

A Case of Chronic Abdominal Pain

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CASE REPORT

A 26-year-old non-smoker, non-alcoholic, male was admitted for the evaluation of chronic abdominal pain which had been persistent for more than six months. He took treatment from different centres, but the pain-relief was only partial. He gave history of passing frequent, foul-smelling stools with Loss of appetite and weight. He had polyuria, there was no polyphagia or polydipsia. He never had cough or fever or vomiting or bleeding per rectum. There was no past or family history of diabetes mellitus or pulmonary tuberculosis.

General and clinical examination of cardiovascular system, respiratory system, abdomen, and central nervous system were unremarkable. Complete haemogram, renal and liver function, lipid and thyroid profile, urine and stool examination, electrolytes, calcium and phosphate were normal. Urine for albumin, ketone and Porphyrins were negative. HBsAg, Anti-HCV, Anti-HIV were also negative. ECG and chest X-ray were within normal limits. Fasting blood sugar was 278mg%, post-prandial blood sugar was 486 mg%, and HbA1C was 10.1%. Serum amylase and lipase were slightly elevated which returned to normal after treatment.

Plain X-ray abdomen supine antero-posterior (AP) view showed presence of multiple calcific foci along the location of pancreas crossing the midline suggestive of pancreatic calcification [Table/Fig-1]. Plain and contrast Computed Tomography (CT) scan of abdomen confirmed the presence of coarse pancreatic parenchymal calcifications with intraductal calculi and also showed diffuse pancreatic parenchymal atrophy [Table/Fig-2A and 2B].

The term pancreatic calcification refers to calcareous deposits both in the duct and parenchyma of pancreas [1]. Many of them were asymptomatic and they were incidentally detected. Calcification is seen on X-rays in about 30-50% of patients with chronic pancreatitis

[2] and more than 75% with tropical pancreatitis. Pancreatic calcification is diagnostic of chronic pancreatitis, even in the absence of the clinical signs and symptoms. The degree of calcification in pancreas correlates with the course of the disease [3]. Endocrine abnormality, particularly diabetes is commonly associated with pancreatic calcification.

DISCUSSION

Zuidema (1959) from Indonesia first reported a series of cases of pancreatic calcification with diabetes mellitus. In India, Kerala has got the highest incidence of tropical pancreatitis in the World. Gee Varghese reported a large series of Tropical Calcific Pancreatitis (TCP), typically those patients had pain in childhood, diabetes in adulthood and death in prime time of life. TCP is a form of chronic calcific non alcoholic pancreatitis which is progressive and a highly variable disease with a triad of symptoms like abdominal pain, diabetes and steatorrhea [4], which is seen exclusively in developing countries.

TCP is characterized by young age of onset, extensive pancreatic calcifications, formation of large calculi in the pancreatic duct and high risk of pancreatic cancer [5]. When diabetes is associated with TCP, it is called Fibrocalculous Pancreatic Diabetes (FCPD) [4]. The diabetes which occurs in FCPD is severe, insulin dependent, ketosis resistant and more prone for complications like pancreatic malignancy.

In 1969, Olurin et al., reported a series of 45 cases of pancreatic calcification which concludes that diabetes mellitus was the major and common complication [1]. The exact etiopathogenesis is still unclear, the hypothesis proposed are malnutrition, toxins, cassava consumption (cyanogen toxicity), familial and genetic factors (SPINK1-serine protease inhibitor kazal Type 1 gene), oxidative



[Table/Fig-1]: Plain X-ray abdomen supine antero-posterior view showing the presence of multiple calcific foci along the location of pancreas crossing the midline (white arrows) suggestive of pancreatic calcification. **[Table/Fig-2A]:** Axial Plain CT abdomen showing coarse pancreatic parenchymal calcifications (white arrows) with diffuse pancreatic atrophy **[Table/Fig-2B]:** Coronal contrast CT abdomen maximum intensity projection image showing diffuse coarse pancreatic parenchymal calcification (white arrows)

stress, anti oxidant depletion, free radical injury and trace element deficiency [4]. There is irreversible destruction and fibrosis of both exocrine and endocrine pancreas leading to ductal strictures with dilatation, stasis resulting in atrophy of pancreas, calcification and calculi formation [5].

The pathological features of TCP include a shrunken, fibrotic pancreas with dilated ducts and ductules with intraductal calculi ranging in size from small sand particles to 4.5cm. Extensive intra- and interlobular fibrosis with periductal fibrosis can be seen on microscopy. Inflammatory cell infiltrate is sparse in advanced stages [5].

Pancreatic calcifications are easily seen on plain radiographs. Imaging findings in ultrasound, CT and magnetic resonance imaging (MRI) include dilatation of main pancreatic duct and its side branches, ductal irregularities with strictures, atrophy of the pancreas, and pancreatic calcification. Although, endoscopic Retrograde Cholangiopancreatography (ERCP) is considered as a gold standard for diagnosing pancreatic disease, it is an invasive technique and has several drawbacks, including failure to cannulate minor papilla, a high rate of complications, such as ERCP-induced pancreatitis, radiation, and use of iodinated contrast medium. However, Magnetic Resonance Cholangiopancreatography (MRCP) together with MRI is a non-invasive technique without radiation and can be done together with MRI in a single study, which can delineate the parenchymal morphology in detail [6].

The management of TCP includes treatment of abdominal pain, steatorrhea in addition to treatment of diabetes mellitus with analgesics and pancreatic enzyme supplementation. Majority of the patient's pain responds well to medical treatment. Initially, oral hypoglycemic drugs can be tried, but insulin therapy will be required later. Surgical management includes decompression of the duct, drainage procedures and subtotal or partial pancreatectomy sometimes. Modified Puestow's operation - Lateral pancreateojejunostomy was the common surgery performed for TCP. Endoscopic treatment including stenting, sphincterotomy, and

Extracorporeal Shockwave Lithotripsy (ESWL) and irrigation of the duct are being tried in some centres.

Microvascular complications can occur similar to Type 2 diabetes mellitus, but macrovascular complications are rare. Frequent episodes of hypoglycemia is common in TCP [7]. Early diagnosis and treatment of both exocrine and endocrine dysfunction of pancreas will make the prognosis better .

Since the patient had abdominal pain, steatorrhea and diabetes mellitus, a diagnosis of Tropical Chronic Pancreatitis (TCP) was made and he was started on strict diabetic diet and short acting insulin t.i.d, later changed to pre- mixed insulin b.i.d. After treatment with supportive drugs like analgesics, H2 blockers, pancreatic enzyme supplementation and good glycemic control, abdominal pain was relieved. Liberal calories and high protein intake allowed the lean patient to gain weight. He is on regular follow-up in our diabetic clinic.

CONCLUSION

Management of patients with TCP is really a challenge to the physician. This report highlights the importance of simple investigation like plain abdomen X-ray which gave a clue to the diagnosis of chronic abdominal pain.

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