to justify why only some patients with primary hyperparathyroidism and hypercalcemia develop acute pancreatitis. Here we present a case of recurrent acute pancreatitis resulting from hypercalcemia in a 26 - year - old male. Hyperparathyroidism was suspected when despite severe pancreatitis calcium level remained high and parathormone level was grossly raised.***

Keywords: Primary hyperparathyroidism, Acute Pancreatitis, Hypercalcaemia, Renal calculi, Spleenic calcification

1. Introduction

Primary hyperparathyroidism (PHPT) syndrome is an endocrine disorder, characterized by excessive secretion of parathyroid hormone from one or more parathyroid glands. The elevation of PTH usually leads to hypercalcemia and hypophosphatemia. Patients may present with classic skeletal disease, recurrent nephrolithiasis, or be asymptomatic, detected on routine biochemical screening. The clinical profile of PHPT in the western countries had shifted from a symptomatic disorder, toward a more asymptomatic state. However, PHPT has a variable clinical expression and symptomatic PHPT is still the predominant form of disease in many developing countries, with skeletal manifestation (osteitis fibrosis cystica) being very common. Earlier studies from India have attributed the greater severity of PHPT to delayed diagnosis and widely prevalent vitamin D deficiency. Hypercalcemia is considered to be a rare cause of pancreatitis but the true cause and effect relationship between PHPT and pancreatitis remains controversial. PHPT has been associated with different types of pancreatitis. Despite its rarity, a cause and effect relationship is still suggested by the fact that parathyroidectomy seems to prevent recurrence of pancreatitis.

Some patients suffer from 2 or more attacks of pancreatitis before a diagnosis of PHPT is made. We present a patient who had repeated admissions for acute pancreatitis, 4 episodes over a period of 18 months. PHPT was diagnosed after the fourth episode.

2. Case Presentation

A 26 year old male was admitted to tertiary care center in solapur with complaints of abdominal distention, breathlessness and loss of appetite. An initial evaluation revealed severe anemia and deranged coagulation profile. Diagnosis of chronic liver disease was made.

Patient was born of normal vaginal delivery with increase bleeding from umbilical cord at the time of birth. At 18 month of age patient presented with prolong bleeding from lips and Hematoma on lateral aspect of thigh (vaccine injection site). On evaluation Coagulation studies - Platelet count was 250000/mm2 , Bleeding time was >20min, Clotting time was >20 min (blood did not clot even after 4 hrs), Adhesion Test was Positive, Prothrombin time was 180+ sec (control - 18 sec), Partial Thromboplastin Time with Kaolin (PTTK) was 200+ sec (control - 38 sec), (PTTK correction - ½ patient plasma + ½ control plasma - 30 sec, ½ patient plasma + ½ aged serum – 180 sec, ½ patient plasma+ ½ adsorbed plasma – 46 sec), Factor Inhibitor were Absent, Thrombin test was 180+ sec (control - 16 sec), Fibrinogen was 40 mg%% (Normal 150 - 450 mg%). Diagnosis of Factor I (Fibrinogen) Deficiency was made.6 cryoprecipitates were transfused. Patient was treated with ayurvedic medications for next 1 and ½ years.

At age of 13 years, patient was admitted with complaints of abdominal pain with vomiting. On evaluation serum amylase was 21 IU/L, serum lipase was 4648 U/Lf. Abdominal ultrasound showed bulky pancreas with increased echogenicity and locculated margins, which was suggested of acute pancreatitis. Moderate ascites was also detected. Patient was diagnosed with Acute Pancreatitis and treated conservatively.

In the last 1 and 1/2 years, patient has been admitted three more times, each time with similar complaints. On present admission, patient had abdominal distention with respiratory distress and loss of appetite. On examination he also had severe pallor. On investigation **Investigations**	**Results**
Haemoglobin	5.5 gm/dl
Total count of WBC	11000/mm2
Platelet count	127000/mm2
Creatinine	1.3 mg/dl

ALT (SGPT)	69 U/L
S. Lipase	3263 U/L
S. Amyalse	56 U/l
S. Calcium	11.2 mg/dl
S. Alkaline Phosphatase	183 U/L
S. Parathyroid Hormone (PTH)	243.2 pg/dl
S. Magnesium	1.5mg/dl
S. Inorganic Phosphate	2.5mg/dl
Prothrombin Time	23 sec
Activated Partial Thromboplastin Time	>1 min

Contrast enhanced triple phase abdominal CT revealed liver cirrhosis with regenerative nodules; portal vein cavernoma with dilated porto - caval tributaries; severe pancreatic atrophy with pancreatic parenchymal calcification; bulky spleen with calcification in anterero - inferior part of spleenic parenchyma. Large right renal calculus in renal pelvis causing mild obstructive uropathy in right kidney with cortical scarring was also seen along wiith moderate ascites.

3. Discussion

The association between pancreatitis and PHPT is controversial. The first report of PHPT associated with pancreatic lithiasis was published in 1947 by. During 1950s and 1970s, the Mayo Clinic found that out of 1153 patients with PHPT, only 17 (1.5%) had coexisting pancreatitis, while alternative explanations for pancreatitis were found for several patients. However, several studies have suggested a causal association between pancreatitis and PHPT. The prevalence of acute pancreatitis in PHPT has been estimated to be between 1.5% and 13%.

PHPT has been associated with different types of pancreatitis. A study involving 83 cases of pancreatitis combined with PHPT found that about 70% of the patients suffered from acute relapsing or chronic pancreatitis. Some patients suffer from 2 or more attacks of pancreatitis before a diagnosis of PHPT is made. In a study from India, pancreatitis was associated in 6 of 87 patients (6.8%) with PHPT. Pancreatitis was the presenting symptom in 5 patients. All patients with a past history of pancreatitis had suffered two or more attacks.

Hyperparathyroidism causes a hypocoagulable state but the correlation between Hyperparathyroidism and Hypofibrinogenemia is yet to be studied.

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