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Idiopathic Pancreatitis Associated with Inflammatory Bowel Disease

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The list of extraintestinal manifestations of inflammatory bowel diseases does not classically include pancreatitis and pancreatic insufficiency. We report here six cases of unexplained pancreatitis associated with inflammatory bowel disease (five patients with Crohn's disease, one with indeterminate colitis). None of the classical etiologies for pancreatitis was found in our patients; moreover none of them had duodenal localization of Crohn's disease or sclerosing cholangitis, two conditions in which pancreatitis associated with inflammatory bowel disease has been previously described. Pancreatitis was painless (or was associated with moderate and atypical abdominal pain) in four of our six cases; no pancreatic calcification was found in any case; in three patients a total or subtotal exocrine pancreatic insufficiency was evidenced. Endoscopic retrograde pancreatography performed in four subjects showed normal or minimally altered pancreatic ducts even in those with severe pancreatic insufficiency. These cases emphasize the existence of a probably nonfortuitous association of inflammatory bowel disease with pancreatitis. Its recognition could make a significant contribution in the management of inflammatory bowel disease.

Many extraintestinal manifestations (1, 2) have been described in association with inflammatory bowel diseases (IBD). Pancreatitis is not classically included in these descriptions: it is not mentioned in two major works on extraintestinal manifestations of IBD (1, 2) totaling 202 patients with ulcerative colitis (UC) and 1067 subjects with Crohn's disease (CD) and is not quoted in two recent textbooks of gastroenterology (3, 4). The aim of the present paper is to present six cases of IBD associated with unexplained pancreatitis and, in three of them, total exocrine pancreatic insufficiency.

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MATERIALS AND METHODS

These six patients were seen between 1976 and 1984 and are drawn from a series of 553 subjects suffering from IBD and followed during the same period in our department (331 had CD, 176 with UC and 46 with indeterminate colitis) (Tables 1 and 2).

Case 1. A previously healthy 21-year-old man was admitted in April 1982 for a six-month history of diarrhea (3–4 loose and bloody stools daily), epigastric and lower left quadrant pain, 7-kg weight loss, and moderate hyperamylasemia. Physical examination was unremarkable. Routine laboratory data were normal except for a microcytic (mean corpuscular volume: 78 μm^3) hyposideremic (serum iron: 15 $\mu g/dl$) anemia (hemoglobin: 9 g/dl). D-Xylose and Schilling test were normal, but a massive steatorrhea was found (90 g/24 hr). Pancreatic investigations (Table 2) revealed total exocrine pancreatic insufficiency; none of the usual causes of pancreatitis was found (see below).

Small bowel x-rays were normal; upper digestive endoscopy up to the angle of Treitz showed a normal mucosa, and three duodenal biopsy samples were normal.

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TABLE 1. GENERAL DATA AND INFORMATION ON INFLAMMATORY BOWEL DISEASE (IBD)*

	Case					
	1	*2*	*3*	*4*	*5*	*6*
Age (years)	21	62	30	18	31	23
Sex	M	M	M	F	F	F
IBD						
Nature	CD*	CD	Indeterminate	CD	CD	CD
Location	Colon	Ileum, colon	Colon	Colon	Ileum, colon	Colon
Extradigestive manifestations	0	0	0	EN, episcleritis, bronchial tuberculoid granulomas	0	0
Chronology of diagnosis of IBD and IP*	Simultaneous	IBD 16 yr before IP	Simultaneous	IBD 4 mo after IP	IBD 18 mo before IP	IBD 17 mo after IP

*CD = Crohn's disease; EN = erythema nodosum; IP = idiopathic pancreatitis.

Total colonoscopy showed a normal rectal mucosa and the presence of red plaques, microulcerations, and granular mucosa from the sigmoid colon up to the right hepatic flexure; the right colon and terminal ileum were normal. Multiple colonic biopsy samples showed a moderate to severe nonspecific inflammatory infiltrate of the lamina propria, cryptitis, erosions, several lymphoid follicles, and one small epithelioid granuloma; there was no depletion of goblet cell mucin. A one-month course of prednisone (1 mg/kg body weight) induced a clinical and endoscopic remission of the colitis; total exocrine pancreatic insufficiency persisted on a second Lundh meal in July 1982, and pancreatic extracts were prescribed. The colitis remained quiescent during 20 months but flared up in April 1984 and was associated at that time with an ileal involvement.

Case 2. A 62-year-old man, previously known to have an asymptomatic gallbladder stone and quiescent Crohn's disease, was admitted in June 1980 for acute abdominal pain. Terminal ileitis had been discovered in 1954 at laparotomy for a lower right quadrant mass: appendicectomy was carried out and tuberculoid granulomas were found at histological examination of the appendix. In the following year, he underwent a right hemicolectomy and ileectomy. In 1960, mild diarrhea recurred and he received intermittent sulfasalazine therapy. A nonstenotic recurrence of Crohn's disease was documented on the preanastomotic ileum in June 1979; no treatment was prescribed since he was almost asymptomatic.

At the end of June 1980, he experienced three severe attacks of upper abdominal pain with vomiting, diarrhea, and low-grade fever (37.8° C). On admission, physical

TABLE 2. PANCREATIC INVESTIGATIONS*

	Case					
	1	*2*	*3*	*4*	*5*	*6*
Maximal amylase levels (ULN*), blood/urine	×2/×1	×3/×2	×2/×8	×11†/×12	×4/×2	×3/×3
Diabetes mellitus	No	No	Yes	No	No	No
Fecal fat (g/24 hr)	90	64	16	ND‡	ND‡	3
Lundh's test						
Lipase (N > 300 units/ml)	0§	0	26	ND‡	ND‡	275
Chymotrypsin (N > 50 units/ml)	0§	0	6			46
Pancreatic calcification	No	No	No	No	No	No
Pancreatic ultrasonogram	Normal	ND‡	Pancreatic enlargement	Pancreatic enlargement	ND‡	Normal
ERP¶	Normal	ND‡	Minimal main duct irregularities	Slightly dilated and irregular ducts	ND‡	Minimal main duct irregularities
Laparotomy	ND‡	Diffusely rock-hard pancreas	ND‡	ND‡	Foci of cytosteatonecrosis	Enlarged, hard, and nodular pancreas

*ULN = upper limit of normal.
†Lipase blood levels were at 344 units (N < 200).
‡ND = not done.
§Total pancreatic insufficiency was confirmed by a secretin–cerulein test.
¶ERP = endoscopic retrograde pancreatography.

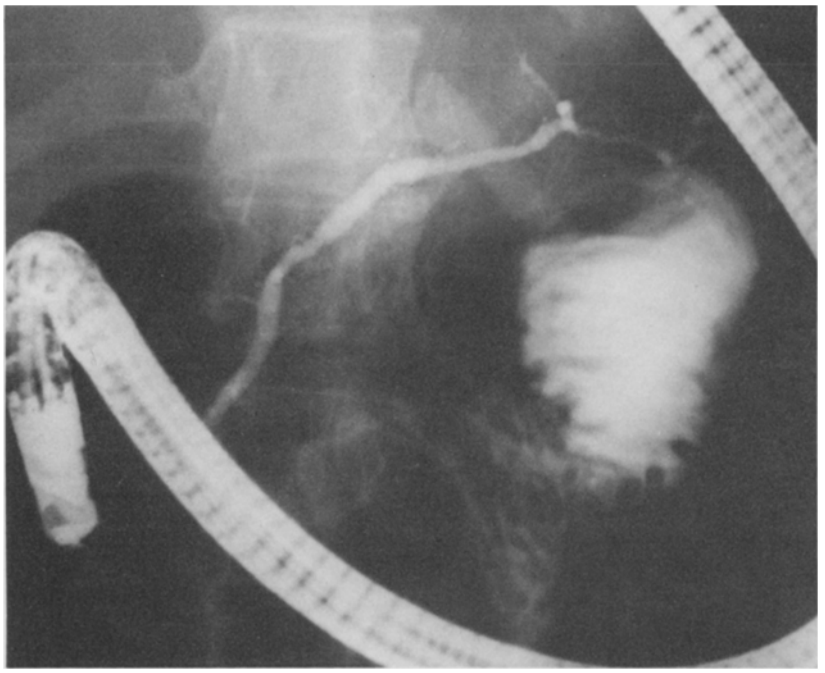

Fig 1. Endoscopic retrograde pancreatography showing moderate dilatation and irregularity of the main pancreatic duct with irregularities and dilatations of secondary ducts in the tail of the pancreas (case 4).

examination was normal. Plain film of the abdomen only showed the radiopaque gallstone; a barium follow-through was identical to that of June 1979. Biological investigations only showed increased levels of plasma and urine amylase (Table 2). A cholecystectomy and a liver biopsy were performed in July 1980. The gallbladder contained two stones and biliary sludge; peroperative cholangiogram only showed moderate compression of the retropancreatic common bile duct. The pancreas was obviously and diffusely abnormal, enlarged, rock-hard, and very suggestive of chronic pancreatitis. There was no cytosteatonecrosis. On histological examination, the gallbladder showed chronic cholecystitis and the liver was normal. The patient made an uneventful recovery and is presently asymptomatic except for two loose stools per day. Pancreatic evaluation (Table 2) showed total exocrine pancreatic insufficiency; no cause of chronic pancreatitis was found (see below).

Case 3. A 30-year-old man was admitted in August 1981 for bloody diarrhea. His past medical history included an idiopathic pneumothorax and the resection of a benign tumor of the tongue. In June 1981 he began to experience 5–6 liquid stools with blood and mucus, abdominal pain, and fever. On admission he had lost 8 kg; physical examination was otherwise normal. Routine biological examinations only showed a low serum iron (6.4 μg/dl) and a mild diabetes mellitus; on colonoscopy the mucosa was normal in the rectum but diffusely granular and erythematous from the sigmoid to the transverse colon. Colonic biopsy samples showed a histological picture suggestive of ulcerative colitis: cubic epithelium, loss of mucosecretion, a few crypt abcesses, and nonspecific infiltration of the lamina propria with lymphocytes and plasma cells. Fecal fat was at 16 g/24 hr; D-xylose absorption, small bowel x-rays, and duodenal endoscopy with biopsy samples were normal. Pancreatic investigations are shown in Table 2 and revealed exocrine insufficiency; a search for the usual causes of pancreatitis was negative (see below).

The patient received sulfasalazine (2 g/24 hr) and pancreatic extracts and improved rapidly with normal colonic endoscopy and biopsy samples in November 1981. Ten months later he was asymptomatic while receiving no treatment; fecal fat was 6 g/24 hr; a second Lundh meal showed that lipase and chymotrypsin concentrations were just below the lower limit of normal.

Case 4. An 18-year-old woman was referred in January 1983 for endoscopic wirsungography because of unexplained hyperamylasemia. This previously healthy young woman developed four attacks of erythema nodosum confirmed by skin biopsy between December 1981 and November 1982. The etiology of the skin lesion remained obscure despite multiple investigations including a normal colonoscopy and ileoscopy. Routine biochemistry revealed hyperamylasemia and hyperamylasuria. On admission the patient was symptom-free; physical examination only showed a few areas of brownish discoloration of the lower limb skin. Pancreatic evaluation is shown in Table 2 and Figure 1. Several investigations failed to find a cause for the pancreatic disease (see below).

In April 1983 she was readmitted for diarrhea of two months' duration (4–5 liquid stools with blood and mu-

cus), dyspnea, and a further attack of erythema nodosum. Bronchoscopy showed granulations scattered on an erythematous mucosa; bronchial biopsy samples disclosed a few epitheloid cells. On colonoscopy, small ulcerations of the right colon and a thickened and reddened mucosa in the transverse and left colon were found. The rectum was normal; several biopsy samples showed superficial ulcerations and tuberculoid granulomas with epithelioid and giant cells; small bowel barium x-rays were normal. Ocular examination revealed an episcleritis. Digestive and extradigestive symptoms improved on steroids. She suffered from three further attacks (September 1983, January and May 1984) of colitis and erythema nodosum, which responded to oral steroids; mild hyperamylasuria was again documented in January 1984.

Case 5. A 31-year-old woman was admitted in July 1976 for a severe attack of colitis. She had been followed elsewhere since 1966 for ankylosing spondylitis. The first intestinal manifestations occurred in February 1975, and she received intermittent treatment with sulfasalazine and rectal betamethasone. Her last intake of sulfasalazine was in May 1976. In June 1976, an exacerbation of intestinal symptoms resisted a treatment by oral prednisolone (20 mg/day) and she lost 10 kg. On admission she was acutely ill, with fever (39.5° C), 15 bloody and liquid motions per day, and abdominal pain. Hyperamylasemia was documented on four occasions. Barium enema showed a diffuse pancolitis with widespread ulcerations. The patient was started on total parenteral nutrition, intravenous hydrocortisone hemisuccinate (300 mg/day), cephalothin, and gentamicin. After a transient improvement, her state worsened and a colectomy with ileorectal anastomosis and diversion ileostomy was performed. During this operation foci of cytosteatonecrosis were found in the pancreatic area. A liver biopsy was done. Colonic histology showed continuous and diffuse lesions with deep ulcerations, considered suggestive of ulcerative colitis. Liver biopsy sample was normal except for a mild steatosis.

On the 21st postoperative day on occlusive syndrome with tenderness of the left upper quadrant led to a second laparotomy: a prepancreatic abcess was found and drained. The ileorectal anastomosis was normal as well as the gallbladder. A fragment of necrosis was sampled and shown to consist of cytosteatonecrosis at histology. Data concerning the etiology of the pancreatitis are given below. Ileostomy was closed in February 1978, but persisting diarrhea and a rectal stenosis with rectovaginal fistula led to a proctectomy, a short ileectomy, and ileostomy. The presence of lesions in the ileal resection (ulceration and sclerosis) led finally to the diagnosis of Crohn's disease. There was no lesion on a small bowel follow-through performed in 1982.

Case 6. A 23-year-old woman was admitted in October 1983 for chronic diarrhea. She first sought medical advice in July 1981 for abdominal pain and diarrhea; hyperamylasemia (1.7× upper limit of normal), and leukocytosis (13,000 white blood cells/mm^3) were found. Upper digestive fibroscopy revealed a duodenal ulcer, and the patient received cimetidine (1 g daily for 21 days). In September 1981, cimetidine was resumed for recurrent epigastric pain.

A week later, an acute abdominal syndrome with fever and hyperamylasemia led to a laparotomy. There was no ulcer perforation but the pancreas was grossly enlarged and hard with a few whitish nodules, one of which was sampled. At histological examination the pancreatic capsule was thickened by a fibrosis which penetrated deeply and dissociated pancreatic acini; there was a dense polymorphonuclear infiltrate with numerous abcesses. The patient made an uneventful postoperative course and continued to receive cimetidine until April 1982: a parietal cell vagotomy was then performed for ulcer relapse. The pancreatic area was found to be normal at this operation.

In February 1983, diarrhea recurred and she lost 5 kg: on admission she had 10 liquid and bloody stools per day and appeared chronically ill. Colonoscopy showed a normal rectum and the presence of aphthoid and superficial ulcerations and erythema with intervening normal mucosa on the rest of the colon, a picture typical of Crohn's disease. Six colonic biopsy samples showed nonspecific colitis (without tuberculoid granuloma). Small bowel x-rays were normal as well as upper digestive endoscopy. Prednisolone (1 mg/kg/day) and cimetidine (1 g/day) were started; 48 hr later she suffered from epigastric pain radiating to the back with hyperamylasemia (Table 2). There was no ulcer recurrence on repeated endoscopy; cimetidine was stopped and steroids pursued and the patient recovered rapidly. The results concerning the etiology of the pancreatitis are shown below. In December 1983, while she only received 25 mg of prednisolone, there was a flare-up of the colitis associated with epigastric pain radiating to the back and hyperamylasuria (2× upper limit of normal). Metronidazole was added, and the symptoms subsided.

Etiological Investigations for Pancreatitis. None of our patients had an history of alcoholism (alcohol intake was nil in cases 2, 4, 5, and 6, and less than 50 g/day in cases 1 and 3; they all had normal serum γ-glutamyl transpeptidase) or of familial pancreatitis. Serum and urinary calcium and phosphorus, serum total lipids and triglycerides, and serum α_1-antitrypsin were normal in all patients. Gallbladder ultrasonogram was normal in patients 1, 3, 4, and 6. None of our patients received azathioprine, thiazide diuretics, estrogens, furosemide or tetracycline, drugs reported to cause pancreatitis (5, 6). Sweat tests showed normal chloride concentration in cases 2 and 6. None of the patients had duodenal Crohn's disease (as shown by duodenoscopy in cases 1–4 and 6, and by barium x-rays in case 5). Search for sclerosing cholangitis, an entity reported to be associated with pancreatitis (7) was negative: all patients had normal serum alkaline phosphatase levels; there was no evidence of sclerosing cholangitis on intravenous (cases 1, 4–6), peroperative (case 2), and endoscopic retrograde (case 3) cholangiograms; patients 2 and 5 had normal liver histology.

DISCUSSION

Our six patients had an IBD associated with an idiopathic pancreatitis (IP). Five of them had CD, with tuberculoid granulomas in three subjects (cases 1, 2, and 4), an ileal recurrence after resection in two

(cases 2 and 5), and typical colonoscopic findings in three (cases 1, 4, and 6). Patient 3 had an indeterminate colitis sparing the rectum but with a histological picture suggestive of UC on biopsy samples.

None of the classical etiologies for pancreatitis was found in our patients. Patient 2 had a gallbladder stone: biliary stone may induce acute or relapsing pancreatitis, but it has never been incriminated as a cause for chronic pancreatitis with total exocrine pancreatic insufficiency (3, 8). In patient 6, two attacks of pancreatitis coincided with a recent intake of cimetidine, a drug reported, although rarely, to induce acute pancreatitis (9). However clinical, biological, and anatomical remission of the pancreatitis occurred while she was still on cimetidine and she had a further relapse of pancreatic pain and hyperamylasuria two months after the last cimetidine ingestion. Patient 5 developed acute pancreatitis while she was receiving corticosteroids, but the causal relationship between these compounds and pancreatitis is largely unproved (10). Two of our patients (cases 2 and 5) has received sulfasalazine. In the literature the causal relationship between this drug and pancreatic disease was clearly shown in only one patient who developed acute attacks of pancreatitis while he received the drug (11). In fact one of our patients (case 2) had chronic pancreatitis, and the other one had stopped the drug one month before the onset of the acute attack of pancreatitis. None of our patients received azathioprine, a drug reported to cause pancreatitis in CD (6).

The first suggestion of a significant association between pancreatic lesions and IBD was reported in 1950 by Ball et al (12), who found 14% and 53% of gross and histological pancreatic lesions, respectively, at a postmortem study of patients with UC. The same laboratory found mild to moderate pancreatic fibrosis at necropsy in 15 of 39 patients with CD (13). None of them had previous clinical evidence of pancreatic disease. In a systematic study of the pancreatic response to intravenous secretin in IBD (14), borderline abnormalities were found in 18% of the cases but, here again, the relevance of these findings is doubtful in the absence of any information on the clinical status of these patients. These results were not confirmed in a more recent study of the pancreatic fluid and bicarbonate response to secretin in CD (15).

More recently, clinically significant and non-drug-induced pancreatitis were reported in two subgroups of IBD patients: (1) patients with duodenal CD (16–22) in whom the role of a reflux of duodenal contents into the pancreatic duct through an incompetent ampulla (19) or of a direct ampullary involvement with stenosis (22) have been advocated in the pathogenesis of the pancreatitis; (2) patients (mostly UC) with sclerosing cholangitis or pericholangitis (23–27), two biliary tract diseases known to be associated with pancreatitis in the absence of any intestinal pathology (7, 28–30). Our six patients did not belong to any of these subgroups: they had no evidence of duodenal CD as shown by endoscopy (cases 1–4 and 6) and radiology (cases 1–6), or of biliary tract disease (except for one gallbladder stone) as judged from biological and morphological investigations (31).

Thus, our six cases represent apparently idiopathic pancreatitis associated with IBD. To the best of our knowledge only five similar cases (31, 32) have been briefly reported in the French and English literature so far (four of them come from a radiological series of abnormal pancreatograms). Although our small series does not allow us to establish whether IBD-associated pancreatis has specific clinical features, a few points can be emphasized: (1) Abdominal pain was absent or moderate and probably accounted for by the intestinal disease in four of our patients. In these subjects, it was steatorrhea (cases 1 and 3), painless hyperamylasemia (case 4), and the incidental finding of an enlarged and hard pancreas at laparotomy for acute calculous cholecystitis (case 2) that led to the diagnosis of pancreatitis. (2) Pancreatic calcifications were absent in our cases and were reported in only one of the previously published cases (32). (3) Three of our patients had total or subtotal exocrine pancreatic insufficiency and, surprisingly, the two of them who underwent endoscopic retrograde pancreatography had a normal or minimally altered Wirsung duct. Slightly abnormal pancreatograms were also found in three of the four patients described by Axon et al (33). (4) The time relationship between IBD onset and IP discovery was variable. They were diagnosed simultaneously in two patients; IP was detected four and 17 months before IBD in two other cases: in one of them (case 4) colonoscopy was normal at this time. In the two other patients, the diagnosis of IBD antedated that of IP by 1.5 to 16 years. (5) Finally it is of interest to notice that, in the only case where pancreatic histology is available, there was no evidence of a specific localization of CD.

Although the link between IBD and IP remains

obscure, we feel that a fortuitous association can be reasonably ruled out in view of the low incidence of both diseases in the general population (4, 34). In our series the calculated frequency of IP in CD is 1.5% (5 of 331); this figure is probably an underestimate since we have not performed systematic pancreatic investigations in CD patients. Only a carefully designed prospective study of pancreatic function (and when needed, morphology) in IBD could assess the true frequency of this association. In these patients pain and diarrhea are likely to be ascribed to the intestinal disease. Yet, the recognition of the pancreatic involvement could make a significant contribution to their management.

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