Painless Tropical Chronic Pancreatitis with Extensive Calcification

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Chronic pancreatitis (CP) is caused by a wide variety of causes such as alcohol, biliary diseases, trauma, infections, autoimmune diseases, and hereditary disorders. Patients with CP present with disabling abdominal pain, weight loss, exocrine insufficiency, and diabetes and are at risk for malignant transformation. Pancreatic calcification narrows down the diagnosis of CP. Uncommon causes of calcific pancreatitis include autoimmune pancreatitis, hereditary pancreatitis, tropical pancreatitis, groove pancreatitis, and pancreatitis associated with cystic fibrosis, Ascaris, and ectopic pancreatic tissue. Calcific pancreatitis in young diabetics of tropical countries could be either due to alcohol or tropical pancreatitis. We report a young man with weight loss and steatorrhea, but without abdominal pain whose evaluation led to a diagnosis of tropical CP and diabetes.

Keywords: Calcific pancreatitis, Chronic pancreatitis, Diabetes, Exocrine dysfunction, Tropical pancreatitis

INTRODUCTION

Chronic pancreatitis (CP) leads to the replacement of secretory parenchyma of the pancreas by fibrous tissue and is characterized by disabling pain, diabetes, and malnutrition.1 The etiology of CP includes toxins (alcohol, cigarette smoke), infections (HIV, mumps), drugs (valproate, azathioprine), metabolic abnormalities (chronic kidney disease, hypercalcemia), genetic abnormalities (CFTR and SPINK1 mutation), gallstones, and recurrent attacks of acute pancreatitis.1 In the Asia-Pacific region, the etiology of CP consists of alcohol, idiopathic, tropical, biliary and hereditary causes.2 CP in the young may arise due to hereditary, infective, toxic, or idiopathic reasons.

CP is the most common cause of pancreatic calcification. Pancreatic calcification also arises due to malignancies such as neuroendocrine tumors, mucinous neoplasms, and pancreatic adenocarcinoma.3 Uncommon causes of calcific pancreatitis include autoimmune pancreatitis, hereditary pancreatitis, tropical pancreatitis, groove pancreatitis, and pancreatitis associated with cystic fibrosis, Ascaris, and ectopic pancreatic tissue.4 In Asia and in the Indian subcontinent, tropical pancreatitis constitute a sizeable portion of patients with CP.5 Tropical pancreatitis has been reported in the south Indian states, Orissa, Sri Lanka, Indonesia, Malaysia, China and in African countries such as Uganda and Nigeria.2,6 This entity has also been known by other names such as chronic calcific pancreatitis and non-alcoholic tropical pancreatitis. Patients with tropical CP are generally young and have ketosis-resistant diabetes; large ductal calculi are observed, more so in the pancreatic head. When tropical CP is associated with frank diabetes, it is labeled as fibrocalculous pancreatic diabetes (FCPD) that is characterized by a triad of abdominal pain, malabsorption related steatorrhea, and diabetes.7 We report a patient with tropical CP with endocrine and exocrine insufficiency but without the characteristic abdominal pain of pancreatitis.

CASE REPORT

This 27-year-old farmer presented to the outpatient department of our hospital with 3 months’ symptoms of easy fatigability, lethargy, progressive weight loss (5 kg in 3 months), and frequent passage of bulky oily stools. He had been treated with iron and vitamin supplements by a private practitioner and was referred for evaluation of steatorrhea. He denied history of alcohol consumption, drug intake, recurrent pain abdomen and other gastrointestinal and osmotic symptoms. There was no family history of diabetes. Clinical examination was normal except for low body mass index (18.4 kg/m²). His investigations
included: Random blood sugar 363 mg%, normal renal and liver function tests, glycosuria without ketonuria and negative stool examination for ova and cysts. An abdominal radiograph revealed dense pancreatic calcification that was confirmed with a computed tomography of the abdomen (Figure 1a-d). His sugars were controlled with twice daily subcutaneous premixed insulin. Steatorrhea could not be proven because of lack of facilities in our institute. He was initiated on pancreatic enzyme supplementation (thrice daily with meals) and a fat-restricted diet, with which his symptoms subsided.

**DISCUSSION**

CP is caused by a large number of etiological factors. Young individuals are more likely to either have a hereditary, alcohol-related, cystic fibrosis-related, or pancreas divisum related pancreatitis. Gallstones, viruses, trauma and drugs cause CP without calcification. The differential diagnosis of pancreatic calcification is also broad and include alcohol, hereditary, infectious, autoimmune diseases, and malignant causes. The pattern of calcification, pancreatic atrophy, and dilated ducts help in narrowing the diagnosis even further. Diffuse parenchymal distribution of calcification, intraductal calcification, and parenchymal atrophy generally favors a diagnosis of CP. The calcification in alcohol-related pancreatitis is generally speckled and diffuse with irregular margins. Alcohol still remains the most common cause of pancreatic calcifications. Conversely, the calculi in tropical CP are large, discrete and are located in the pancreatic duct causing its dilatation, especially in the head of the pancreas. Tropical CP is a triad of abdominal pain, steatorrhea, and diabetes (spectrum of impaired glucose tolerance to frank diabetes). According to one study, tropical CP was the most common cause of CP causing diabetes in south India. This condition is male predominant, seen almost exclusively in non-alcoholics from lower socio-economic groups of tropical countries. India and Pakistan have the highest prevalence in Asia. Genetic susceptibility (SPINK 1 mutation) leading to loss of protease inhibition, malnutrition, and dietary toxins especially from cassava, are some of the etiological factors. In a study from Bangladesh, patients with tropical CP had selective preservation of alpha cell function in the pancreas along with CP. Steatorrhea is uncommon, being observed in ~20% and has been attributed to low-fat diet. They are not ketosis-prone due to preserved beta cell function and reduced glucagon reserve. Fecal fat estimation followed by calculation of the coefficient of fat absorption is the gold standard for steatorrhea, but cumbersome for the patient. Hence, less expensive tests such as fecal chymotrypsin, fecal elastase, and serum trypsin have been used instead of fecal fat estimation. As none of these tests were available, a therapeutic trial was given in our patient.

**CONCLUSION**

Diabetes in the young, when associated with pancreatic calcifications, should make one consider CP of either tropical, hereditary, or alcohol-related etiologies. Based on the nature, distribution of calcification and ductal dilatation tropical CP with diabetes (FCPD) was diagnosed. Unusual in this patient was absence of episodic attacks of abdominal pain and the immensely large calculi taking the shape of the entire pancreas. Even without chronic pain, imaging of the pancreas must be considered in young diabetics to rule out tropical CP related diabetes

**REFERENCES**


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