

Acute pancreatitis revealing ulcerative colitis—A case report

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ABSTRACT

The association of acute pancreatitis with ulcerative colitis [UC] has been described in the literature. It is usually induced by drugs, but sometimes it may be idiopathic. This association remains rare. We report the case of a patient who was treated in our department. **Medical observation:** Mr. KA, a 60-year-old man, was admitted in our unit for management of acute epigastric. The clinical examination at the admission was normal. After eliminating a cardiac or surgical cause, the diagnosis of acute pancreatitis was made on the basis of the presence of a serum lipase up to 5 times the normal level and pain intensity. An abdominal scanner tomography was performed for the assessment of the pancreatitis. It has shown a pancreatitis stage C associated with a thick rectosigmoidien that was discovered incidentally. Symptomatology was enriched 10 days after by the occurrence of rectal bleeding. A lower endoscopy was performed after the improvement of the pancreatitis and had shown an ulcerative colitis on pancolitis which was confirmed by biopsy. In order to search other causes of this pancreatitis, other tests were made [a biliary IRM, endoscopic ultrasonography, autoimmune tests] and the results were negative. We concluded an idiopathic pancreatitis. The ulcerative colitis was classified as moderate and the patient was put on oral corticosteroids with depression. The evolution was marked by a clinical and biological improvement of pancreatitis and colitis. **Conclusion:** The association of idiopathic pancreatitis with ulcerative colitis is rare. The case of our patient is the first case reported in our series with 400 cases of ulcerative colitis diagnosed in our service.

KEYWORDS

Acute Pancreatitis; Ulcerative Colitis

1. INTRODUCTION

The association of acute pancreatitis with ulcerative colitis (UC) has been described in the literature [1-3]. It is usually due to use of drugs [1,2,4,5] like salicylates and immunosuppressants (azathioprine and 6 mercaptopurine). However, this association may sometimes be idiopathic [3-6] or as part of extraintestinal manifestations of inflammatory colitis which are very rare. We report the case of a patient who was admitted in our hospital for acute pancreatitis and next examinations have revealed an asymptomatic ulcerative colitis.

2. MEDICAL OBSERVATION

Mr. KA, 60 years old, was admitted to our department for management of acute epigastric pain associated with early post prandial vomiting. The initial clinical examination objectified a mild epigastric tenderness. After removing cardiac or surgical causes, diagnosis of acute pancreatitis was made on the presence of a serum lipase up to 5 times the normal and pain intensity. The initial laboratory tests were normal including liver and kidney function, blood count and lipid profile, except the C-reactive protein which was elevated (CRP = 102). For assessing the severity of this pancreatitis, abdominal CT scan was performed and has revealed a stage C according to the Balthazar classification associated with a thickening of rectum and sigmoide [images 1 and 2]. The pancreatitis was managed by stopping feeding, rehydration and analgesic treatment for 72 hours with clinical improvement and disappearance of epigastric pain. Symp-

tomatology was enhanced 10 days after by lower intestinal bleeding and diarrhea (4 stools per day), all operating in a context of apyrexia and impairment of general condition. A lower endoscopy was performed and has objectified an erythematous and ulcerated mucosa without intervals of healthy mucosa from the anal canal to the right colon. We have concluded to the diagnosis of ulcerative colitis which was confirmed next by the results of biopsies. For searching the causes of pancreatitis, a cholangio MRI and a bili ultrasonography were normal, and autoimmune tests (anti LKM1, anti-smooth muscle, anti-mitochondrial, Anti-nuclear antibodies and dosage of IgG4) were also normal. We concluded an idiopathic pancreatitis. The colitis was classified as moderate and the patient was put on oral corticosteroids. The evolution was marked by an improvement in clinical and biological status of pancreatitis and colitis.

3. DISCUSSION

The association between pancreatic lesions and chronic inflammatory bowel disease has been reported since 1950 by Ball *et al.* [7] This autoptic study in 86 patients with UC, revealed lesions of pancreatitis in 14% - 53% of cases. The occurrence of pancreatitis associated with ulcerative colitis has been reported in specific contexts: alcoholism, viral infection, abdominal trauma, cholelithiasis, malnutrition, hypertriglyceridemia, hyperparathyroidism and drug (mesalamine, azathioprine, 6-mercaptopurine, steroids and metronidazole) [1,2,4,7-11]. In the absence of an evident cause pancreatitis including drug intake, pancreatitis is considered idiopathic. The pathophysiology of this association remains poorly understood, several theories have been put forward: the first believes that this association is fortuitous and the second refers to the fact that the pancreatitis could be an extra-intestinal manifestation of UC [3,12]. There are several mechanisms that can explain the increase of pancreatic enzymes in IBD: the first one is that the pancreas can be reached directly by the extension of IBD, the second potential mechanism is secretion of inflammatory mediators and cytokines due to the IBD, and the third mechanism is the inflammation of the pancreatic ducts. This explains the impaired pancreatic exocrine function which is the most common pancreatic abnormality in IBD. The association of hepatobiliary manifestations [sclerosing cholangitis, pericholangitis, chronic pancreatitis] to UC may precedes or occur after the diagnosis of UC, and this association is considered as an autoimmune affection in patients genetically predisposed. HLA B28 and DR3 were observed in case of association of sclerosing cholangitis with UC [13]. In case of Crohn's disease, the presence of papilledema secondary to inflammation of the duodenal mucosa may increase the risk of acute pan-

creatitis, however, this theory could not be mentioned in ulcerative colitis [3,14]. A study of Stokers *et al.* showed an elevated auto antibodies directed against the pancreas in 40% of patients with Crohn's disease and a high prevalence of these autoantibodies in first-degree relatives of patients with chronic inflammatory bowel disease [15]. Our patient had no significant medical history, presented no risk factor for acute pancreatitis and had no history of recent drug taking including those described above. In the various cases reported in the literature, the occurrence of pancreatitis precedes the onset of ulcerative colitis by a mean interval of 7 months [1 - 12 months]. The UC can be mild or severe, limited to the rectum or spread to the entire colon. The association of acute pancreatitis with UC is characterized by the presence of mild symptoms, moderate elevation of pancreatic enzymes, the absence of abnormalities in pancreatic imaging, sometimes a narrowing of the pancreatic duct at the endoscopic retrograde cholangiopancreatography and favorable prognosis of the pancreatitis [16].

4. CONCLUSION

Idiopathic acute pancreatitis may be the mode of revelation of UC, however, this association is rare, and its pathophysiology is poorly understood. The case of our patient remains the first one we found among 400 cases of ulcerative colitis diagnosed in our service.

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