Acute Pancreatitis: Association with Primary Hyperparathyroidism: A Case Report

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Abstract

Association between primary hyperparathyroidism and acute pancreatitis is very rare. We report a case of a 45-year-old female presented with acute pancreatitis. Primary hyperparathyroidism was diagnosed after that episode of pancreatitis. She had no additional risk factor for pancreatitis. It’s been 10 months after her successful parathyroid surgery, there has been no recurrence of abdominal pain and her serum calcium is within normal limits. The purpose of reporting this specific case is that this pathology is uncommon in occurrence and indicates the importance of keeping in mind about all the causes of pancreatitis before declaring it as idiopathic.

Conflict of Interest: None
Received: 07.02.2022
Accepted: 02.05.2022
www.banglajol.info/index.php/JSSMC

Key Words:
Primary hyperparathyroidism, pancreatitis, scintigraphy, parathyroid surgery

Introduction

PHPT is defined as hypercalcemia in the presence of an unsuppressed and therefore relatively, or absolutely, elevated PTH level. Patients are typically identified incidentally with an elevated total calcium or following routine assessment of bone densitometry (DEXA scan). Most patients will, however, have some vague constitutional symptoms, such as fatigue, muscle weakness, depression, or some mild memory impairment on questioning.

The presence of kidney stones remains the most common clinical manifestation of symptomatic PHPT. PHPT may present with pancreatitis, although it is rarely seen in patients with milder forms of the disease.¹

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.² Some patients suffer from 2 or more attacks of pancreatitis before a diagnosis of PHPT is made.³

We present a patient who was admitted for acute pancreatitis then diagnosed as having PHPT thereafter.

Case report

A 45-year-old female was first admitted to a district hospital with severe abdominal pain. There she was treated conservatively. After 2 days when her symptoms didn’t subside then she was referred to our institute. She was clinically suspected to have acute pancreatitis. She had elevated serum amylase and serum lipase level. On CT scan of whole abdomen, she had enlarged pancreas with irregular outline.
As a part of severity scoring of acute pancreatitis, she serum calcium level was advised. It showed calcium- 12.59 mmol /l (raised) (8.5-10.30 ). The serum calcium at the upper limit of normal gave clue that she may have any hypercalcemic disorder. Then serum parathyroid hormone and phosphate level was done which showed PTH1255.9pg/ml (raised) (15-65), Inorganic phosphate- 1.5 mg/dl (low) (2.5-5).

**Ultrasonography of neck** revealed one small hypoechoic area measured about 5 x 4.5 mm in size seen in right lobe of thyroid gland, suggestive of enlarged left sided parathyroid gland (parathyroid adenoma).

**Fig.-1:** CT scan of whole abdomen showing enlarged pancreas with irregular outline

**Fig.-2:** Ultrasonography of neck showing parathyroid adenoma

**Fig.-3:** Parathyroid scintigraphy showing left parathyroid adenoma

**Fig 4:** SPECT image of parathyroid gland

**Parathyroid scintigraphy**
Early static image shows thyroid gland is normal in position, right lobe is mildly enlarged in size. The focal area of increased radiotracer concentration is seen in the lower pole of left lobe with homogenous radiotracer concentration in rest of thyroid gland. Delayed static image shows persistent focal activity in above mentioned site with partial washout of tracer from rest of thyroid gland.

SPECT-CT Image shows, focal area of increased radiotracer concentration in lower pole of left lobe at the level of c6-c7 vertebrae in left paratracheal region impression -scan is positive for parathyroid adenoma/hyperplasia in the left lobe of thyroid gland.
She denied any history of bone pains, bone fractures, neuropsychiatric symptoms, or muscle weakness. Parathyroid adenoma was removed. Histopathology report showed compatible with parathyroid adenoma.

Fig.-5: Section shows parathyroid adenoma, partly covered by capsule (H&E *80)

Fig.-6: Parathyroid adenoma H&E*120

The postoperative period was uneventful. Ten months after successful parathyroid surgery, there has been no recurrence of abdominal pain and his serum calcium is within the normal range. His current biochemical parameters are serum calcium 8.6mg/dL; iPTH 19.7 pg/mL.

Discussion

The association between pancreatitis and PHPT is controversial. The first report of PHPT associated with pancreatic lithiasis was published in 1947. But several studies have suggested a causal association between pancreatitis and PHPT. 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.

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.

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. Another explanation is that hypercalcemia leads to the formation of pancreatic calculi, ductal obstruction, and subsequent attacks of acute or chronic pancreatitis.

In our patient serum calcium level was done as a part of assessing severity in her first episode of acute attack. This helped in diagnosis of the cause as well. In a way it prevented further attack also if it were undiagnosed.

References

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