CASE REPORTS

Symptomatic chronic calcific pancreatitis in a patient with idiopathic ulcerative colitis and sclerosing cholangitis

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Although sclerosing cholangitis is well recognized to occur in patients with idiopathic inflammatory bowel disease, pancreatitis as a complication of ulcerative colitis is uncommon. We describe a patient who had idiopathic ulcerative colitis, primary sclerosing cholangitis and calcific pancreatitis with endocrine pancreatic deficiency, a rare combination. [Indian J Gastroenterol 2000;19:29-30]

Key words: Inflammatory bowel disease, pancreas, extraintestinal manifestation

Primary sclerosing cholangitis (PSC) is a well recognized hepatobiliary complication of idiopathic inflammatory bowel disease (IBD). Involvement of the pancreas in this disease is uncommon. Even when it occurs, it is usually mild and asymptomatic. We describe a patient with idiopathic ulcerative colitis who had PSC and symptomatic calcific pancreatitis associated with endocrine pancreatic deficiency.

Case Report

A 19-year-old man presented with history of diarrhea with blood and mucus for 3 years, jaundice for 3 years and recurrent episodes of abdominal pain for one and a half years. He had previously received treatment with oral corticosteroids for a few weeks and 5-aminosalicylic acid (5-ASA) with remission in diarrhea and bleeding. Jaundice was fluctuant and was associated with moderately severe pruritus. Six months prior to presentation, he had developed ascites which lasted for 3 months and improved with salt restriction and diuretics. There was no history of hepatic encephalopathy. He had frequent episodes of severe abdominal pain, suggestive of pancreatic pain, which used to improve with analgesics, intravenous fluids and restriction of oral intake. Pain was not associated with any local or systemic complications of pancreatitis. There was no history of biliary colic, alcohol abuse, drug intake (other than 5-ASA), abdominal trauma or worm infestation. There was no history of diabetes mellitus or steatorrhea.

A survey of previous investigations revealed conjugated hyperbilirubinemia (serum bilirubin 4 to 15 mg/dL), persistently elevated serum alkaline phosphatase (4 times the upper limit of normal), near normal serum transaminases and hypoalbuminemia (serum albumin 2.3-3.0 g/dL). Serum amylase had been high (4-5 times the upper limit of normal) during two episodes of abdominal pain.

Physical examination revealed mild pallor, moderate icterus, flexion contractures of both knee joints and marked girdle muscle wasting. Abdominal examination revealed mild hepatomegaly (span 12 cm) and splenomegaly (3 cm below left costal margin), but no shifting dullness. Chest, cardiovascular and neurological systems were unremarkable. There was no evidence of any fat-soluble vitamin deficiencies or ophthalmologic, cutaneous or articular manifestations of IBD.

Investigations: hemoglobin 8.9 g/dL, dimorphic peripheral blood picture, serum bilirubin 17 mg/dL (conjugated 11.6), serum protein 7.4 g/dL (albumin 2.3), serum AST 24 KU, ALT 56 KU, alkaline phosphatase 418 IU (normal <125), prothrombin time 18 seconds (control 14), activated partial thromboplastin time 40 seconds (control 27), fasting blood sugar 257 mg/dL, serum amylase 282 U/L (normal < 108), and serum creatinine 1.8 mg/dL. Plain abdominal X-ray revealed focal calcifications at first lumbar vertebral level, suggestive of pancreatic calcification.

Upper gastrointestinal endoscopy revealed two columns of grade I esophageal varices. Colonoscopy revealed pancolitis with loss of vascular pattern, a broken light reflex, and a few pseudopolyps scattered all over the colon. There was no hyper trophy, friability or ulceration. Terminal ileal mucosa was normal. Colonic mucosal biopsies revealed cryptitis, crypt loss, crypt branching and edema of the lamina propria with mixed inflammatory cell infiltration, suggesting active ulcerative colitis. Distal ileal biopsies were unremarkable. Double contrast barium enema revealed loss of haustration, mucosal irregularities suggestive of fine ulcerations and multiple pseudopolyps scattered all over the colon.

Ultrasoundography and contrast-enhancement computed tomography (Fig) revealed splenomegaly, collateral vessels at the splenic hilum, fine calcifications in pancreatic parenchyma and dilated pancreatic duct with intraductal calculi. Common bile duct was dilated.

Fig: Computed tomogram showing dilated pancreatic duct (oblique arrows), intraductal calcifications (vertical arrow) and splenomegaly

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not seen and intrahepatic biliary radicles were normal. Liver appeared normal in size and density. Endoscopic retrograde cholangiopancreatogram revealed normal common bile duct, a beaded left hepatic duct and marked pruning with difficulty in opacifying intrahepatic biliary ducts; these findings were suggestive of PSC. Pancreatic ductal system showed changes suggestive of grade IV chronic pancreatitis (Cambridge classification). Fecal chymotrypsin was 13.1 U/g (N>8.4). HBsAg and antibodies to human immunodeficiency virus were absent. Liver biopsy was not done.

A diagnosis of idiopathic ulcerative colitis with PSC and chronic calcific pancreatitis was made. The patient was treated with a 2000-Kcal, 60-g-protein diet, supplementation of vitamin A, vitamin D and calcium, 5-ASA, ursodeoxycholic acid, analgesics and pancreatic enzyme supplementation. His abdominal pain improved over the next few days and bilirubin level fell to 11.0 mg/dL. The patient refused endoscopic intervention or surgery for the bilo-pancreatic abnormalities.

Discussion

Our patient had chronic idiopathic ulcerative colitis and PSC leading to poor liver function and portal hypertension. The unusual feature was the occurrence of chronic calcific pancreatitis.

Liver and biliary tract abnormalities account for a significant proportion of extracolonic manifestations of idiopathic IBD. However, involvement of the pancreas is uncommon. In one study of 237 patients with IBD, 37 (16%) had laboratory abnormalities indicating hepatobiliary disease; these abnormalities were encountered more often in patients with Crohn’s disease than in those with ulcerative colitis (30.4% and 11.2%, respectively). Twenty-three of these 37 patients had cholangiographic changes suggestive of PSC; 11, in addition, had pancreatic ductal changes on pancreatography. None of the latter patients had symptoms due to pancreatic disease. In another study of 42 patients with PSC who had undergone endoscopic retrograde cholangiopancreatography, 3 (7.1%) had morphologic changes suggestive of chronic pancreatitis; however, 2 of these had history of alcohol abuse. In a third report, 4 of 17 patients with PSC had pancreatic ductal changes; however, none of these patients had any pancreatic symptoms and secretin test was abnormal in only one patient.

Our patient had symptomatic chronic calcific pancreatitis associated with endocrine pancreatic insufficiency and required frequent hospital admissions for pain relief. Such severe changes are unusual in patients with IBD and PSC. Further, he had no history of alcohol consumption. Coexistent tropical calcific pancreatitis is possible; however, he belonged to the northern part of India, where this disease is uncommon.

The reason for pancreatic damage in patients with IBD and PSC may lie in the embryologic origin of the exocrine pancreas. Damage to biliary structures in patients with IBD and PSC is believed to be mediated by antibodies directed against colonic mucosa, which cross-react with biliary epithelium. Origin of the pancreas and biliary tree in the form of a common bud during embryological life may imply that pancreatic ductal changes too may be related to these antibodies.

References


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