Ultrasonography revealed grade I parenchymal changes in the kidney and a cystic area 2 cm in size in the body of the pancreas. Upper gastrointestinal endoscopy was normal. Duodenal biopsy did not reveal any parasites or crypt villous changes. MRI (T2W axial imaging) showed a 5 cm x 4 cm x 3.5 cm well-defined soft-tissue mass in the body of the pancreas. Splenic vessel angiography showed tumor blush in the corresponding area with multiple feeding arteries.

Percutaneously, the diaphragm was controlled with ocetration. During surgery, palpation of the pancreas revealed a single 4 cm x 5 cm tumor in the body and tail junction. Superior mesenteric artery and portal vein were not involved. Distal pancreatectomy and splenectomy was done. Histology of the resected specimen showed pancreatic tissue with partly circumscribed tumor composed of papillary structures with thin fibrovascular cores and lined by columnar to cuboidal epithelial cells with vesicular nuclei and moderate amounts of cytoplasm. Immunohistochemistry (Fig) showed staining of tumor cells with monoclonal antibodies to VIP. Pancreatic lymph nodes were also involved by the tumor.

After surgery, diaphragm stopped completely. At 18 months of follow up, she was completely asymptomatic. Repeat CT scan of the abdomen did not reveal recurrence of tumor.

The most consistent features associated with VIPoma are episodic severe secretory diarrhea, hypochlorhydria, hypokalemia and metabolic acidosis. VIP has been shown to stimulate adenylate cyclase activity and hence secretion by the enteroctyes. Only 20% of patients with VIPoma exhibit flushing. Other abnormalities like hypomagnesemia and hypophosphatemia, as seen in this case, are possibly secondary to profuse diarrheal losses. Potassium loss is due to its passive movement into the stool water as part of the bulk fluid flow through the small intestine, although some active secretion by the colon may also contribute. Presence of steatorrhea in this patient is unexplained; it is an uncommon occurrence in VIPomas.

The prognosis is good in childhood VIPoma. Diagnosis is often delayed despite radiological and biochemical methods. In our case, two years elapsed before diagnosis and the tumor had already metastasized to the lymph nodes. Greater clinical awareness of this syndrome would lead to earlier detection and improve the chances of successful resection and symptomatic cure.

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Acute pancreatitis in a child with idiopathic ulcerative colitis on long-term 5-aminosalicylic acid therapy
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Acute pancreatitis is a rare but known complication of inflammatory bowel disease in adults. In children, only a few cases with this complication have been reported. We describe a 10-year-old boy with ulcerative colitis who developed acute pancreatitis while on long-term treatment with 5-aminosalicylic acid. [Indian J Gastroenterol 2000;19:195-196]

Key words: Inflammatory bowel disease, pancreas

Acute pancreatitis is a rare but known complication of 5-aminosalicylic acid (5-ASA) therapy in adults with inflammatory bowel disease. It classically appears within the first few days or weeks after initiation of therapy. We report a 10-year-old boy with idiopathic ulcerative colitis on 5-ASA who developed a mild clinical episode of acute pancreatitis.

A 10-year-old boy was admitted to our hospital with bloody diarrhea, abdominal pain, and tenesmus of one-year duration.
Sigmoidoscopy and histology confirmed the diagnosis of idiopathic ulcerative colitis. He was started on oral 5-ASA (Mesacol; Sun Pharmaceuticals, Ahmedabad) 400 mg twice daily and steroid enemas (dexamethasone). Within a month, he improved with resolution of diarrhea and weight gain. Only 5-ASA was then continued.

Five months later, he started passing small amounts of fresh blood admixed with stools of normal consistency which lasted for two weeks. The dose of 5-ASA was raised to 800 mg twice daily. A day later, he had severe mid-epigastric pain with vomiting, worsening with food intake and causing him to sit up and lean forward. There was no history of abdominal trauma, mumps in the neighborhood or school contacts, or any other drug intake preceding this illness. On examination, he had marked tenderness in the epigastrium.

Investigations: serum amylase 614 SU/dL (normal <400), increasing to 870 SU/dL after ten days. Serum calcium (9.5 mg/dL), serum phosphorus (5.7 mg/dL), ALT (29 U/L), serum lactate dehydrogenase (417 U/L), serum creatinine (0.7 mg/dL) and fasting serum triglycerides (102 mg/dL) were normal. Blood culture was sterile; antinuclear antibody was negative, IgM antibody titers against mumps virus was not elevated, and X-ray abdomen displayed a dilated left colon. Ultrasonography revealed hypoechoic edematous pancreas suggestive of acute pancreatitis; there were no gallstones and the biliary tract was normal.

Drug-induced pancreatitis was suspected and 5-ASA was stopped. He was admitted, given parenteral hydration, kept nil oral and on nasogastric tube drainage for three days. He was asymptomatic from the third hospital day and was started on oral feeds from the fourth day, which he tolerated. Gastroduodenoscopy on the seventh day showed no ulceration or inflammation near the ampulla of Vater. Duodenal contents did not reveal any evidence of biliary microlithiasis; biopsy of gastric antrum and duodenum did not show definitive evidence of Crohn's disease.

He was advised repeat colonoscopy and resumption of 5-ASA in graded doses under monitoring once serum amylase reached normal limits. His parents however preferred to take him to his local gastroenterologist for further management.

In contrast to adults, only a few reports of acute pancreatitis occurring in children with ulcerative colitis are available in literature. In adults, the usual promoting factors were drugs (azathioprine and salazosulfapyridine) and mechanical alterations of the bile duct (primary sclerosing cholangitis) or the pancreas (pancreas divisum). Hyperamylasemia and hyperlipasemia without clinical or radiological evidence of acute pancreatitis occurred with a greater frequency (21.3% of patients with ulcerative colitis) than did acute pancreatitis (2.1% of patients with ulcerative colitis) in a study of 179 patients with inflammatory bowel disease.

The role of 5-ASA in inducing acute pancreatitis in inflammatory bowel disease within the first few days or weeks of therapy has been documented. Acute pancreatitis resolved rapidly on withdrawal of the drug, and in some cases recurred on restarting 5-ASA in smaller doses. There has been only one report of acute pancreatitis occurring in two adults after being on long-term therapy with 5-ASA. In one patient, it occurred after 3 months of treatment whereas in the other, it occurred after two years. In both, the pancreatitis followed a benign course and recurred within 12-24 hours of performing rechallenge with 5-ASA.

In children, the association between 5-ASA and acute pancreatitis has been reported in only one instance so far.

The possibility of acute pancreatitis being an extra-intestinal manifestation of ulcerative colitis in this child cannot be ruled out. Definitive proof that 5-ASA caused pancreatitis requires that pancreatitis develop during treatment with the drug, that other likely causes of pancreatitis are absent, that pancreatitis resolved upon discontinuation of the drug, and that it recurred upon readministration of the drug. In this instance, the first three criteria were fulfilled. Rechallenge with 5-ASA was advised but could not be carried out. The mechanism of 5-ASA-induced acute pancreatitis is postulated to be a hypersensitive or allergic reaction.

Drug-induced acute pancreatitis must be considered when abdominal pain occurs or increases during therapy in a patient with ulcerative colitis. It warrants discontinuation of the drug if a similar response is obtained within a few days on rechallenge.

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