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Case Report

A case report: acute necrotizing pancreatitis secondary to hypercalcemia in case of parathyroid adenoma

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ABSTRACT

The incidence of primary hyperparathyroidism (PHPT) in India is 2.5/1000 individuals. The major cause for PHPT is parathyroid adenoma. To arrive at the correct diagnosis, clinical setting, biochemical and radiological investigations, the status of other glands assessed lntraoperatively and finally histopathological confirmation is essential. The association between acute pancreatitis and PHPT has widely been reported in literature, but a causal relationship remains controversial. This case report is about a patient admitted with acute pancreatitis as a first manifestation of a parathyroid adenoma. The purpose of this paper is to keep in mind the metabolic etiology, by evaluating calcium and parathyroid hormone (PTH) in the differential diagnosis of non-Biliary, non-alcoholic acute pancreatitis.

Keywords: Hyperparathyroidism, Parathyroidectomy, Acute pancreatitis, Parathyroid adenoma, 99mTc-Sestamibi scan

INTRODUCTION

Primary hyperparathyroidism is part of a spectrum of parathyroid proliferative disorder that includes parathyroid adenoma, parathyroid hyperplasia, and parathyroid carcinoma. Serum parathyroid hormone and serum calcium levels are usually increased in patients. An inflammatory condition known as acute pancreatitis affects the exocrine portion of the pancreatic gland. Up to 75% of the time, gallstones and alcohol addiction are the main contributing factors.[1] 5%–10% of cases of pancreatitis are caused by metabolic disorders.[1] The coexistence of the two diseases has widely been reported in literature, but a causal relationship remains controversial.

We report a case of hypercalcemia-induced acute pancreatitis as the manifestation of a benign parathyroid adenoma in a female patient.

CASE REPORT

A 41-year-old female presented with complaints of severe abdominal pain with abdominal distention since 3 weeks. Tenderness was noted at the epigastric area with no palpable masses. There was bruising around the umbilicus with no obvious ascites. She had undergone left side Double J stenting 15 days back for left upper ureteric calculi. The patient looked ill and dehydrated. Laboratory results showed a calcium value of 11.78 mg/dl with an intact PTH level of 1589 pg/ml. Also her serum amylase level was 885 U/l and serum lipase gives a value of 1013 U/l. No risk factors causing acute pancreatitis were found, such as alcohol consumption, hyperlipidemia and gallstones. A right-sided neck swelling was obvious on inspection; on palpation a mobile non-tender nodule could be felt. Ultrasound scan of abdomen was reported as acute pancreatitis with phlegmonous collection in peripancreatic area with no gallstones. Ultrasound scan of neck showed 3×1 cm size of hypoechoic well defined lesion in thyroid

bed posteroinferior to right lower pole of thyroid in right parathyroid region. A neurological examination was unremarkable. She has normal thyroid-stimulating hormone level. Her renal functions were within normal limits. Computed tomography (CT) scan of abdomen showed edematous pancreas and large fluid collection (10×3 cm) in body and tail of pancreas extending to splenic hilum. Pancreas shows decrease enhancement on post contrast study giving a picture of acute pancreatitis with pancreatic necrosis of around 30%. CT scan of neck showed well defined hypodense enhancing lesion of size 23×12×11 mm on posteroinferior aspect of lower pole of right lobe of thyroid. Patient then underwent a 99mTc-sestamibi scan. The scan suggests a parathyroid adenoma. Patient was diagnosed with acute necrotizing pancreatitis secondary to hypercalcemia in case of parathyroid adenoma. Treatment of hypercalcemia was induced with Furosemide. After treating symptomatically and monitoring the serum calcium levels patient improved gradually. Patient was taken for minimally invasive right parathyroidectomy. The preoperative parathyroid hormone level was 1842 pg/ml and 10 minutes post tumor excision the Parathyroid hormone levels dropped to 182.2 pg/ml, a nearly 90% fall in PTH levels suggestive of successful tumor excision.

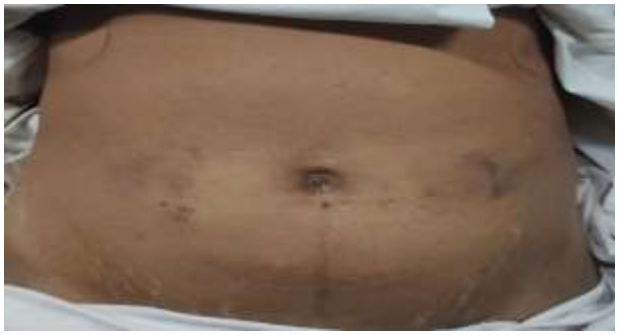

Figure 1: Cullen sign (superficial bruising around umbilicus) in acute pancreatitis.

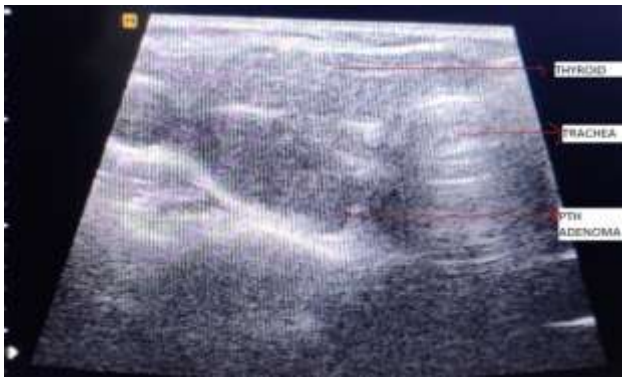

Figure 2: Star mark representing the PTH adenoma in USG picture.

Final histopathology report was consistent with parathyroid adenoma. Post-operative period was uneventful. In the first 24 hours, an intravenous infusion (drip) of calcium gluconate was given to prevent bone hunger syndrome. Solid diet was started in the same 24 hours, and the patient was discharged on the second postoperative day with oral Calcium carbonate and calcitriol pearls gradually tapered until discontinued after 2 weeks.

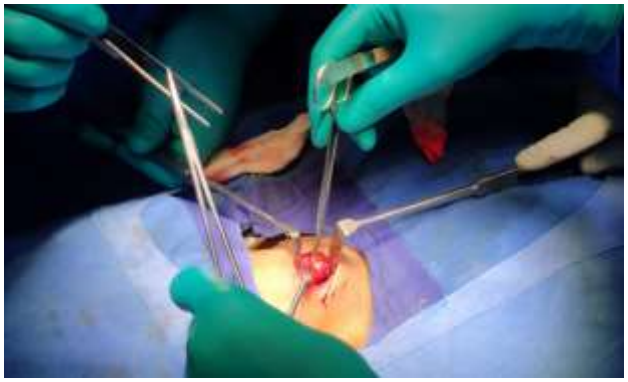

Figure 3: Intra-operative picture of minimal invasive parathyroidectomy.

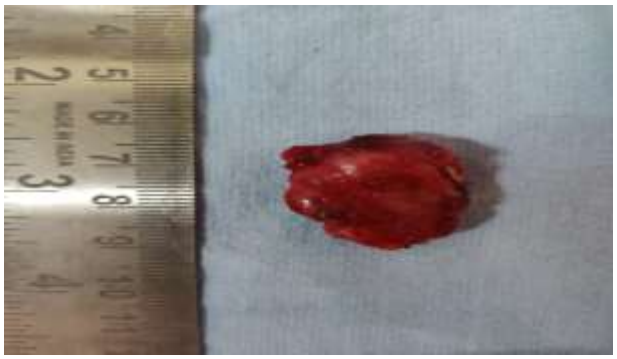

Figure 4: PTH adenoma specimen.

DISCUSSION

Acute pancreatitis due to PHPT-induced hypercalcemia is rare. Acute pancreatitis caused by PHPT is triggered by three different pathways. One of these is increased serum calcium levels brought on by PHPT, which can hasten the pancreas' conversion of trypsinogen to trypsin, leading in pancreatic autolysis and consequent acute pancreatitis. Second, calcium buildup can encourage the development of pancreatic calculi, ductal blockage, and future episodes of acute pancreatitis. Thirdly, people with PHPT are significantly more likely to get acute pancreatitis if they have genetic variations in the cystic fibrosis trans-membrane conductance regulator and serine protease inhibitor Kazal type 1 genes in addition to hypercalcemia.[1]

Imaging cannot detect normal parathyroid glands because they are too small, but parathyroid illness often causes the glands to grow and become visible. The main imaging modalities used to visualize diseased glands are sonography and 99mTc preoperative sestamibi (MIBI) scans.[2]

On clinical ground our patient had history of urinary calculi followed by abdominal pain which upon further blood evaluation gives a picture of parathyroid adenoma. The first line of investigation after suspecting a PHPT picture complicating to other forms of presentation should be ultrasonography as it is non-invasive. The MIBI scan is a viable option for identifying thyroid and parathyroid tumors because it is 90% sensitive for localizing a parathyroid adenoma. Acute pancreatitis is typically only occasionally caused by hypercalcemia brought on by hyperparathyroidism. Between 1.5% and 7% of patients with primary hyperparathyroidism were found to have acute pancreatitis.[3] Elevated serum calcium associated with pancreatitis should alert the physician to either hyperparathyroidism or malignancy.

Hyperparathyroidism, which develops when the usual feedback control by serum calcium is disrupted or there is an elevated production of PTH, has been mostly caused by parathyroid adenoma.[2] According to clinical investigations, individuals presenting with parathyroid tumor frequently have hypercalcemia. Ultrasonography and 99mTc-MIBI scintigraphy make parathyroid adenoma detection easier because of the rise in PTH levels and parathyroid function.[4] In affluent nations, the asymptomatic variant of PHPT is more prevalent, but the symptomatic variations are more common in India.[5]

The surgical exploration of the neck and removal of pathological parathyroid glands is the treatment for parathyroid tumors. This is followed by a biopsy of parathyroid gland to check for the presence of an adenoma or multiple gland hyperplasias. It is vital to take into account the superior exploration of the mediastinum if a parathyroid tumor was not discovered.[6]

The surgical procedure most frequently used to treat primary hyperparathyroidism is minimally invasive parathyroidectomy. The best application of intraoperative PTH monitoring is as a supplement to preoperative imaging, enabling more targeted surgeries. To help assess the quantity of surgical treatment necessary to be considered optimal, intra operational PTH monitoring can provide crucial information in just a few minutes. Success is defined using the Miami criteria: a fall in PTH level of >50% at 10 min post-excision compared to baseline (pre-operative).[2]

CONCLUSION

According to the literature review and studying this case report of acute pancreatitis caused by PHPT induced hypercalcemia, we want to emphasis on measuring the levels of calcium and parathyroid hormone in cases of acute pancreatitis where there is no evidence of gallstone or alcohol consumption. Symptomatic parathyroid adenoma cases present with high preoperative PTH and serum calcium level in a directly proportional manner. Once the biochemical and radiological investigation confirms the presence of parathyroid adenoma, surgical excision is the primary and only modality of treatment. Post excision of gland in our case, PTH levels have returned to normal within 10 minutes of surgery which is conclusive evidence of successful excision of the parathyroid adenoma and symptomatic improvement was noted after the surgery was performed.

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REFERENCES

1. Moula FZB, Ihab Y, Jaouad N, Arsalan O, Malki HOE, Chefchaouni AMC, et al. Parathyroid Adenoma Revealed by Acute Pancreatitis: Case Report. SAS J Surg. 2022;8(8):505-7.
2. Kantharia SR, Kantharia RA, Reddy PK, Mistry YM. Parathyroid adenoma: a case report. Int J Otorhinolaryngol Head Neck Surg. 2020;6(8):1533-7.
3. Egea Valenzuela J, Belchí Segura E, Sánchez Torres A, Carballo Alvarez F. Acute pancreatitis associated with hypercalcemia. A report of two cases. Rev Esp Enferm Dig. 2009;101(1):65-9.
4. Mahmodlou R, Sedokani A, Pezeshk A, Najafinejad B. Giant parathyroid adenoma: a case report. J Med Case Rep. 2022;16:150.
5. Jena A, Patnayak R, Suresh V, Kalawat TC, Phaneendra BV, Lakshmi AY, et al. Parathyroid adenomas: A case series and clinicopathological study from a tertiary care center in South India. Med J DY Patil Univ. 2016;9:495-8.
6. Prihantono P, Palinggi E, Haryasena H, Hamdani W, Binekada IMC. Surgical Treatment for Parathyroid Adenoma: A Case Report. Open Access Maced J Med Sci. 2019;7(15):2497-501.

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