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Case Report

Acute pancreatitis as a rare initial manifestation of 99mTc sestamibi-negative parathyroid adenoma: a case report

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ABSTRACT

Parathyroid adenoma is the most common cause of hyperparathyroidism. Hypercalcemia secondary to a benign parathyroid adenoma is a rare cause of acute pancreatitis. This report describes a 57-year-old woman who presented to us with acute pancreatitis. On further evaluation, she was found to have elevated calcium and parathyroid hormone levels. Imaging revealed a 99mTc sestamibi-negative parathyroid adenoma. Diagnosis was confirmed by washout parathyroid hormone measurement from the fine needle aspiration cytology (FNAC) sample. The patient then underwent left superior parathyroidectomy. Postoperatively, the patient had no further episodes of acute pancreatitis or other manifestations of hypercalcemia. The mainstay of treatment for patients with primary hyperparathyroidism includes appropriate resuscitation and stabilization with anti-hypercalcaemic measures, such as hydration and forced calciuresis, followed by definitive surgery.

Keywords: Acute pancreatitis, Hyperparathyroidism, Parathyroid adenoma

INTRODUCTION

A parathyroid adenoma (PA) is a benign tumour of the parathyroid glands and is usually responsible for 80% to 85% of hyperparathyroidism cases.[1] The clinical presentation of primary hyperparathyroidism (PHPT) ranges from asymptomatic cases to those with classical features of hypercalcaemia, such as painful bones, kidney stones, and abdominal and psychic groans, whereas acute pancreatitis (AP) is a rare initial manifestation. Acute pancreatitis is an inflammatory condition that is associated with significant morbidity and can be a potentially fatal condition with an overall mortality of up to 20%.[2] Given the dangers of delayed diagnosis of acute pancreatitis, it is crucial to be aware of rare causes and atypical presentations. One such rare cause is hyperparathyroidism with hypercalcemia.

The association between hypercalcemia and pancreatitis is supported by studies showing that parathyroidectomy can prevent the recurrence of pancreatitis in patients with hyperparathyroidism.[3] Addressing the root cause of hypercalcemia and surgically managing a parathyroid adenoma can aid in the treatment of pancreatitis related to PHPT. Herein, we report on a case of a 57-year-old woman without any common risk factors for pancreatitis, who was admitted to the emergency department with a diagnosis of acute pancreatitis. Further workup revealed PHPT due to a functioning 99mTc sestamibi-negative parathyroid adenoma.

CASE REPORT

A 57-year-old woman presented to the emergency department with sudden-onset epigastric pain radiating to the back and multiple episodes of non-bilious, non-bloody vomiting for two days. She did not pass stools for three

days; however, however passed flatus freely. There were no similar episodes in the past. She did not undergo any previous abdominal surgeries and was not on any regular medications.

On examination, she was found to be conscious and oriented but severely dehydrated, afebrile, with pulse rate of 120/min and blood pressure of 140/80 mm Hg. Physical examination revealed diffuse abdominal tenderness with voluntary guarding. There was no mass palpable in the abdomen, no free fluid and bowel sounds were present. Digital rectal examination revealed collapsed and empty rectum with normal stool staining. Her other systemic examination findings were clinically unremarkable. All baseline blood investigations were done, which showed leucocytosis with elevated amylase and lipase values suggestive for acute pancreatitis (Table 1).

Table 1: Showing all laboratory investigations done during admission.

Baseline blood investigations	Values	Reference values
White blood cell count ($\times10^9$/l) (% neutrophils)	22.82 (82%)	4-11 (45-70%)
Serum amylase (U/l)	426	22-80
Serum lipase (U/l)	410	<60
Serum calcium (mg/dl)	21	8.5-10.1
Alkaline phosphatase (U/l)	599	45-129
Vitamin D total (ng/ml)	6.38	>30
Phosphorus (mg/dl)	1.9	2.5-4.9
Parathyroid hormone (pg/ml)	1221	15-65

Contrast-enhanced computerized tomography (CECT) scan of the abdomen was performed and showed acute necrotising pancreatitis with severity score of 10/10, mild ascites and right-sided pleural effusion.

On further evaluation for the aetiology of acute pancreatitis, she was found to have serum calcium of 21 mg/dl. Thus, serum parathormone (PTH) levels were tested and found to be 1221 pg/ml. Patient was admitted to the surgical intensive care unit where she developed an episode of supraventricular tachycardia and underwent cardioversion. Repeat calcium values ranged from 18-21 mg/dl and patient was diagnosed to be in hypercalcaemic crisis. She was started on anti-hypercalcaemic measures, including intravenous fluids with forced calciuresis with diuretics to normalize her serum calcium levels, and underwent two cycles of haemodialysis. Serum calcium decreased to 12 mg/dl.

Ultrasonography (USG) of the neck revealed a 2.7×1.8×1.2 cm hypoechoic mass, likely representing a left parathyroid adenoma. To confirm, technetium-99m methoxy-isobutyl-isonitrile (Tc-99m MIBI) dual-phase parathyroid scintigraphy was done which showed no abnormal tracer uptake (Figure 2). USG-guided fine needle aspiration cytology (FNAC) was done, samples were sent for both pathological and biochemical analysis (Figure 1). Washout PTH (PTHw) measurement was done and showed PTH value of >50,000 pg/ml, thereby localising parathyroid adenoma. Cytology was reported to be positive for neoplasm showing cohesive cells with coarse chromatin in eosinophilic cytoplasm. Thus, she was diagnosed to have primary hyperparathyroidism (PHPT)-induced hypercalcemia causing acute pancreatitis.

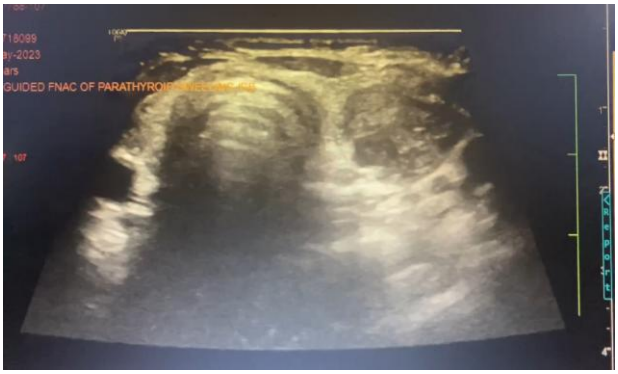

Figure 1: USG neck showing hypoechoic oval lesion in which guided FNAC was performed.

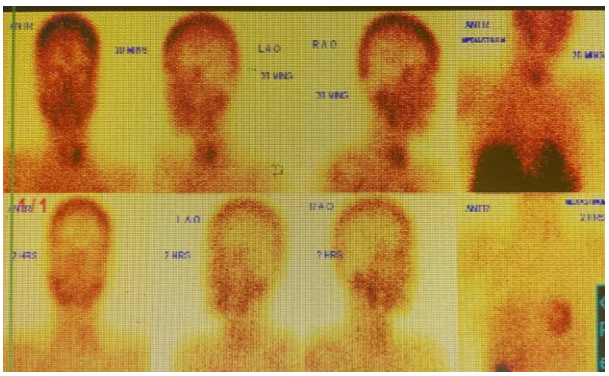

Figure 2: Tc-99m MIBI scan showing no abnormal tracer uptake in the neck.

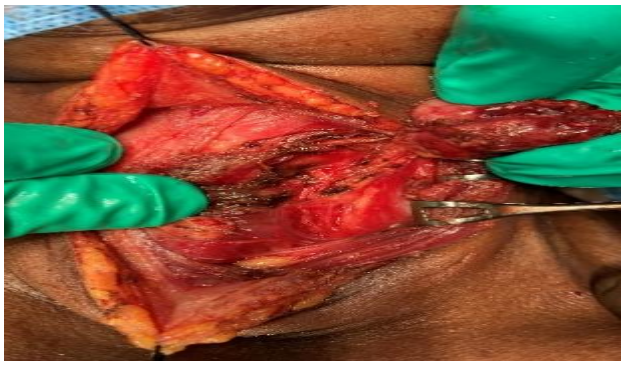

Figure 3: Intraoperative image showing the adenomatous gland prior to its removal.

After proper stabilization and thorough pre-operative work-up, she was planned for 4 gland exploration under

general anaesthesia. Intraoperatively, she was found to have 3×3 cm swelling arising from left superior parathyroid gland and the rest of the glands were normal. (Figure 3). Hence, we proceeded to do a left superior parathyroidectomy (Figure 4).

Her post-operative PTH level was 150 pg/ml, but however, calcium was on a decreasing trend. She had no clinical signs of hypocalcaemia (Figure 5). Patient was started on calcium and vitamin D3 supplements. Further monitoring revealed calcium values to be stable. Histopathology confirmed the diagnosis of parathyroid adenoma. Her post-operative period was otherwise uneventful, and she was thus discharged in a stable condition. Patient has been followed up and there were no further episodes of AP or other manifestations of hypercalcemia over the past 1.5 years.

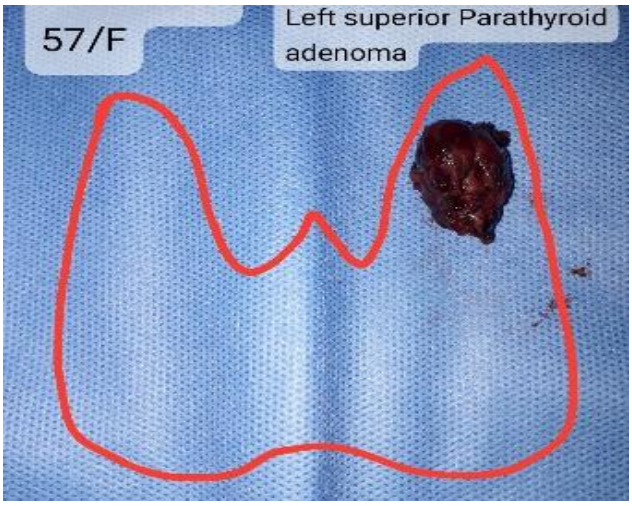

Figure 4: Post-operative specimen image showing left superior parathyroid adenoma.

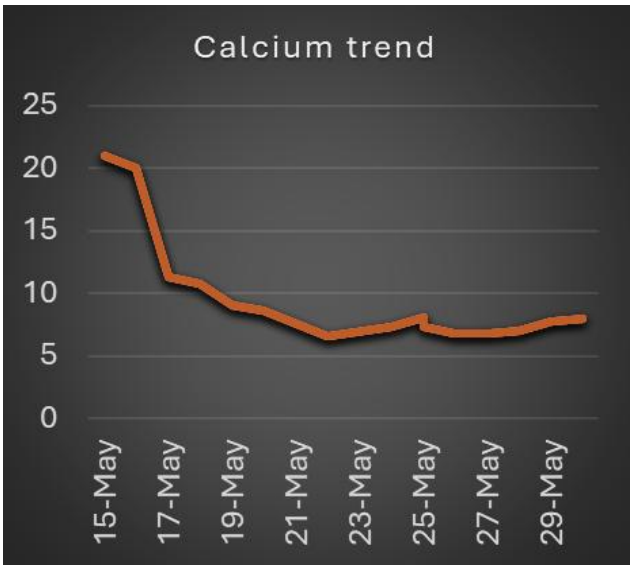

Figure 5: Chart showing the trend of calcium values since admission till discharge.

DISCUSSION

Acute pancreatitis is a commonly encountered life-threatening medical condition with worldwide incidence ranging from 5 to 80 per 100,000 population.[4] The most common causative factors are gall stone disease and alcoholism, however there are various uncommon causes. These constitute a small fraction of cases and include hypertriglyceridemia (1-4%), drug-induced pancreatitis (2-4.8%), trauma (1%), infections, and anatomical and genetic abnormalities which are often overlooked at the time of initial presentation. Hypercalcemia, along with its associated factors, is one of these rare causes (<1%) that warrants careful consideration.[5]

The first documented case of acute pancreatitis related to hyperparathyroidism was reported by Smith and Cooke in 1940.[6] In 1966, Pyrah et al attempted to summarize the conditions under which pancreatic disease occurred in association with PHPT.[7] Thereby, PHPT was accepted as an aetiology for AP.[8] A study by Bess et al reported that 64.7% of patients had a coexistent etiological factor for AP rendering the causal association contentious and prevalence of pancreatitis among patients with PHPT is between 1.5% and 13%.[9] Our case suggests a true positive causal association between PHPT and pancreatitis for there were no other concomitant risk factors for pancreatitis.

Parathyroid adenoma is the most frequent cause of PHPT with single gland adenoma being the most common pathology, double adenomas constitute 4% of cases, multi-glandular hyperplasia in 10-15% of cases, and rarely parathyroid carcinoma in less than 1% of cases.[10] Majority of patients with PHPT are asymptomatic and are incidentally diagnosed by routine biochemical evaluation showing elevated calcium and PTH values. Classical features of PHPT such as painful bones, kidney stones, and abdominal and psychic groans are encountered infrequently in clinical practice and patients tend to present with non-specific symptoms such as fatigue, sleep and memory disturbances, depression, musculoskeletal pain, polyuria, and polydipsia. PTH acts to increase serum calcium concentration and does so by acting on bones to cause demineralisation and resorption; leading to osteopenia, osteoporosis, and even cyst formation and fibrosis. PTH also increases calcium absorption from the gut and renal tubules, thereby resulting in hypercalcemia which is responsible for the various clinical manifestations.

Hypercalcemia induced acute pancreatitis is believed to be caused by inducing increased intrapancreatic conversion from trypsinogen to trypsin leading to damage of the pancreas.[11] Deposition of calcium in the pancreatic duct leading to pancreatic duct obstruction or underlying genetic variants are also implicated in the pathophysiological mechanisms of acute pancreatitis in PHPT.

After PHPT is diagnosed, imaging is required to locate the abnormally functioning gland. Initial step is USG neck in which these adenomas appear hypoechoic and are easily identified when they are larger than 1cm, but normal glands are too small to be detected.[12] A methoxyisobutylisonitrile (MIBI) scan is the imaging investigation of choice for localising abnormal parathyroid glands. MIBI contains sestamibi molecules that are not taken up by normal parathyroid glands; however, overactive parathyroid tissues rapidly uptake these molecules due to the high affinity of abnormal mitochondria to sestamibi, thereby enhancing the overall sensitivity of the MIBI scan, which ranges from 70% to 85%.[13] Various factors are conducive for MIBI sensitivity, which are gland size, multi-glandular disease, histopathological variants, diseased gland location, the severity of hypercalcemia, and vitamin D levels.[14] Our patient had a negative sestamibi scan thereby making the confirmation of diagnosis difficult. To navigate this, patient underwent FNAC and washout PTH (PTHw) measurement. Cytology is usually not used as a diagnostic tool in PA unlike in thyroid lesions and can only be used as an adjunct.[15]

Doppman et al reported using US-guided FNA of the suspicious lesion and parathyroid hormone washout measurement (PTHw) for the first time in 1983 and found it to have high sensitivity and specificity in identifying parathyroid tissue.[16] This helped us clench the diagnosis in our case and has potential to replace intra-operative PTH monitoring besides enabling surgeons make accurate preoperative evaluation.

Once the adenomatous gland is identified, curative treatment for PHPT is parathyroidectomy. It is of utmost importance to stabilise the patient and correct electrolytes imbalances prior to surgery. Studies have shown that adequate rehydration, calciuresis, and bisphosphonate therapy act as a bridge to definitive surgery.[17] Hypercalcemia needs to be corrected by intravenous hydration, forced calciuresis, administration of bisphosphonates to prevent further bone resorption and haemodialysis may be needed in refractory cases. Meanwhile, this must be orchestrated with the management of AP. Our patient required haemodialysis along with hydration and IV bisphosphonate administration in view of persistent hypercalcemia. Definitive management by surgical removal of the gland was done after stabilising the patient's general condition.

CONCLUSION

Whenever there is an uncertainty regarding the aetiology of AP, consider infrequent causes. Acute pancreatitis accompanied with hypercalcemia should raise the suspicion of PHPT. Presence of Sestamibi negative parathyroid lesions highlight the need for further workup in confirming the diagnosis in such cases. PTHw can be used in accurately identifying parathyroid tissue with high sensitivity and specificity. Curative treatment for PA induced PHPT is surgical excision.

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