

## Case Report

### A Case Report on Recurrent Acute Pancreatitis with Primary Hyperparathyroidism.

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#### Abstract:

Pancreatitis is a common non-bacterial inflammatory disease caused by activation, interstitial liberation and auto digestion 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 due to parathyroid adenoma in a 38-year-old man. Hyperparathyroidism was suspected when despite severe pancreatitis calcium level remained high and parathormone level was grossly raised

**Key words:** Acute pancreatitis, Hypercalcemia, Primary hyperparathyroidism, Parathyroid adenoma.

#### Introduction:

Acute pancreatitis may have a great variety of etiologies. It is accepted that alcohol consumption and biliary lithiasis are responsible for almost 80-90% of all cases. Other, much more infrequent causes include toxics or drugs, neoplastic obstruction of the bile tract

or the sphincter of Oddi, metabolic disorders such as hypertriglyceridemia or hypercalcemia, trauma, ischemia, infection, autoimmune diseases, etc. Up to 10% of cases are described as idiopathic pancreatitis because no main cause of the disease could be established.

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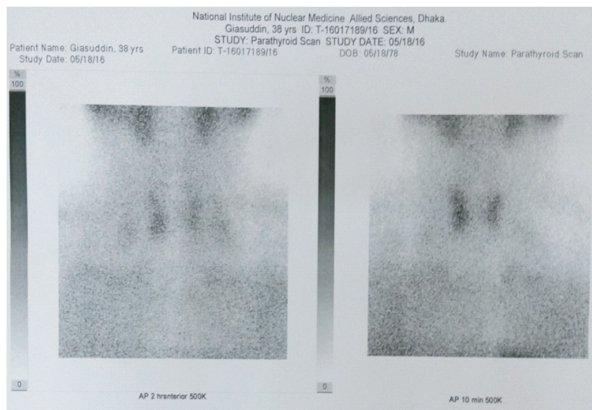
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Patients with hyperparathyroidism and hypercalcemia present with an increased risk of suffering acute pancreatitis, about 10 times above that of the general population. Nevertheless, pancreatic disease is a rare complication in these patients (approximately 2%). Elevated serum calcium levels associated with different mutations in several genes could be responsible for this predisposition in some patients with hyperparathyroidism. In this respect some studies have been already carried out with the SPINK1 (serine protease inhibitor Kazal type 1), CFTR (cystic fibrosis transmembrane conductance regulator), and CASR (calcium-sensing receptor) genes. Acute pancreatitis episodes are commonly seen in patients already diagnosed with hyperparathyroidism, or immediately after parathyroidectomy, but it is exceptional as a first manifestation of hyperparathyroidism.

#### Case Report:

A 38-year-old diabetic male with hypertension was admitted on 12 April 2016 in Bangabandhu Sheikh Mujib Medical University (BSMMU) Dhaka with recurrent episodes of upper abdominal pain for last 2

years. Each episode occurred 1-2 months interval and persisted for about 5-7 days. He gave no history of indigestion, vomiting, change of bowel habit or change in color of his stool or urine. He smoked ten cigarettes per day for fifteen years but had no history of drug or alcohol abuse. He remained completely symptom free in between the episodes. He admitted himself to a private hospital 10 months back for abdominal pain with high serum amylase and Magnetic Resonance Cholangio Pancreatography (MRCP) was done which was negative, whereupon he was diagnosed with idiopathic pancreatitis. Further evaluation to confirm the etiology of recurrent pancreatitis, we found elevated serum calcium level and parathyroid levels. Ultrasound examination and SPECT scan with Tc-99 m-sestamibi localized a solitary mass below the right lobe of the thyroid gland (Figure I).



**Figure 1:** <sup>99m</sup>Tc-sestamibi scintigraphy of parathyroid glands; Early & delayed images showed fairly uniform and radiotracer concentration & persistence of radiotracer concentration in lower pole of right lobe respectively- suggestive of parathyroid adenoma/hyperplasia in lower pole of right lobe

The thyroid gland was not enlarged. The rest of the physical examination was unremarkable.

Investigations (Table I) revealed a high white blood cell count with normal hemoglobin and platelet counts, raised glucose level, elevated lipase, high calcium and low inorganic phosphate with raised parathyroid hormone (PTH) levels. Kidney and liver function tests, albumin and triglyceride levels were all within normal limits. USG of thyroid and <sup>99m</sup>Tc-sestamibi scintigraphy of parathyroid glands revealed right sided parathyroid adenoma. Following his recovery from acute pancreatitis, the patient then underwent an open bilateral neck exploration, which revealed a right inferior parathyroid adenoma; the mass was removed.

**Table-I:** Investigations

Investigations	Result
Haemoglobin	13.8 gm/dl
ESR	75 mm in 1 <sup>st</sup> hour
Total count of WBC	16000/mm <sup>3</sup>
Platelet count	350000/mm <sup>3</sup>
FBS	7.4 mmol/L
Sugar 2hrs ABF	15.1 mmo/L
HbA <sub>1c</sub>	9.1%
Creatinine	0.89 mg/dl
ALT (SGPT)	54 U/L
S. Lipase	3250 U/L
F. Triglycerides	138 mg/dl
S. Albumin	40 gm/L
S. Calcium	11.2 mg/dl
S. Alkaline Phosphatase	179 U/L
S. Parathyroid Hormone (PTH)	233.4 pg/ml
S. Magnesium	1.7 mg/dl
S. Inorganic Phosphate	2.2 mg/dl
USG of Abdomen	Normal
X-Ray abdomen	Normal
MRCP	Normal
USG of Thyroid Gland	A hypo echoic well circumscribed soft tissue mass(measuring 17.4 x 11.4 mm) is seen in lower pole of right lobe of thyroid gland- suggestive of a Parathyroid gland.
<sup>99m</sup> Tc-sestamibi scintigraphy (Figure I)	Early & delayed images showed fairly uniform and radiotracer concentration & persistence of radiotracer concentration in lower pole

### Discussion:

Hyperparathyroidism is a rare cause of pancreatitis. The hypercalcemia seen with PHPT has been associated with both acute and chronic pancreatitis since the mid-20<sup>th</sup> century. The first report dates back to 1940 by Smith and Cook<sup>1</sup>. Prinz et al<sup>2</sup> in their study of 1475 patients with acute pancreatitis found HPT accounting for only five (0.4%) cases. However, in patients with HPT and resulting hypercalcemia, pancreatitis occurs ten to twenty times more often than

in the general population. The main causes of primary HPT are single or double parathyroid adenoma (80%), hyperplasia of all four or more existing parathyroid glands (15-20%) and rarely cancer (2%) of the parathyroid gland<sup>3-5</sup>. It is postulated that hypercalcemia leads to accelerated intra-pancreatic conversion of trypsinogen to trypsin, which causes the pancreatic damage<sup>6</sup>. Normally hypocalcaemia is expected during an attack of pancreatitis and hypercalcemia is a strong clue for suspecting HPT<sup>7-9</sup>.

Thus, whenever acute pancreatitis is associated with hypercalcemia, the clinician should entertain a possibility of hypercalcemia related pancreatitis and try to exclude HPT. Regarding treatment; presence of PHPT does not alter the acute management of pancreatitis episodes, which should focus on diligent supportive care<sup>10</sup>. After the acute attack has resolved, patients should undergo elective parathyroidectomy to definitively treat the PHPT. Parathyroidectomy relieves pancreatic pain in majority of patients<sup>11</sup>. In two studies showed either recurrence of pancreatitis in the 1 patient who underwent resection<sup>12</sup> or attributed some of the improvement to a reduction in heavy alcohol use<sup>13</sup>.

### Conclusion:

Acute pancreatitis is one of the symptoms of primary hyperparathyroidism caused by a parathyroid adenoma. Hypercalcemia and hyperparathyroidism should be a high index of suspicion in all fresh cases of acute pancreatitis even though primary hyperparathyroidism is a rare cause. Any oversight will result in diagnostic delays. Complementary explorations such as serum calcium and intact parathyroid hormone levels, and imaging techniques such as cervical ultrasounds, computed tomography and scintigraphy using <sup>99m</sup>Tc-Sestamibi, should be ordered which will lead to confirm clinical suspicion. Minimally invasive surgery following modern imaging technique carries an excellent prognosis.

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