AN INTERESTING CASE OF PANCREATITIS

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Abstract:
We report a case of male patient with pancreatitis associated with hypercalcemia as first manifestation of primary hyperparathyroidism caused by a benign bilateral inferior parathyroid adenoma. Initially the acute pancreatitis was treated conservatively. The patient subsequently underwent surgical resection of the parathyroid adenoma. Although the association of parathyroid adenoma-induced hypercalcemia and acute pancreatitis is a known medical entity, it is very uncommon. The pathophysiology of hypercalcemia-induced acute pancreatitis is therefore not well known, although some mechanisms have been proposed. It is important to treat the provoking factor. Therefore, the cause of hypercalcemia should be identified early. Surgical resection of the parathyroid adenoma is the ultimate therapy.

Keyword:
pancreatitis, primary hyperparathyroidism, parathyroid adenoma.

A forty seven year old gentleman presented with upper abdominal pain for one week duration. Pain increased on food intake, decreased on stooping forward and radiated to back. There was no history of vomiting, gastrointestinal bleed, jaundice, respiratory distress and abdominal distention. He was not an alcoholic and smoker. On examination, vitals were stable. Abdominal examination was unremarkable except epigastric tenderness. Basic blood investigations, liver function tests, renal function tests were within normal limits. Serum amylase was 1300 IU/L. Triglycerides, serum calcium and phosphorus were within normal limits. CECT abdomen showed edematous pancreas with ductal calculi, liver abscess, bilateral renal calculi. Patient was treated with IV fluids, antibiotics, analgesics and he improved symptomatically. Five months later he again presented with pancreatitis. MRCP revealed acute on chronic pancreatitis with resolved liver abscess. Metabolic parameters was repeated. Serum calcium was 12.4
mgs% and serum phosphorus was 2.2 mgs%. Serum parathyroid hormone level was 232 pg/ml which was highly elevated. Tc-99m sestamibi dynamic study revealed tracer retention in lower poles of thyroid gland. Thyroid function tests were normal. Bilateral inferior parathyroidectomy with total thyroidectomy was done, since the thyroid was found to be multinodular. Post operatively parathyroid hormone level came down to 11 pg/ml and serum calcium was 10 mgs%. Histopathology report revealed colloid goitre with bilateral inferior parathyroid adenoma. Patient improved and there were no further episodes of pancreatitis on follow up. He was advised on lifelong oral thyroxine.

**Discussion:**

Acute pancreatitis caused by primary hyperparathyroidism [PHPT] induced hypercalcemia is a rare condition. It was first described in 1957.

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**Tc-99m sestamibi dynamic study - 45 mins**

**Tc-99m sestamibi dynamic study - 1.5 Hours**

**CECT ABDOMEN - PANCREATIC CALCIFICATION AND RENAL CALCULI**

**HISTOPATHOLOGY - PARATHYROID ADENOMA**

Final diagnosis was bilateral inferior parathyroid adenoma with primary hyperparathyroidism presenting as pancreatitis.

**Discussion:**

Acute pancreatitis caused by primary hyperparathyroidism [PHPT] induced hypercalcemia is a rare condition. It was first described in 1957.
by Cope et al [1]. Since then, the relationship between PHPT and pancreatitis has been questioned, but nowadays PHPT has been acknowledged as an accepted etiology of pancreatitis. The prevalence of acute pancreatitis in patients with PHPT is estimated between 1.5% and 7% [2]. Some studies have associated hyperparathyroidism with pancreatitis, however, the prevalence of acute pancreatitis in patients with PHPT seems no different from that in the general population. Therefore, there appears to be no direct causal relationship between PHPT and acute pancreatitis based on epidemiological data. However, it has been shown that hypercalcemia of any cause can lead to acute pancreatitis. When this combination occurs, pancreatitis is likely to be severe and the degree of hypercalcemia may play an important role in this association. Three pathophysiological mechanisms are suggested. The deposition of calcium in the pancreatic duct may cause pancreatic duct obstruction [3]. Hypercalcemia may also lead to activation of trypsinogen within the pancreatic parenchyma causing autodigestion of the pancreas [4]. Finally, genetic variants in SPINK 1 (serine protease inhibitor Kazal type 1) and CFTR (cystic fibrosis transmembrane conductance regulator) genes in combination with hypercalcemia increase the risk of developing acute pancreatitis in patients with PHPT [5]. Usually, acute pancreatitis is associated with a decrease in serum calcium levels. Based on the Ranson grading, low serum calcium levels have prognostic importance. Therefore, it is uncommon to detect hypercalcemia in a patient presenting with severe acute pancreatitis. This unusual condition should always alert physicians to the presence of hyperparathyroidism. Metabolic parameters may be normal during the acute phase of pancreatitis. Hence the levels should be repeated once the patient is symptom free. In our patient the initial work up of serum calcium was normal. It was only in the repeat examination it was found to be elevated.

In order to complete the diagnosis, parathyroid hormone levels should be determined and imaging of the parathyroid glands is important. In this case, diagnosis of the parathyroid tumor was made by 99mTc-Sestamibi scintigraphy. Surgical resection and histological examination of the tumor is the ultimate therapy and parathyroidectomy prevent the recurrence of pancreatitis.

However, this patient already has chronic pancreatitis which is a rarer presentation of hypercalcemia than acute pancreatitis. As chronic pancreatitis is not reversible, parathyroid surgery cannot cure the chronic pancreatitis. However it reduces the further insults to the pancreas. Further follow up in necessary in these patients to predict the outcome.

Subsequent rapid hypocalcemia may develop following parathyroid removal requiring calcium supplementation. Good cooperation between gastroenterologists, endocrinologists and surgeons is important in treating this rare phenomenon of pancreatitis caused by PHPT-induced hypercalcemia.

**Conclusion:**

Primary hyperparathyroidism is a rare but treatable cause of pancreatitis. Metabolic parameters should be repeated even if the initial measurements are normal if the clinical suspicion is high. Timely identification of the cause will avoid unnecessary biliary and pancreatic surgeries.
REFERENCES:


