INTERNATIONAL JOURNAL OF MEDICAL AND APPLIED SCIENCES
E-ISSN:2320-3137

Case Report

PRIMARY HYPERPARATHYROIDISM PRESENTING AS RECURRENT ACUTE PANCREATITIS - A RARE CASE

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Abstract :
Primary hyperparathyroidism is excessive secretion of parathyroid hormone from one or more parathyroid glands. It results from an abnormally high level of serum calcium and an increased level of parathyroid hormone. However, hypercalcemia is thought to be a rare cause of pancreatitis as cause and effect relationship between primary hyperparathyroidism and pancreatitis remains controversial. Acute pancreatitis is an uncommon presentation of primary hyperparathyroidism. Patients may suffer from two or more attacks of pancreatitis before the diagnosis. Here we present a rare case of primary parathyroidism secondary to parathyroid adenoma who underwent repeated admissions with the diagnosis of acute pancreatitis for a period of nine months.
Keywords :Acute pancreatitis; Hypercalcemia; Primary hyperparathyroidism; Parathyroid adenoma

CASE REPORT

A 32 year old, non-alcoholic, male patient presented with complaints of epigastric pain which was acute onset, progressive, radiating to back and aggravated on food intake and on lying down and relieved on sitting position, with no diurnal variation.

A detailed history revealed that the patient had similar complaints since past 4 months and had referred to local hospital for it. Patient underwent a detailed examination followed by routine blood examination. All the blood investigations turned out normal except a raised level of serum amylase and lipase. The abdominal sonography showed a bulky pancreas with distended gall bladder, and bilateral renal calculi. Subsequent USG after 3 days revealed features of pancreatitis with cholilithiasis and with bilateral renal calculi. Patient underwent elective laparoscopic cholecystectomy in view of gallstones pancreatitis. Patient improved symptomatically following the surgery and was discharged 5 days later.

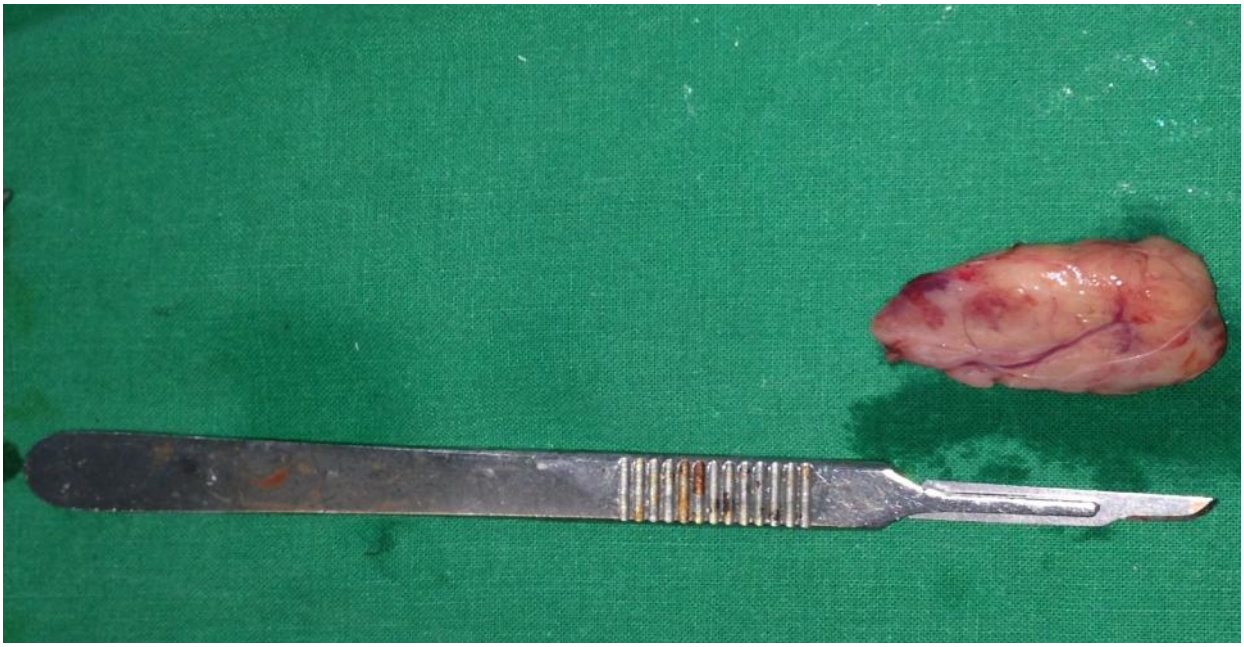
After four weeks, patient complained of similar epigastric pain. His repeat USG showed a repeated attack of acute pancreatitis with post cholecystectomy status and bilateral renal calculi. The blood tests also showed a high level of serum amylase and lipase. Patient was managed medically for acute pancreatitis and discharged once the symptoms subsided. After one month, patient again had similar complaints. The abdominal CT scan showed bulky pancreas with fluid collection adjacent to it with splenomegaly, prominent CBD and bilateral

renal calculi. This episode of pancreatitis was also managed medically and patient was discharged subsequently.

The patient was referred to our hospital when he developed subsequently during another episode of similar symptoms. His abdominal CT showed a bulky pancreas with a pseudo cyst posterior to the head, with size of 4.8 x 2.8 x 2.6 cm. Also there were few well defined hypo dense areas adjacent to head and body of pancreas involving porta hepatis and lesser sac measuring up to 10.9 x 7 x 5.2 cms, with a prominent pancreatic duct (4 mm in the body region). Based on the history blood investigations and imaging studies, patient was diagnosed as a case of biliary pancreatitis with post cholecystectomy status and managed conservatively.

On repeat USG after one week, there was evidence of new cystic lesion, approximately 7.5 x 4.3 cm with internal septations sub adjacent to the head and uncinate process. Subsequent USG done after 10 days showed normal head, body and tail of pancreas. Also, the hypoechoeic lesion sub adjacent to uncinate process of pancreas had reduced in size (3 x 2.7 x 2.1 cm).. A CA-19.9 level was done to rule out neoplastic etiology but was found to be within the normal limits (17.45 U/ML). Based on these findings the patient was diagnosed to have acute biliary pancreatitis with post cholecystectomy status.

Patient was thoroughly investigated for these repeated episodes of acute pancreatitis. It was then noticed that the patient had a higher level of serum PTH (240.9 pg /mL). The ultrasound of showed a well-defined hypoechoeic solid mass measuring 3.0 x 1.3 x 1.8 cm, seen, inferior to the right lobe of thyroid and was diagnosed as right para-thyroid adenoma. On further work up the serum calcium levels were also found high (13.7 m/dL). The patient was suspected to have hyperparathyroidism induced acute recurrent pancreatitis. Further, the patient developed a pancreatic pseudo cyst of size 14.5 x 8.0 cm. Patient underwent an elective parathyroid gland excision surgery with gastrocystostomy. The histological study of the excised gland confirmed the diagnosis of parathyroid adenoma. Further the patient is symptom free and has not developed any further episode of pancreatitis post surgery.



DISCUSSION

Acute pancreatitis caused by PHPT-induced hypercalcemia is a rare condition. It was first described in 1957 by Cope et al.[8] Since then, the relationship between PHPT and pancreatitis has been questioned, but nowadays PHPT has been acknowledged as an accepted aetiology of pancreatitis. The prevalence of acute pancreatitis in patients with PHPT is estimated between 1.5% and 7%.[9] 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.[10] Therefore, there appears acute pancreatitis based on epidemiological data.

PHPT has been associated with different types of pancreatitis, such as acute, subacute, or chronic calcifying 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. The same was true in our case that is, the patient had six episodes of acute pancreatitis over a period of nine months before he was diagnosed to have PHPT. 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. Serum calcium estimation after the first episode of pancreatitis would have eliminated this delay. Hence, it is important to estimate serum calcium after an episode of unexplained acute pancreatitis which will minimize the delay before the diagnosis of PHPT is made.[6]

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. Hypercalcemia may also lead to activation of trypsinogen within the pancreatic parenchyma causing auto digestion of the pancreas. Finally, genetic variants in SPINK 1 (serine protease inhibitor Kazal type 1) and CFTR (cystic fibrosis trans membrane conductance regulator) genes in combination with hypercalcemia increase the risk of developing acute pancreatitis in patients with PHPT.[9]

Usually, acute pancreatitis is associated with a decrease in serum calcium levels. Based on the Ranson grading, low serum calcium levels have prognostic importance. This unusual condition should always alert physicians to the presence of hyperparathyroidism or malignancy. In order to complete the diagnosis, parathyroid hormone levels should be determined and imaging of the parathyroid glands is important. It is also important to take into account the possibility of an ectopic localization of a parathyroid adenoma as a cause of acute pancreatitis.[9] Hypercalcemia can lead to de novo activation of trypsinogen to trypsin, resulting in autodigestion of the pancreas and subsequent pancreatitis. Another explanation is that hypercalcemia leads to the formation of pancreatic calculi, ductal obstruction, and subsequent attacks of acute or chronic pancreatitis. Also, genetic risk factors may predispose patients with PHPT to pancreatitis. The calcium level is probably of major importance in the development of pancreatitis. The mean calcium values among patients with PHPT and pancreatic disease have been reported to be significantly higher than those in patients with PHPT without pancreatic involvement.[6]

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