

CASE REPORT

ABDOMEN GROANS: A RARE CAUSE OF PANCREATITIS

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ABSTRACT: Hyperparathyroidism and hypercalcaemia are considered to be a rare cause of acute pancreatitis. The relationship between hyperparathyroidism and pancreatic inflammatory disease remains controversial.⁽¹⁾ But it has been shown that surgical correction of parathyroid disease and normalization of serum calcium levels may ameliorate the acute pancreatitis.⁽²⁾ A case of acute pancreatitis and hyperparathyroidism due to parathyroid gland hyperplasia occurred in a 51-years-old woman is reported. After the excision of parathyroid gland the serum calcium levels and the function of the pancreas returned to normal. This suggests a cause and effect relationship between hyperparathyroidism and acute pancreatitis.⁽³⁾

INTRODUCTION: Although an uncommon complication, hypercalcemia of any cause can lead to acute pancreatitis. Proposed mechanisms include deposition of calcium in the pancreatic duct and calcium activation of trypsinogen within the pancreatic parenchyma.⁽⁴⁾ There are numerous reports associating hyperparathyroidism with pancreatitis.⁽⁵⁾ However, the actual incidence of acute pancreatitis in patients with hyperparathyroidism is low. The low incidence of pancreatitis in patients with chronic hypercalcemia suggests that other factors (e.g. acute elevations of serum calcium) are responsible for pancreatitis in these patients. We here present a 51 year old women with acute pancreatitis (probably recurrent pancreatitis⁽⁶⁾) due to hypercalcemia caused by primary hyperparathyroidism.

CASE REPORT: 51 year old female, presented with epigastric pain, vomiting with intermittent fever for 5 days. History of similar episodes 7 years & 3 years back were treated conservatively as acute gastritis without much evaluation. She is a known diabetic & hypertensive on regular treatment under good control. On examination she was dehydrated. Pulse-82/min, BP-110/70mm Hg. CVS, RS, CNS-unremarkable. P/A-mild tenderness in the epigastrium.

Lab studies revealed CBC –normal, RFT, LFT-normal; AMYLASE-971U/L & LIPASE-2060U/L (Grossly elevated⁽⁴⁾). USG abdomen-minimal pancreatic fat stranding with prominent pancreatic duct. Patient was diagnosed as acute pancreatitis and was started on IV fluids, analgesics & kept NPO. Meanwhile further work up for the cause of pancreatitis was initiated.

CT abdomen was done and the findings were consistent with USG report and ruled out any congenital defect. Other causes like: mechanical toxic, traumatic, drug induced causes were ruled out both by history & necessary investigations. Then serum Calcium & Triglycerides were done for metabolic causes. TGL-122mg % & Ca-12mg% (elevated).

In view of hypercalcemia patient was further investigated for the cause of the same. As a next step i PTH was done iPTH levels-155(12-65pg/dl)-increased; phosphorous-2.2mg%-decreased vit D-13.30ng/ml (low)^(7, 8)

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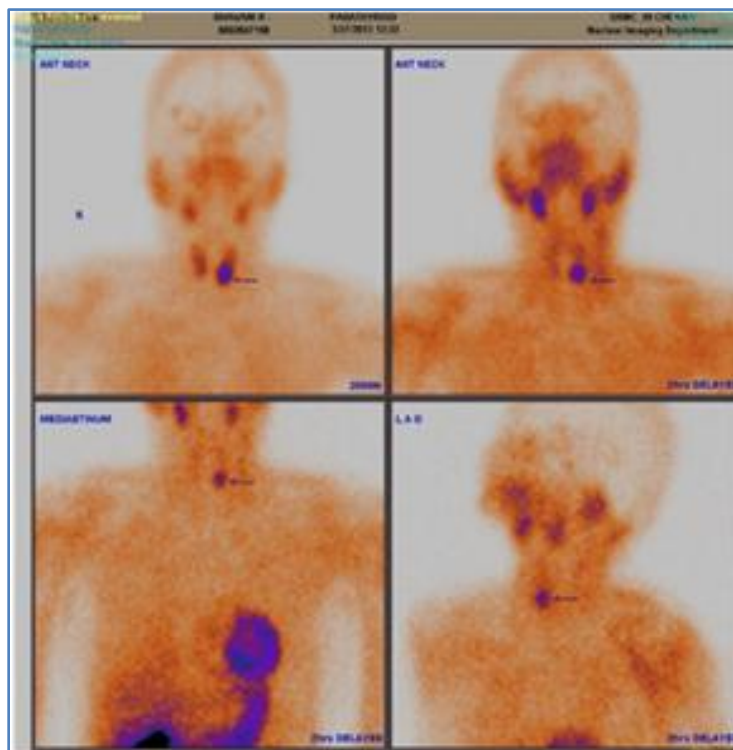


Fig. 1: 99M TC MIBI scan showing increased uptake in Inferior Pole of Left Lobe of Thyroid

Sestamibi scan of parathyroid as shown in fig. 1 showed aberrant tracer uptake in lower pole of left lobe of thyroid; 24 hour urinary calcium-300mg (normal); BMD showed no evidence of osteoporosis⁽⁸⁾

With all the above findings we finally made a diagnosis of primary hyperparathyroidism (PHPT) which is responsible for hypercalcemia causing pancreatitis.

Then we sought the help of a surgeon for removal of Parathyroids Intra op findings showed nodule on left lobe of thyroid. Hence left hemi thyroidectomy with left superior & inferior parathyroidectomy. On the operative table PTH assay was done both before and after removal of parathyroids. PTH value decreased after the removal of parathyroids. HPE showed hyperplasia of parathyroid glands with no evidence of adenoma/malignancy & also nodular goiter of thyroid. Serial levels of PTH, Ca, Lipase and amylase are shown in table as shown below.

Timing of test	PTH	Calcium	Amylase	Lipase
Admission	200	12.1	971	2060
Intra op	360			
After removal	180			
Post Op day2	100	10	65	85
Discharge	80	10	62	64
Follow up-1 m	60	9	52	60

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Repeat sestamibi scan shows absence of increased tracer uptake region in delayed film as shown in Fig. 2.

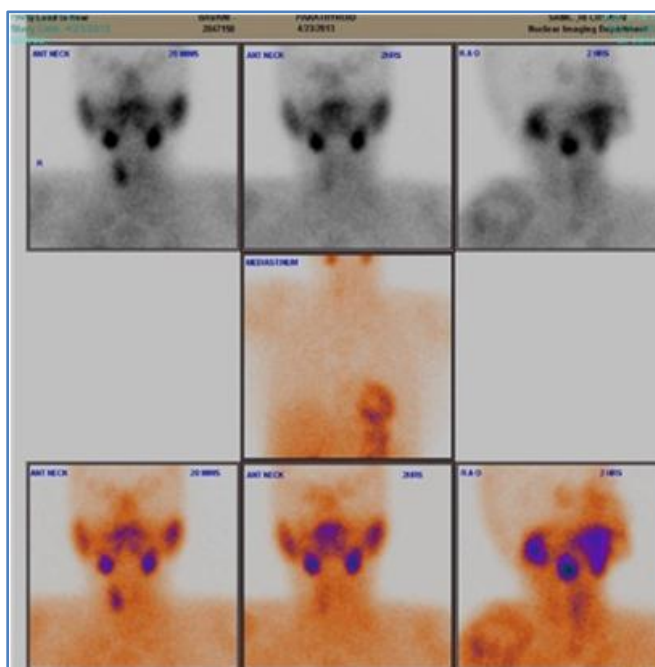


Fig. 2: Sestamibi scan shown absence of tracer in the inferior pole of left thyroid in delayed film (3rd scan)

Patient is being followed up even now almost 2 years after surgery and she has not developed further episodes of pancreatitis with normal, calcium & normal PTH levels.

DISCUSSION: Acute pancreatitis is an inflammatory condition of the pancreas characterized clinically by abdominal pain and elevated levels of pancreatic enzymes in the blood.⁽⁴⁾ The pathogenesis of acute pancreatitis is not fully understood. Nevertheless, a number of conditions are known to induce this disorder with varying degrees of certainty, with gallstones and chronic alcohol abuse accounting for 75 percent of cases. This list will undoubtedly continue to grow, and the number of cases diagnosed as "idiopathic" will decrease as our understanding of the disease improves. Hypercalcemia is one of the rare causes of pancreatitis.

There are 2 mechanisms of hypercalcemia-induced pancreatitis. Hypercalcemia can lead to de novo activation of trypsinogen to trypsin, resulting in autodigestion of the pancreas and subsequent pancreatitis.⁽⁹⁾ Another explanation is that hypercalcemia leads to the formation of pancreatic calculi,⁽¹⁰⁾ 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.

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The association between pancreatitis and PHPT is controversial.⁽¹⁾ The first report of PHPT associated with pancreatic lithiasis was published in 1947.⁽¹⁰⁾ The Mayo Clinic experience between 1950 and 1975 found that out of 1153 patients with PHPT, only 17 (1.5%) had coexisting pancreatitis, and alternative explanations for pancreatitis were found for several patients.⁽³⁾ But several studies have suggested a causal association between pancreatitis and PHPT.⁽³⁾ The prevalence of acute pancreatitis in PHPT has been estimated to be between 1.5% and 13%.

Despite its rarity, a cause and effect relationship is still suggested by the fact that parathyroidectomy seems to prevent recurrence of pancreatitis⁽⁶⁾ as seen in our case. Nearly 100% improvement in pancreatitis symptoms after the cure of PHPT has been reported.

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