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RSSDI Diabetes Update 2016

Editor-in-Chief
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Chapter 3

A Comprehensive Look at Fibrocalculous Pancreatic Diabetes

Beatrice A M, Subhankar Chowdhury

INTRODUCTION

Fibrocalculous pancreatic diabetes (FCPD) has been classified by the World Health Organization (WHO) as a secondary cause of diabetes due to disease of the exocrine pancreas.[1] It is a condition in which, in addition to diabetes being present, there is also evidence of chronic pancreatitis of unknown origin with large intraductal pancreatic stones.[2] Patients frequently have a low body mass and a history of chronic abdominal pain and require insulin treatment, although, unlike in type 1 diabetes mellitus, they are not prone to ketosis. Fibrocalculous pancreatic diabetes is likely to be a multifactorial disease, with genetic and environmental components to both the diabetes and chronic pancreatitis.

THE DISEASE IN THE PAST

Tropical pancreatitis (TP) was first described by Zuidema[3] from Indonesia. Later, Geevarghese[4] described several patients from Kerala state in southwest India, and the disease was reported from many tropical countries in Africa and Asia. The etiology of TP remains unknown. Since poverty and malnutrition were common in the countries from where the disease had been reported, malnutrition was suspected as a major causative factor and hence the disease was even described as "nutritional pancreatitis". With reference to Indian subcontinent, earlier clinic-based studies had suggested that the prevalence of FCPD is higher in states like Kerala, Tamil Nadu, and Orissa compared to the rest of the country. The term "tropical", reflecting geographical distribution, became a part of the disease's name, and by conjuring images of poverty and malnutrition, led to a tendency among early workers to treat TP as "a disease of deprivation".

Diagnosis of diabetes is made in the majority of patients between the ages of 10 years and 40 years. The classical clinical description of TP was that of a child, adolescent, or young adult, who presented with recurrent upper abdominal pain, often with diabetes mellitus, and had signs of malnutrition, nutrient deficiencies, cyanotic hue of the lips, bilateral enlargement of parotid glands, a pot belly and, sometimes, nutritional edema. Demonstration of high blood sugar level and of pancreatic calculi on plain abdominal X-ray confirmed the diagnosis. Up to 90% of patients required insulin, often in high doses, for control of diabetes. Episodes of hypoglycemia were common, whereas ketosis was uncommon.

THE CHANGING PARADIGM

Recent reports suggest a change in the clinical presentation that may be attributed to improved nutritional status. Recent studies have shown that while the majority of patients were lean, severe malnutrition was uncommon; many patients were of ideal body weight and an occasional patient even obese. Most of the patients were aged 10–30 years when the diagnosis is made. Owing to the longevity of the patients due to better pain management and diabetes control, both microvascular and macrovascular complications have been demonstrated in spite of FCPD being a secondary form of diabetes.

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Metabolic and Environmental Factors

Nutrition

Protein calorie malnutrition has long been suspected as a likely cause for tropical calcific pancreatitis because of the fact that the disease occurs predominantly in tropical countries where malnutrition is common and because of the reports from some of them including India, Uganda, and Nigeria reveal that 80–90% of the subjects with calcific pancreatitis come from poor socioeconomic strata.[5] Chronic protein under nutrition leads to structural as well as functional alterations in the pancreas. Whether malnutrition is a cause or effect of this disease is a question which is still unanswered.

Environmental Toxins

Clinicopathological studies suggested toxiconutritional factors, particularly dietary toxic factors in a background of low-protein high-starch diets and deficiencies of vitamins and minerals, in the etiology of this disease. Various toxic factors suspected included cyanoglycosides from tapioca (cassava), a staple food in these regions, ergot-like toxins from plants and indigenous medicines, serotonin from bananas, trace heavy metals like thorium and cerium from the soil, and undernutrition associated with dietary deficiencies of protein and amino acids, especially tryptophan, cysteine, and methionine, and minerals like iodine and magnesium.[4,6] However, there was no definite experimental proof available despite intense studies carried out by several groups of research workers from different countries during the last five decades. Theoretically, excessive cassava consumption could lead to TP in several ways. First, cassava is 87.5% carbohydrate with negligible (0.7%) protein content and essential amino acids (404 mg/100 g). Second, it contains cyanogenic glycosides, releasing cyanogens that are tissue toxins. Third, the process of detoxification of cyanogen utilizes and depletes the body of essential amino acids, namely, methionine and cysteine, which are essential for maintaining the normal structure and function of the pancreas. Though there is some geographical association between cassava consumption and tropical pancreatitis, this association is not consistent. This theory has also not found wide acceptance because of the following reasons: (i) cassava does not feature in the diet of many people who develop TCP; (ii) there was no difference in cassava consumption between patients with TCP and those without TCP; (iii) patients with TCP from Northern India do not consume cassava;[7] and (iv) long-term cassava consumption did not produce diabetes or pancreatitis in a rat model.[7]

Studies have revealed that the diet of patients with TP was poor in proteins, very poor in fats, and rich in carbohydrates.[8] Our study group demonstrated similar findings in the FCPD patients (unpublished data).

Genetic Factors

It has been shown that the initial event in patients with acute pancreatitis is premature and inappropriate intracellular activation of trypsinogen to trypsin in the presence of calcium. Appropriate calcium levels are critical for this activation. Activated trypsin further activates proenzymes, leading to a cascade effect. About 20% of prematurely activated trypsin is inhibited by a protein coded by SPINK1 gene (pancreatic secretory trypsin inhibitor or, secretory pancreatic inhibitor neutral Kajal type I), and the remaining trypsin is split by another protein coded by the PRSS1 or cationic trypsinogen gene. Mutations in the SPINK1 (a substitution of asparagine by serine at codon 34 in exon 3-N34S) or PRSS1 gene may remove the brake on prematurely activated trypsin and facilitate acinar cellular disruption and pancreatitis.[9] The other gene implicated in the causation is the cystic fibrosis transmembrane regulator on chromosome 7.[4]

Micronutrient Deficiency and Oxidative Stress

According to the oxidant stress theory of pancreatitis proposed by Braganza, the toxic effects of oxygen derived free radicals and lipoperoxidases can cause pancreatic damage. Exposure to xenobiotics, induction of detoxifying mixed function oxidases, and excess production of unmitigated metabolites and free radicals have been proposed to cause damage to cellular membranes. An imbalance between these toxic substances and antioxidants could lead to pancreatic injury.[10] Deficiency of antioxidants such as vitamins A, C, and E has been demonstrated in chronic pancreatitis. Malnutrition has also been shown to induce a state of defective ability to scavenge free radicals, which could enhance the susceptibility for organ damage. However, the free radical hypothesis is by no means proven and certainly merits further studies.

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CLINICAL FEATURES

The clinical picture of FCPD consists of the following four cardinal features:

1. Abdominal pain
2. Pancreatic calculi
3. Malabsorption leading to steatorrhea
4. Diabetes.

The criteria proposed by Mohan et al.[11] for the diagnosis of FCPD:

- Occurrence in a tropical country
- Diabetes by WHO study group criteria
- Evidence of chronic pancreatitis: pancreatic calculi on X-ray or at least three of the following:
  - Abnormal pancreatic morphology by sonography
  - Chronic abdominal pain since childhood
  - Steatorrhea
  - Abnormal pancreatic function test
- Absence of other causes of chronic pancreatitis, i.e., alcoholism, hepatobiliary disease, or primary hyperparathyroidism, etc.

Although these were the criteria laid down way back in 1988 and clinicians have resorted to using the American Diabetes Association definition for diagnosis of diabetes, this does not create any diagnostic ambiguity because patients almost always have grossly elevated blood sugars at presentation.

However, all features need not be present in every patient. Abdominal pain is usually the first symptom in TP. The general theory is that FCPD is the end point of TP, i.e., TP is the prediabetic stage of FCPD. The initial presentation is as recurrent severe episodes separated over long periods, followed later by frequent milder episodes or chronic persistent pain. As disease progresses, the episodes and severity of pain may diminish suggesting a "burn out of the disease". However, in many patients, the first sign of the disease maybe detection of calculi, diabetes, and rarely steatorrhea. Malabsorption typically presents as steatorrhea; however, overt steatorrhea maybe lacking as many patients restrict fat in diet. Our study group demonstrated that almost 25% patients have subclinical steatorrhea and all of them have severe exocrine insufficiency by the time they present with diabetes.

INVESTIGATIONS

If pancreatic calculi are present on plain abdominal radiography, the diagnosis is straightforward. However, calculi develop after several years of abdominal pain and about 10% of patients do not develop calculi. Hence, the need for other diagnostic markers. Unfortunately, there are still no sensitive and specific noninvasive blood or urine tests to diagnose early stages of chronic pancreatitis. The investigation for a suspected case of TCP without pancreatic calculi is as follows:

Tests of pancreatic structure (Figs 1 and 2)

- X-ray abdomen
- Ultrasonography
- Computed tomography
- Endoscopic retrograde cholangiopancreatography
- Endoscopic ultrasonography.

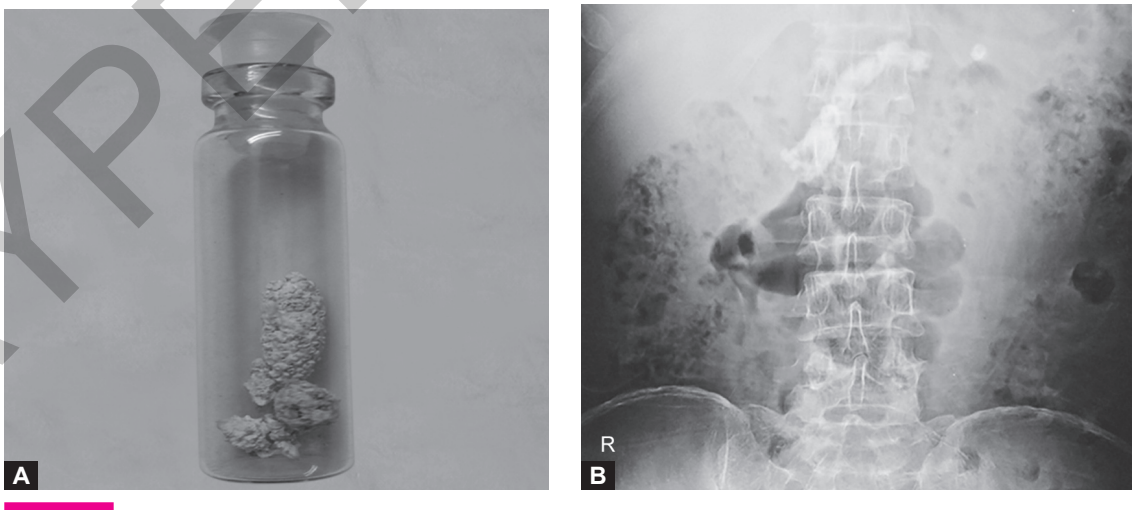

Figure 1 **A,** Pancreatic calculi removed during Frey's procedure from a patient with chronic calcific pancreatitis; **B,** X-ray abdomen showing intraductal pancreatic calculi.

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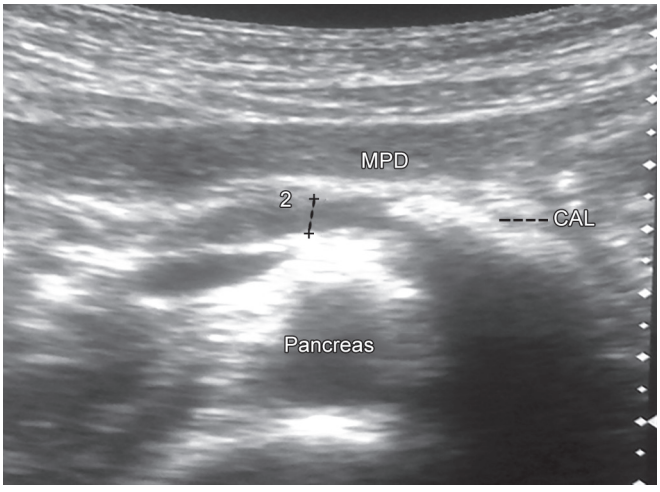

Figure 2 Ultrasound showing features of chronic calcific pancreatitis (main pancreatic duct dilatation and intraductal calculus).

Tests of pancreatic function

- Tests of exocrine pancreatic function
- Tests of endocrine pancreatic function.

Pancreatic function tests are very valuable in cases of chronic pancreatitis particularly when imaging modalities are inconclusive; unfortunately only invasive tests are sensitive in mild cases. They are also valuable to determine if pancreatic insufficiency is contributing to malabsorption, to determine the adequacy of pancreatic enzyme replacement therapy and to assess the extent of insufficiency before undertaking pancreatic resection as a part of surgical management. Though considered to be the gold standard, direct pancreatic function tests such as secretin-pancreozymin (cholecystokinin) test and Lundh meal test are time consuming, invasive, expensive, and hence, beyond the reach of nonspecialized centers. Stool fat estimation is cumbersome and difficult to set up and perform routinely. Most of the centers now perform simple tests such as fecal chymotrypsin. Fecal elastase is being more commonly used in many centers. Though this is very specific, it is thought to be sensitive only in advanced stages of the disease. But as demonstrated by the author's study group, the patients with FCPD already have severe exocrine insufficiency and fecal elastase could be a sensitive marker for pancreatic exocrine function in this setting.

MANAGEMENT

The management of FCPD is aimed at relieving pain and steatorrhea and controlling diabetes. For pain relief, initially nonopioid and later, opioid analgesics are used. The Asia-Pacific consensus report on chronic pancreatitis suggests pancreatic enzymes and nonopioid analgesics as the initial therapy for pain relief in patients with chronic pancreatitis. Use of antioxidants has also been suggested recently for pain relief in chronic pancreatitis. Surgical treatment remains the gold standard for the management of intractable pain and treatment of complications.

Usual indications for surgery are:

- Intractable pain not alleviated by medical therapy with calculi in pancreatic ductal system
- Head mass with suspicion of malignancy

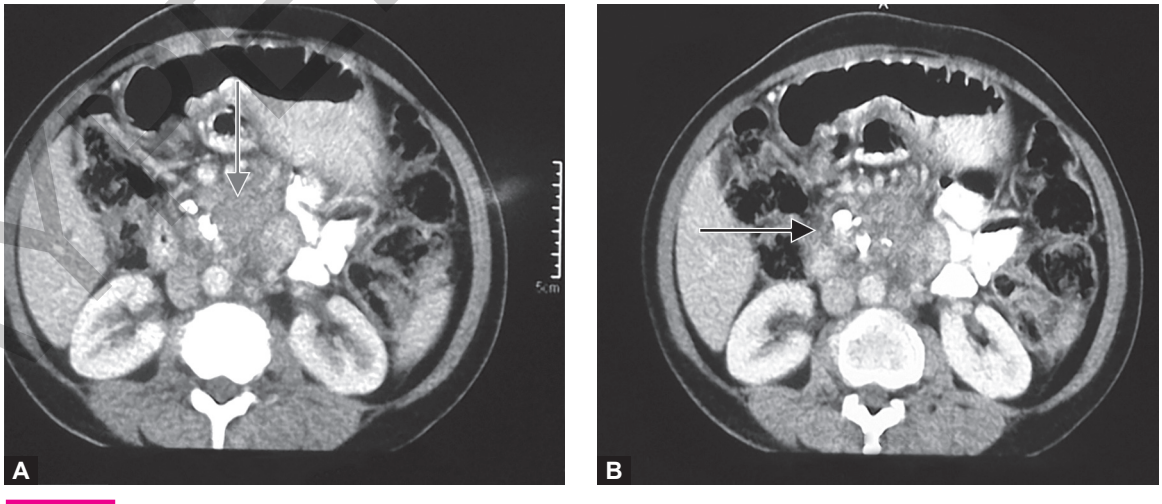

Figure 3 Computed tomography abdomen of a patient with FCPD showing pancreatic calcification (black arrow) and mass in the uncinate process (gray arrow) suspicious of pancreatic carcinoma.

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- Complications such as nonresolving biliary or duodenal obstruction, pseudocysts, pancreatic fistula, and left sided portal hypertension.

The basic principles of diet and exercise are the same as for the other types of diabetes except that a more liberal calorie and protein intake may be advised because of the associated undernutrition. Oral antidiabetic agents may be useful in cases with mild diabetes and relatively early in the course of the disease. Although an early study had demonstrated FCPD patients to be insulin resistant, the author's study group did not find any features of insulin resistance in these patients. Their total body fat was found to be low and the insulin resistance indices calculated by homeostasic model assessment for insulin resistance were low. On the other hand, this subset of diabetic patients in the author's clinic were found to be extremely insulin sensitive, and hence, the author advises careful and slow titration of insulin doses to avoid recurrent hypoglycemia.

COMPLICATIONS

It was formerly believed that FCPD being a secondary form of diabetes, specific diabetic complications were uncommon. Mohan et al.[12] had shown earlier that the sight-threatening forms of retinopathy, namely proliferative retinopathy and maculopathy, do develop in FCPD when followed for long periods. Neuropathy, nephropathy, and left ventricular dysfunction have also been reported in these patients.[13-15] In contrast, macrovascular complications were less common,[16] perhaps owing to the relative youth of the patients, their leanness and the low cholesterol levels.[15] However, ischemic heart disease,[17] peripheral vascular disease, and cerebrovascular accidents have occasionally been reported.[16,18-20] A study has shown the prevalence of abnormal cardiac autonomic function to be as high as 63.3% in FCPD patients, which warrants regular screening of these patient for autonomic dysfunction.[21] The author's study group has demonstrated both microvascular as well as macrovascular complications in these patients. The prevalence of peripheral neuropathy was 28% and that of cardiac autonomic neuropathy was 40%. The major causes of mortality were diabetic nephropathy and carcinoma pancreas. Change in the character of pain, development of jaundice, recent worsening of diabetes, and significant weight loss are red flag signs suggesting development of carcinoma.

CONCLUSION

Fibrocalculous pancreatic diabetes is not an uncommon cause of diabetes in the young in our country, though incidence is probably decreasing. Hence, young onset diabetes has to be evaluated with an ultrasonography of the abdomen to look for pancreatic calculi. Patients may not always be malnourished and diabetes related complications are not uncommon. The clinician has to actively follow-up the patient for development of carcinoma since these patients have a significantly higher risk of developing carcinoma of pancreas than the general population.

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