

Ulcerative Colitis Presenting as Acute Pancreatitis in a 6-Year-Old Patient

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The association of pancreatitis and ulcerative colitis has been well described in the literature.¹⁻¹¹ Most reported patients have experienced acute pancreatitis due to drug toxicity.^{1,5-9,11} The association of pancreatic duct abnormalities and ulcerative colitis has also been described in the literature.^{12,13} Idiopathic pancreatitis is the term used to describe pancreatitis when no causes or associations are identified.³ We report the case of a young patient with acute idiopathic pancreatitis as a presentation of ulcerative colitis.

Case Report

A 6-year-old girl presented with severe abdominal pain and bilious vomiting. There was no history of abdominal trauma or drug intake nor any family history of pancreatitis. Physical examination was remarkable for epigastric tenderness. Serum amylase measured 1,654 IU/L (normal, 5–83 IU/L), and lipase measured 2,343 IU/L (normal, 27–254 IU/L). Urinalysis, serum electrolytes, cholesterol, serum triglycerides, calcium, aspartate aminotransferase, alanine transaminase, bilirubin, alkaline phosphatase, blood urea nitrogen, and creatinine were all normal. Abdominal ultrasound demonstrated increased echogenicity and edema of the pancreas, but there were no biliary duct abnormalities or stones.

The patient was admitted to the hospital with nothing to eat. A central line was placed, with total parenteral nutrition and analgesia administered to the patient. She continued to be symptomatic, with no significant improvement in abdominal pain or pancreatic enzymes over the next week. On the eighth day of admission, the patient developed bloody diarrhea with lower abdominal cramping and tenesmus. Stool specimens for culture and *Clostridium difficile* infection were negative. Colonoscopy

revealed diffuse confluent inflammation of the colonic mucosa with ulcers extending from the rectum to mid-transverse colon. The ascending colon and rectum were spared, and terminal ileum intubation did not show any abnormality. Biopsies of the abnormal mucosa showed cryptitis and crypt abscesses with a predominance of chronic inflammatory cells. Inflammatory bowel disease serology was ordered. The result was positive for anti-neutrophil cytoplasmic antibodies. A diagnosis of ulcerative colitis was thus made based on the combination of clinical symptoms and signs and endoscopic, histologic, and serologic data.

Meanwhile, the patient was started on methylprednisolone sodium succinate (Solu-Medrol, Pharmacia & Upjohn) 1 mg/kg. Forty-eight hours later, the patient's amylase and lipase levels started to drop, and her abdominal pain improved. The number of stools decreased from 8 to 3 per day, free from blood. Over the next 3 days, the patient resumed a low-residue diet with normalization of pancreatic enzymes and no gastrointestinal symptoms. Magnetic resonance cholangiopancreatography (MRCP) was performed 2 weeks later and did not show any abnormalities in the pancreatic or biliary ducts. The patient was followed for the next 3 years. She did not develop any more episodes of pancreatitis, and her ulcerative colitis remained in remission on mesalamine.

Discussion

A thorough review of the literature was conducted to identify children with reports of pancreatitis in association with ulcerative colitis. Prior to our patient, the youngest child previously reported was 15 years old and was on mesalamine for 2 weeks prior to presentation.¹

Identifiable causes of acute pancreatitis include trauma in 13–20% of cases, infection in 3–15%, biliary tract disease in 10–14%, drugs in 11–13%, hereditary predisposition in 7–10%, congenital anomalies in 5%, hypercalcemia in 1–3%, hypertriglyceridemia in up to 3%, and cystic fibrosis in even fewer cases.^{14,15} In our

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patient, however, no cause was identified. Drugs used in ulcerative colitis and implicated in the etiology include: 6-mercaptopurine,⁵ mesalamine,^{1,6,7,9,11} steroids,^{8,11} and metronidazole.^{10,11} Our patient was not on any of these medications and did not have any of the predisposing factors. In addition, MRCP ruled out any pancreatic or biliary duct abnormalities, unlike the cases previously reported.^{12,13}

It remains a matter of debate whether idiopathic pancreatitis is coincidental or can be considered a rare extraintestinal manifestation of inflammatory bowel disease.¹⁶ Autopsy studies in ulcerative colitis have revealed the presence of macroscopic or microscopic pancreatic lesions in 14–53% of 86 cases.¹⁷ As in this case report, only 1 previously reported case had similar simultaneous presentation.⁴ However, this patient was 57 years old. In all other reported cases, there was a significant length of time between the episode of pancreatitis and the onset of ulcerative colitis,^{2,3} or the patient had been on medications as mentioned earlier. Furthermore, our patient did not experience a recurrence of her pancreatitis as long as her ulcerative colitis was in remission.

It has been speculated that papillary edema due to duodenal mucosal involvement in Crohn's disease may comprise the pathophysiology of associated idiopathic pancreatitis.¹⁸ Such an etiology is not valid in ulcerative colitis because of the absence of duodenal involvement.

Conclusion

Although rare, acute idiopathic pancreatitis can be a presentation of ulcerative colitis. Pediatric gastroenterologists are urged to consider expanding the work-up of acute idiopathic pancreatitis to include screening for ulcerative colitis when management of the condition does not result in clinical improvement. Prospective controlled trials are needed in this age group to attempt to determine the frequency. This will direct further recommendations in regard to guidelines for such screen-

ing. Studies that seek the possible pathophysiology of such associations are also needed.

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