Idiopathic Pancreatitis May Be Associated with Ulcerative Colitis

**Key words:** idiopathic pancreatitis, ulcerative colitis, extraintestinal manifestation

A 36-year-old man was admitted to our hospital on October 2, 2000 because of left hypochondriac pain four days before and elevated pancreatic enzymes. He had experienced the same abdominal pain on August 30 and September 14, 2000. There was no history of trauma, precipitating viral infection, habitual alcohol consumption, gallbladder disease, or taking medications. On physical examination, there was localized tenderness in the left upper quadrant.

Laboratory examinations showed Hb of 15.0 g/dl, platelets of 211,000/μl, WBC count of 8,900/μl, and C-reactive protein (CRP) of 1.1 mg/dl. Blood chemistry tests revealed aspartate aminotransferase of 14 IU, alanine aminotransferase of 27 IU, alkaline phosphatase of 355 IU, albumin of 4.3 g/dl, globulin of 2.3 g/dl (γ-globulin 13.6%), total bilirubin of 0.9 mg/dl, serum amylase of 764 IU/l (pancreatic% 99%), urine amylase of 5,004 IU/l (pancreatic% 99%), lipase of 2,577 IU/l, total cholesterol of 164 mg/dl, triglyceride of 101 mg/dl, calcium of 8.8 mg/dl, and phosphorus of 2.9 mg/dl. Autoantibodies including antinuclear antibody, SS-A and SS-B antibodies, smooth muscle antibody, and rheumatoid factor were all negative. Abdominal ultrasonography, contrast-enhanced computed tomography scan and magnetic resonance (MR) demonstrated a cystic lesion 2.5 cm in diameter in the tail of the pancreas. There was no choledolithiasis. MR cholangiopancreatography showed a normal pancreatic duct, biliary duct, and gallbladder. A diagnosis of idiopathic acute pancreatitis was made based on the above findings. The patient became asymptomatic and laboratory data were normalized with total parenteral nutrition in about 4 weeks. The cystic lesion of the pancreatic tail dissapeared in the imaging study.

On June 26, 2001, he was transferred to our hospital with complaints of bloody diarrhea and lower abdominal pain for 10 days. Physical examination was normal except for blood in the rectum. Laboratory data including amylase and lipase were normal except for CRP of 0.4 mg/dl. Abdominal ultrasonography showed normal pancreas. Colonoscopy revealed diffuse, continuous proctitis with absent vascular pattern, and contact bleeding with skip lesion of the orifice of the appendix. Biopsy specimens of the rectal mucosa and the orifice of the appendix showed mucosal inflammation and crypt abscess formation. The endoscopic and histologic features were consistent with a diagnosis of ulcerative colitis (UC). He was treated with oral 5-aminosalicylic acid (1,500 mg/day) and made a rapid clinical recovery and became asymptomatic after two weeks.

UC is associated with numerous extraintestinal manifestations, including pancreatitis. The association between pancreatic disease and UC was first suggested by Ball et al in 1950. Autopsy research in UC showed the presence of macroscopic and microscopic pancreatic lesions in 14% and 53% of 86 cases (1). Some case reports of UC-associated pancreatitis have been published during past years.

UC and pancreatitis are linked, although these may occur together by chance. This suggestion is supported by the absence of known causes of pancreatitis. The recognized causes of pancreatitis including alcohol abuse, gallstones, underlying hepatobiliary complications, hyperlipidemia, hypercalcemia, drugs, and pancreatic tumor were excluded in the present case. We thus considered the pancreatic disease in this case as one of the extraintestinal manifestations of UC, although there was no distinct evidence which suggested the association of the two. Although the pathogenesis of UC-associated pancreatitis is not still understood, an autoimmune mechanism is suggested as its cause (2). However, this case had no findings which suggested the relation of autoimmunity.

Most cases of UC-associated pancreatitis manifest pancreatitis after or concomitant with the clinical onset of UC, but there are several cases in which pancreatitis was found to occur before UC (3–7). Our case also presented with pancreatitis before the clinical manifestation of UC. In previous reports, the diagnosis of pancreatitis preceded UC-related manifestation by a median interval of 7 months with a range of 1 to 120 months (3–7). UC after the onset of pancreatitis shows various presentations such as mild (3, 4) or severe symptoms (5, 6) and to various extents such as only the rectum (4) to total colon (5). In this case, pancreatitis was detected 9 months before the onset of UC and the activity of UC was mild. These observations suggest that the clinical onset of pancreatitis may not be correlated to the activity of UC. UC-associated pancreatitis shows various presentations such as acute pancreatitis like ours and chronic pancreatitis. The common clinical characteristics of acute pancreatitis associated with UC are as follows: mild symptoms, moderate elevation of serum pancreatic enzymes, no abnormal findings in the imaging study (sometimes stricture of the main pancreatic duct in endoscopic retrograde pancreatography), good prognosis. Our case also showed the above characteristics.

When pancreatitis occurs in the patient with UC, it is easy to consider the association of the two. However, when pancreatitis precedes the symptoms of UC, it is difficult to consider the association of UC. Therefore, when idiopathic pancreatitis is found, the association of UC needs to be considered. It is necessary to follow the patient to confirm...
whether UC will occur or not.

Akihiro OKANO, Hiroshi TAKAKUWA and Akiyoshi NISHIO

From the Department of Gastroenterology, Tenri Hospital, Tenri
Received for publication July 11, 2002; Accepted for publication December 2, 2002
Reprint requests should be addressed to Dr. Akihiro Okano, the Department of Gastroenterology, Tenri Hospital, 200 Mishima-cho, Tenri, Nara 632-8552

References