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A Rare Case Of Atrophy Of Pancreas In A Patient Of Chronic Pancreatitis

Dr.Insha Wali
Pg Resident, Department Of General Surgery, Sharda School Of Medical Sciences & Research, Greater Noida-201310, Uttar Pradesh

Dr.Ashok Kr Gupta
Professor, Department Of General Surgery, Sharda School Of Medical Sciences & Research, Greater Noida-210310, Uttar Pradesh

Dr.Vikram Singh Chauhan
Prof & Head, Department Of General Surgery, Sharda School Of Medical Sciences & Research, Greater Noida 201310, Uttar Pradesh

Dr.Dewat Ram Nakipuria
Associate Professor, Department Of General Surgery, Sharda School Of Medical Sciences & Research, Greater Noida-201310, Uttar Pradesh

Abstract
Background*: Chronic pancreatitis (CP) is a progressive inflammatory condition of the pancreas characterized by prolonged inflammation leading to irreversible damage, resulting in both endocrine and exocrine pancreatic insufficiency (EPI). This report presents the case of a 47-year-old female with a history of recurrent abdominal pain, vomiting, and weight loss. Diagnostic imaging revealed findings consistent with chronic calcific pancreatitis. Despite medical management, including pancreatic enzyme replacement therapy (PERT), the patient experienced recurrent episodes of acute exacerbation. Subsequent imaging revealed extensive pancreatic autolysis and the presence of a large stone within the pancreatic duct. The management of CP remains challenging, often requiring a multimodal approach involving pain management, enzyme replacement therapy, and, in refractory cases, surgical intervention. Surgical options include decompression procedures and resection surgeries, each with varying degrees of morbidity and long-term outcomes. In conclusion, CP represents a complex disease continuum necessitating a personalized treatment approach, with surgery playing a crucial role in selected cases refractory to medical and endoscopic therapies.*

Keywords*: Atrophy of Pancreas, Chronic Pancreatitis (CP), Exocrine Pancreatic Insufficiency (EPI), Pancreatic Enzyme Replacement Therapy (PERT)*

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I. Introduction:

Chronic pancreatitis (CP) is defined as prolonged inflammation of pancreas associated with irreversible destruction of exocrine parenchyma and in late stages destruction of endocrine parenchyma. This leads to exocrine pancreatic insufficiency or EPI, unrelenting abdominal pain, malnutrition. It is a fibrosing disorder characterized by stricture or stones.[1]

Although acute pancreatitis (AP) and chronic pancreatitis were believed to be two distinct entities,[12] lot of data supports that AP, recurrent AP (RAP) and **CP represent a disease continuum.**[13][14] The two-hit hypothesis model can also be used to outline the pathogenesis of CP in the setting of pre-AP, RAP with risk factors. An initial hit (episode of AP) activates the immune system, leading to either recovery or progression to chronic pancreatitis.[15] Overall, approximately 20% patients with AP have a recurrence rate of 36% of RAP which ultimately develop CP. [13}

The Toxic- metabolite theory indicates that ethanol injures pancreatic stellate cells and causes their proliferation resulting in extracellular matrix remodelling leading to pancreatic fibrosis and acinar cell loss.

The most common cause of pancreatitis is gall stone and alcohol abuse. Gall stone induced pancreatitis continues to have a mortality rate of approximately 10 percent. Early diagnosis of biliary pancreatitis remains

problematic, but clinical and biochemical factors along with imaging play a major role in establishing diagnosis. Bile duct strictures are common complication in patients with advanced chronic pancreatitis and have variable presentation ranging from an incidental finding to overt jaundice to cholangitis.

II. Case Report

We present a case of a **47 year old female** who came to the OPD of our hospital of Sharda School of Medical Sciences & Research with the chief complaint of pain in the upper abdomen for the last 1 year. The pain was sudden in onset, severe in intensity, gradually progressive, radiating to the back, relieved by intravenous medications. She gave a history of on-off vomiting since 1 year. Vomitus contained food particles which was non-billious. She also gave a history of high grade fever sine 4 months which was insidious in onset, gradually progressive, intermittent, no diurnal variation, associated with chills, relieved on taking per oral medication. She also complained of anorexia since 4 months. Weight loss history of 7 to 8 kilograms in 4 months.Surgical history included tubectomy done 22 years back and laparoscopic cholecystectomy done 15 years back.

She had pulmonary tuberculosis 30 years back for which she had taken 6 months of treatment. She also has bronchial asthma for 10 years, type 2 diabetes mellitus for 8 years and hypertension for 4 years for which she was on regular medications.

One year back, due to repeated episodes of severe pain, mild diarrhoea and vomiting the patient visited a nearby hospital. Ultrasonography was suggestive of dilated CBD(13mm), atrophic pancreas with parenchymal calcification and dilated MPD (6.8 mm) suggestive of chronic calcific pancreatitis. She was started on tablet panlipase 25,000 U.

6 months later following the diagnosis she was once again admitted for acute on chronic pancreatitis. The abdomen was not distended, tender at right hypochondrium and epigastrium. This time the ultrasonographical finding was suggestive of dilated CBD (14mm), dilated MPD (4.7mm) with ? calculus at the head of pancreas, pancreatic atrophy. MRCP was done, which showed chronic calcific pancreatitis, dilatated CBD (10mm), no calculus in CBD, with gradual smooth tapered narrowing of distal end, dilatated MPD. Her haemoglobin was 8.3 mg/dl, TLC was 9000. The patient had no signs of jaundice. LFT was normal with slight raise in ALP levels, but serum lipase levels were <10. CA 19-9 was also within normal limits. Her random blood sugar levels were less <200mg/dl till the date of discharge.

In the present admission, CECT whole abdomen was suggestive of **completely autolysed pancreas**, which even in the background of chronic pancreatitis was a rare finding. MPD was dilatated (9.8 mm) and showed a stone at the head region of size 8.4 x 7.7 mm. The patient was conservatively managed by giving IV fluids, IV antibiotics, IV analgesics, tablet Panlipase 25,000 U thrice daily, tablet Tryptomer 25mg once at night. Diabetes was managed by giving injection regular insulin according to sliding scale thrice daily. Input-output charting was being done, RBS-charting, along with vital monitoring and abdominal girth charting. A week later the patient was symptomatically better. Patient was planned for ESWl with MPD stenting.

III. Discussion

Risk factors associated with CP summarized namely toxic/metabolic (alcohol, smoking, hypercalcemia, hyperlipidaemia, toxins), genetic/heredity (SPINK 1 gene, PRSS 1 gene), recurrent acute pancreatitis, familial aggregation of cases, autoimmune, obstructive and idiopathic.

CP has the highest prevalence rate in India 125/100,000 population.[2][3] Population studies indicate that men are more commonly affected than females (6.7 vs 3.2 per 100,000 population). Mean age of patients with CP has been reported to be in late twenties and early thirties.[40[5]

Typically, the patient presents with upper abdominal pain radiating to the back, endocrine and exocrine pancreatic insufficiencies due to pancreatic atrophy. The dysfunction of endocrine causes diabetes mellitus.

The exocrine pancreatic insufficiency (EPI) is influenced by the amount of pancreatic remanant. Pancreatic enzymes include proteases (trypsin, chymotrypsin, elastases), pancreatic lipase, pancreatic amylase, deficiency of which will cause malabsorption, steatorrhea.it is defined as the presence of 7g of stool fat/day. [6] The problem with CP is that any clinical manifestation of exocrine pancreas insufficiency occurs late in the course of the disease, after approximately 90% of exocrine pancreas has been destroyed and secretion of pancreatic enzymes is <10% of the normal. [7]

The **Management of CP is challenging and requires a personalized approach.** Most patients remain symptomatic despite therapy. There are multifactorial causes of pain in CP with inflammatory and neuropathic components.[16] **Pain** can be caused due to stone or stricture. Another cause of pain in CP can be due to structural changes of the intra-pancreatic nerves and functional changes in both pancreatic nociceptors and spinal and central neurons involved in pain signalling and perception. Third cause of pain can be due to the complications of CP which might include autolysis, pseudocyst, bile duct or gastric outlet obstruction, duodenal obstruction, or due to malignant transformation. Thus, therapy directed at pancreas for pain management is usually ineffective.[17] Adjuvant pain medications such as tricyclic anti-depressants, gabapentin, pregabalin and SSRIs may also be used

in combination with opioids.[18] Large trials of antioxidants have reached different conclusion. Little evidence supports the use of octreotide.

Alcohol and smoking abstinence is mandatory. In conjunction, **Pancreatic enzyme replacement therapy (PERT)** is given due to autolysis of pancreas in CP given as soon as EPI is diagnosed independent to the degree and presence of steatorrhea.[8]It is given to avoid malnutrition, vitamin deficiencies and to improve the nutritional status.[8] Severe EPI tend to develop between 5-10 years following an initial diagnosis of CP. Additionally, a baseline evaluation of nutritional status is appropriate when patients begin PERT. The preparation is called Pancrelipase or Pancreatin. Very few side effects have been observed while using PERT, which include transient nausea, bloating and hypersensitivity. Only one serious adverse event has been reported in January 1994, Smyth et al described 5 children with cystic fibrosis developed colonic obstruction after using high levels of PERT due to fibrosing colonopathy (FC).[9][10][11]

Pancreaticogenic diabetes or type 3c diabetes mellitus (DM) is a secondary complication in patients with pancreatic disorders such as AP, CP as well as pancreatic cancer. About >50% of the patients with pancreatic disorder develop DM, due to loss of islet cells and thus counter regulatory hormones are also lost.

The requirement for endoscopic/ resection/ drainage procedures varies from one case to another, and is tailor made for each individual. In case of pancreatic abscess and pseudo-cyst pigtail catheterization is the best form of treatment along with antibiotics. **Endoscopic** cystogastrostomy/ cystoduodenostomy is better preferred for drainage of pseudocyst than open cystogastrostomy/cystoduodenostomy as it is less invasive. Various techniques like endoscopic pigtail with a) dialation + stenting of MPD b) lithotripsy and stone extraction in pancreatolithiasis or c) bile duct stenting done for distal CBD stenosis, are taken into consideration after appropriate evaluation of the requirement of the patient.

Various **decompression procedures** are Duval's procedure, Puestow's procedure, Partington Rochelle procedure aka longitudinal pancreatico -jejunostomy (LPJ). Pustow's is the least morbid and preserves the most pancreatic tissue but only has 50% long-term pian relief.[20]

Various **resection surgeries** include, Berger's procedure or duodenum preserving pancreatic head resection (DPPHR), pancreatico-duodenectomy (Kausch-Whipple procedure), pylorus-preserving pancreatico-duodenectomy (Traverso-Longmire) and near or total pancreatectomy. Various trials have indicated the superiority of DPPHR over partial pancreatectoduodenectomy.[19] Total pancreatectomy has high post-operative morbidity rate (40-50%).[20]

Other surgeries are grouped under a heading called **hybrid surgery** namely Frey's procedure which includes some portion of resection of head of pancreas as well as the MPD is also opened. Another such procedure is the Hamburg procedure.Although, the morbidity and mortality of patients in CP is very high, nearly half of the patients will ultimately require surgery, although surgery may not lead to complete decline of symptoms in patient of CP.

IV. Conclusion:

Chronic pancreatitis (CP) poses a significant clinical challenge due to its progressive nature and debilitating symptoms. This report highlights the intricate interplay between inflammation, fibrosis, and functional impairment in the pancreas, resulting in both endocrine and exocrine dysfunction.

Atrophy of the endocrine parenchyma causes type 3c DM or pancreaticogenic DM, and atrophy of exocrine pancreas causes exocrine pancreatic insufficiency (EPI) In patients with CP, which causes malabsorption, steatorrhea and PERT is the mainstray of treatment along with pain management and antiglycemic drugs. Despite advancements in medical management, including enzyme replacement therapy and pain control, many patients continue to experience recurrent episodes of pain and metabolic disturbances. Surgical intervention, while offering promise in select cases, presents its own set of challenges and risk.

Conflicts Of Intrest

The authors declare no conflicts of interests

Disclosure And Funding

None

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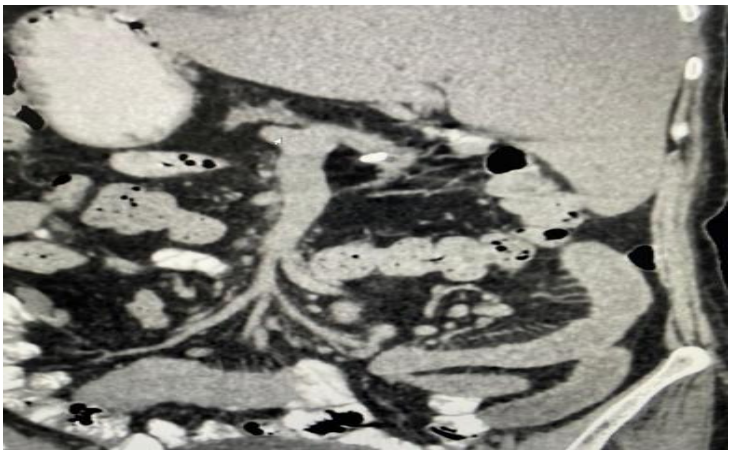
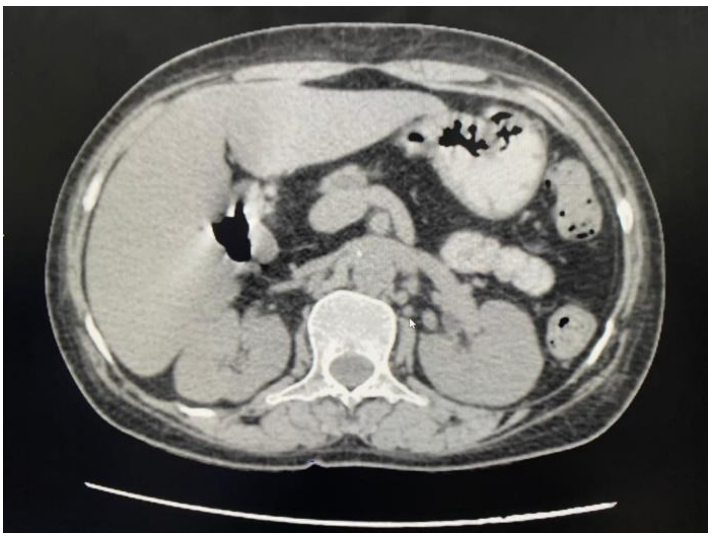


Pic 1 : Stone in Main Pancreatic Duct



Pic 2 : Atrophy of Pancreas