DUAL ETIOLOGY FOR ACUTE PANCREATITIS- GALL STONES AND PARATHYROID ADENOMA DETECTED IN A PATIENT WHO PRESENTED WITH ACUTE PANCREATITIS

JIFFY RASAK VA VKABDULRASAK
Department of Medical Gastroenterology,
CHRISTIAN MEDICAL COLLEGE

Abstract:
Acute pancreatitis refers to an acute inflammation of the pancreas. Worldwide, there are different known causes of acute pancreatitis among which alcohol and gall stones are the commonest. Hypercalcemia is considered a rare metabolic cause of acute pancreatitis. Here we report a patient who presented with acute pancreatitis where evaluation revealed dual etiology - gall stones and hypercalcemia secondary to parathyroid adenoma.

Keyword: Acute pancreatitis, gall stones, parathyroid adenoma

Introduction:
Acute pancreatitis is characterized by acute inflammation of the pancreas. The causes of the acute pancreatitis are varied across different patient populations. In the West, gall stones and alcohol accounts for 60 - 80% of the cases of acute pancreatitis. Certain metabolic conditions like hypercalcemia and hypertriglyceridemia are also known to cause acute pancreatitis. One of the earliest description of acute pancreatitis caused by hyperparathyroidism was given by Smith and Cooke in 1940. Since then, there have been multiple case reports and case series of hyperparathyroidism presenting as acute pancreatitis. International Association of Pancreatology (IAP) and American Pancreatology Association (APA) guidelines recommend both transabdominal ultrasonography and serum calcium levels among the initial tests for etiology work up in a patient presenting with acute pancreatitis. We report a case in which we found both gall stones and parathyroid adenoma in a patient who presented with the first episode of acute pancreatitis.

Case report:
Our patient was a 45 year old housewife from Jharkhand admitted with history of severe epigastric pain which was radiating to the back. The pain was continuous and was associated with recurrent episodes of vomiting. She was admitted in her native town for the same complaints for which she was given
analgesics and intravenous fluids. The pain decreased significantly with the above measures. When she presented to our institute on the fourth day after the onset of pain, she had mild pain persisting and her vomiting had subsided. She did not have associated fever, breathlessness or decreased urine output. She also did not give history of similar complaints in the past. There was no past history of biliary pain, abdominal surgery, abdominal trauma or alcohol intake. She did not have family history of any pancreatic disease. She was not on any medications prior to the onset of this illness.

Her general and systemic examination was normal except for mild epigastric tenderness. Her initial evaluation revealed an elevated amylase, lipase (376 & 424 U/L respectively). Abdominal x rays ruled out any bowel perforation. Her initial liver function tests was normal except for an elevated alkaline phosphatase.(Table 1). However the GGT was normal(17U/L) Table 1

Her initial transabdominal ultrasound revealed a bulky and edematous pancreas. There were no peripancreatic collections. The gall bladder showed multiple calculi and the GB wall was thickened.(figure 1). Cholecdocholithiasis was absent. She improved with conservative management and did not develop any local or systemic complication. Once her general condition improved and the pain subsided, she also underwent an endoscopic ultrasound to confirm absence of cholecdocholithiasis. Her initial blood chemistries revealed hypercalcemia and hypophosphatemia(11.7 mg/dl and 2.4 mg/dl respectively). Repeat values showed that these abnormalities were persistant. Serum parathyroid hormone level was 159.8 pg/ml (Normal: 8-74 pg/ ml). She did not have any renal failure. Ultrasound scan of her neck revealed lesions suspicious of adenoma in her left and right inferior parathyroid glands(figur 2). Parathyroid scintigraphy and SPECT-CT confirmed the presence of a left inferior parathyroid adenoma (figure 3). Her serum prolactin levels and fasting gastrin levels were normal. MRI brain and CT abdomen ruled out pituitary and pancreatic tumours which are common associations of parathyroid adenoma in MEN syndrome. Patient underwent a laproscopic cholecystectomy for her gall stones. She is currently planned for resection of the parathyroid adenoma by the endocrine surgery team.

Figure 1- Gall bladder calculi

Figure 2- left and right inferior parathyroid glands on USG
Discussion:
The exact mechanism of hypercalcemia mediated pancreatic injury is a matter of conjecture. It is well known that calcium is an important second messenger in the acinar cell. The level of calcium in the cytoplasm of acinar cells is tightly regulated. A sustained increase in acinar cell calcium might lead to unopposed activation of pancreatic zymogens including trypsinogen. Hypercalcemia might predispose the pancreatic acinar cell to sustained high calcium levels leading to zymogen activation and acinar cell injury. Most case reports have reported that definitive treatment of hyperparathyroidism by resection of adenomas led to cessation of pancreatitis attacks. However some authors believe that hyperparathyroidism is an innocent bystander that is detected incidentally because of the screening for hypercalcemia in hospitalized patients. In a series of 1153 patients with primary hyperparathyroidism, Bess et al has reported that pancreatitis was reported in only 1.5% of the patients with primary hyperparathyroidism. However later studies reported higher rates of pancreatitis in patients with primary hyperparathyroidism. A cumulative review by Bai et al proved that acute pancreatitis occurs at a higher frequency in patients with primary hyperparathyroidism compared to other hospitalized patients. A case series from northern India has reported that acute pancreatitis was the initial presentation in 6.8% of patients with hyperparathyroidism. A previous study from our institution had reported that in patients with primary hyperparathyroidism, the incidence of pancreatitis was higher among patients with higher levels of serum calcium thus suggesting a causal association. Thus the available literature suggests that hyperparathyroidism is a risk factor for acute pancreatitis.

There are multiple reports of increased incidence of gall stones in patients with hyperparathyroidism. Animal studies have shown that hyperparathyroidism induces gall stones by means of bile stasis and increased in the ionized calcium in bile. The current case report underlines the fact that we should complete the etiologic workup for acute pancreatitis even if an obvious cause is detected early on in the evaluation process. In case there is more than one cause, all of these need to be addressed from the management point of view considering the morbidity and mortality associated with a severe attack of acute pancreatitis.

References:
2 Smith FB, Cooke RT. Acute fatal hyperparathyroidism. Lancet.1940; ii: 560–61
4 IAP/APA evidence-based guidelines for the management of acute Pancreatitis. Panreatology 13 (2013) e1-e15


