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Parathyroid Adenoma inducing Hypercalcemia and Pancreatitis

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A fifteen-year-old female patient presented with acute pancreatitis as the first manifestation of primary hyperparathyroidism due to parathyroid adenoma. Initially the acute pancreatitis was treated conservatively and the high level of serum calcium was corrected partially and then subsequent parathyroidectomy was done. She is doing very well three months following the surgery and the pancreatitis resolved clinically and radiologically.

Although many studies showed the correlation between acute pancreatitis and hyperparathyroidism, the condition is still considered very uncommon. Surgical resection is the main treatment.

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Acute pancreatitis caused by hypercalcemia due to primary hyperparathyroidism is a very rare condition[1]. Primary hyperparathyroidism could be caused by solitary parathyroid adenoma (80%), multi glandular adenoma, parathyroid hyperplasia or carcinoma[2].

Patient may present with skeletal changes, nephrolithiasis, psychiatric manifestations and abdominal pain. Sometimes the patient remains asymptomatic and discovered during routine investigations.

Hypercalcemia is a rare cause of pancreatitis and the exact mechanism of inducing the disease is still controversial. However, it has been shown that hypercalcemia of any cause can lead to acute pancreatitis[3]. This is supported by the cause and effect relationship that parathyroidectomy may prevent the recurrence of the disease[4].

The aim of this report is to present a case of pancreatitis caused by parathyroid adenoma.

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THE CASE

A fifteen-year-old female presented to the emergency department complaining of severe epigastric abdominal pain with frequent attacks of vomiting for one day. The onset of the pain

was gradual for 2 weeks; she was diagnosed with pancreatitis due to iatrogenic use of calcium which was prescribed by another physician for osteopenia a month earlier. The patient had history of falling down two months before her admission to KHUH and had pelvic pain since then. No additional risk factors causing acute pancreatitis were found, such as alcohol consumption, hyperlipidemia and gallstones. No significant family history was revealed.

The patient looked ill and dehydrated; her vital signs were normal. Cardiovascular and chest examinations were unremarkable. Abdominal examination revealed tenderness and there was guarding at the epigastric area, normal bowel sounds and no palpable masses. No abnormality was detected in the musculoskeletal system.

Investigations showed WBCs 14000, lipase 1395u/l, serum calcium 3.5 mmol/L (2.21-2.55), bilirubin total 13 umol/l, bilirubin direct 7.5 (0-3.0), GGT 175 u/l, K 3.2 mmol/l (3.5- 5.1) and albumen 27 g/l (34-50).

US scan was reported as acute pancreatitis with phlegmonous collection in peripancreatic area, the gall bladder was normal, no gallstones could be seen.

CT scan showed edematous pancreas and large fluid collection (10x8 cm) inferior to the tail of the pancreas, see figures 1 and 2. The bone window study showed moderate osteopenia of the examined skeleton with coarse trabecular pattern and well-defined intramedullary lytic areas seen in the left iliac bone. The femoral neck bilaterally and the right pubic bone (superior and inferior) revealed old non-united fracture, see figure 3.

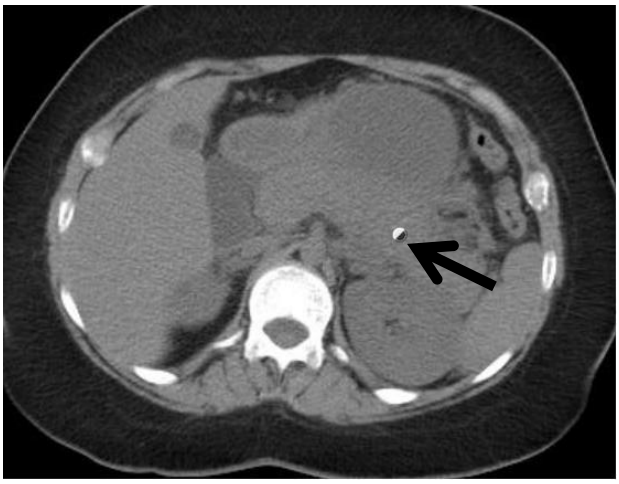

Figure 1: Fluid Collection around the Pancreas

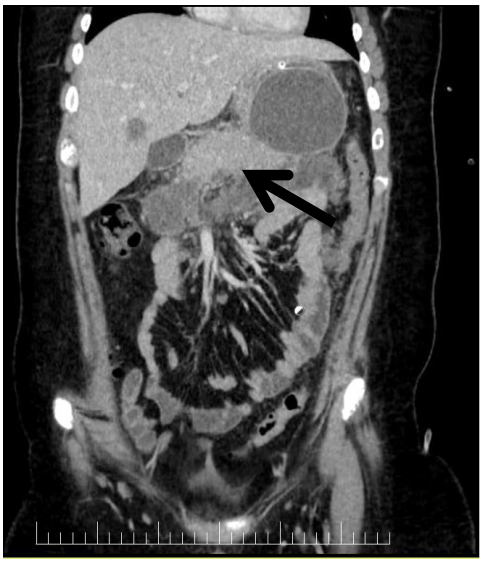

Figure 2: Edematous Pancreas

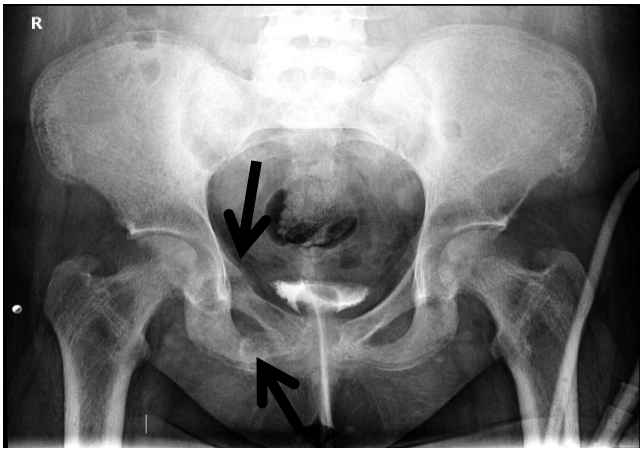

Figure 3: Non-United Fracture of Right Superior and Inferior Pubic Rami

The parathyroid hormone level was above 1900 pg/mL (normal 10-55 pg/mL), which confirmed our suspicion of hyperparathyroidism.

US of the neck showed low echogenic mass of 3.9 x1.3 cm behind the upper pole of the thyroid lobe on the left, probably parathyroid adenoma.

Hands X-ray showed well-defined lytic oval areas of reduced bone density in the right and left proximal phalanx of middle finger, metacarpal bone of right middle and left index fingers as well as the distal left radius. The picture is suggestive of hyperparathyroidism with brown tumors, see figure 4.

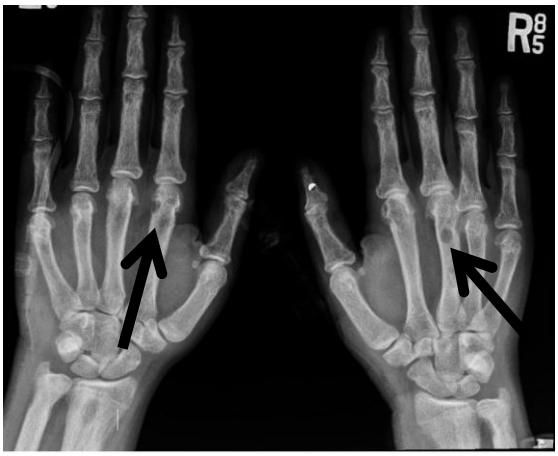

Figure 4: Brown Tumors Indicated by the Arrows

CT of the neck showed 3.5 cm parathyroid adenoma on the left side with bony changes (erosion of the clavicle) and brown tumors, see figures 5 and 6.

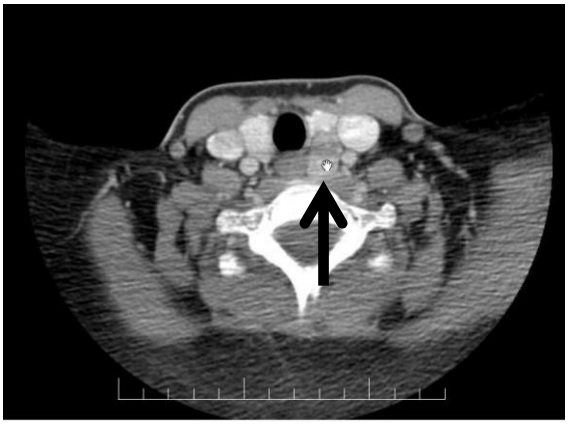

Figure 5: Parathyroid Adenoma

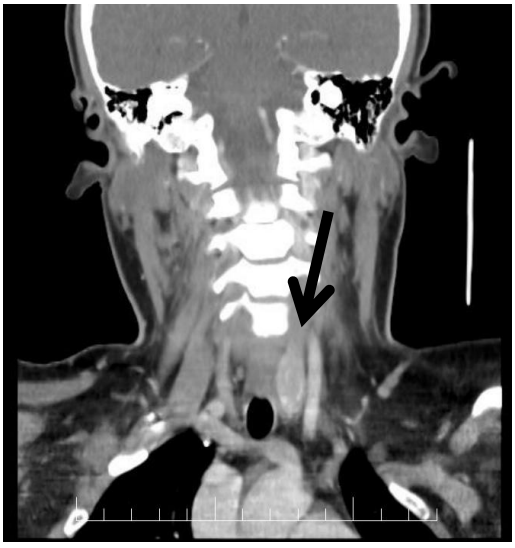

Figure 6: Parathyroid Adenoma

Treatment of hypercalcemia was induced with Lasix infusion initially then 20 mg intravenous Lasix 12 hourly. Gastrin level was normal; therefore, multiple endocrine neoplasia syndrome was ruled out.

After monitoring the serum calcium and correction of the electrolytes imbalance, the patient acute pancreatitis symptoms improved gradually. Serum calcium level dropped to around 3 mmol. Excision of 3.5 cm parathyroid adenoma from the left side was completed; the right side and lower part of the neck were explored to exclude other abnormal parathyroid glands, see figure 7.

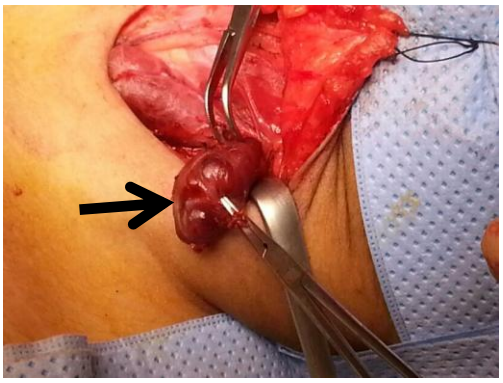

Figure 7: Parathyroid Adenoma

Parathyroid hormone and serum calcium level became normal postoperatively. Aggressive calcium replacement (up to 10 g intravenously) was induced. She was discharged from the hospital nine days postoperatively in good condition and was followed up in the clinic monthly.

Abdominal US scan a month postoperatively revealed no fluid collection around the pancreatitis. Hands X-ray showed improvement of the osteopenia.

DISCUSSION

Hypercalcemia due to hyperparathyroidism is generally a rare cause of acute pancreatitis. It was first mentioned in the literature in 1947 by Martin et al and later by others[5,6]. The Mayo Clinic experience between 1950 and 1975 found that out of 1,153 patients with primary hyperparathyroidism, only 17 (1.5%) had coexisting pancreatitis[7]. In the series of 51 cases of primary hyperparathyroidism by Muthukrushnan et al, four patients had pancreatic calcification and past history suggestive of acute pancreatitis[8]. Several studies had shown the relation between primary hyperparathyroidism induced hypercalcemia and pancreatitis [9]. The prevalence of acute pancreatitis in patients with primary hyperparathyroidism was estimated between 1.5% and 7%[1]. All patients with a past history of pancreatitis had suffered two or more attacks[10]. Our patient was diagnosed after the second attack of pancreatitis.

Acute pancreatitis is usually associated with hypocalcemia and this decrease in serum calcium has significant prognostic factor according to Ranson criteria[11]. Elevated serum calcium

associated with pancreatitis should alert the physician to either hyperparathyroidism or malignancy[10,11].

Pancreatitis induced by hyperkalemia is not well-understood, but several explanation have postulated: hypercalcemia induced primary hyperparathyroidism leads to activation of zymogens, including trypsinogen to trypsin, which result in acinar cell damage, autodigestion of the pancreas and subsequent pancreatitis; hypercalcemia could lead to modification of pancreatic secretion, which may lead to protein plug formation resulting in ductal obstruction and subsequent attacks of acute or chronic pancreatitis[6,12,13]. Genetic risk factors may predispose patients with primary hyperparathyroidism to acute pancreatitis[14].

CONCLUSION

A 15-year–old girl presented with rare case of acute pancreatitis due to hyperparathyroidism caused by parathyroid adenoma. The patient underwent parathyroidectomy followed by conservative management of pancreatitis, which resolved completely. In addition, the case is unique because of the bony pathological changes and the young age of the patient.

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