

## Crohn's disease presenting with acute pancreatitis

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### Abstract

Crohn's disease is often accompanied by extraintestinal inflammation. Acute pancreatitis can be a rare manifestation of Crohn's disease. The present report describes a patient who developed two episodes of pancreatitis before the diagnosis of Crohn's disease. Clinical and laboratory evaluation excluded other causes of pancreatitis, confirming a direct association of the pancreatitis with Crohn's disease. This case report supports the hypothesis that acute pancreatitis may precede the clinical manifestations and diagnosis of the underlying inflammatory bowel disease. (*Acta gastroenterol. belg.*, 2014, 77, 357-358).

**Key words** : pancreatitis, M. Crohn, extraintestinal manifestations

### Introduction

Crohn's disease and ulcerative colitis are chronic inflammatory bowel diseases (IBD). Although the main inflammation is found at the level of the gastrointestinal tract, extra intestinal-manifestations are common (1). The most prevailing are rheumatic with peripheral arthritis and spondylarthropathy, dermatologic with erythema nodosum, ophthalmologic and hematologic. Rare manifestations are primary sclerosing cholangitis, that is found most frequently in association with ulcerative colitis ; osteoporosis, lung disorders and trombo-embolic events.

Although most cases of acute pancreatitis in IBD are medication-related or attributed to gallstones, cases of idiopathic pancreatitis (acute and chronic) are reported and attributed to the disease itself (2-4).

Although the incidence in the literature varies, there is agreement that it occurs more often in IBD than in the general population with a higher incidence in Crohn's disease than in ulcerative colitis (1). A recent review also suggests that it is more frequent in children than in adults (5). Acute pancreatitis in patients with IBD seems to be self-limited and similar in severity to that in the general population (6).

Most of the time, acute pancreatitis is diagnosed after the diagnosis of IBD has been performed. Very few case reports exist where acute pancreatitis presents before the diagnosis of IBD (7).

We describe a case of a young patient with Crohn's disease in which acute pancreatitis developed before the establishment of the diagnosis of the underlying Crohn's disease.

### Case report

A 19 year old male patient was admitted to our emergency department complaining of severe epigastric pain since one week, radiating to the back. The patient had nausea and avoided eating. During physical examination, the epigastrium was very sensitive. Temperature was slightly elevated (37,6°).

Serum lipase was normal at admission (251 U/I, normal value < 300 U/I) but a computed tomography of the abdomen showed an enlarged and oedematous pancreas surrounded by fluid. Lipase levels increased during hospitalisation to 635 U/I. No pseudo cysts or abscesses were noted.

Complete investigation aiming at discovering a possible cause for acute pancreatitis was performed. Abdominal ultrasound ruled out gallstones or dilatation of the bile ducts and MRI pancreas was normal. Liver tests, serum calcium, triglycerides and IgG4 levels were between normal values. Anamnesis was negative for medication, drugs or trauma.

The patient was treated with intravenous fluid replacement and pain medication. The course of the pancreatitis was uneventful and the patient was discharged after 5 days of hospitalisation. Serum lipase at discharge was 34 U/I.

Nevertheless 15 days later the patient returned to the emergency with the same complaints. His lipase level was 178 U/I at admission but increased to 664 U/I. Again there was an uneventful recovery after 3 days.

A few weeks later the patient developed diarrhea. This diarrhea remained for a few weeks. Several cultures of stool were negative. Biochemical investigation showed iron-deficiency anemia. Endoscopic work up with upper gastrointestinal endoscopy was negative. Because the patient refused initially further endoscopic investigation, a video capsule was performed. This showed a pathological aspect of the terminal ileum.

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The patient finally accepted to have a colonoscopy which confirmed the ileitis and showed additionally an aftoid colitis and a deep infected anal fissure, all together very suspicious for Crohn's disease. This was histologically confirmed (granuloma, crypt abscesses). The enteroscopy showed severe erosive jejunitis with on histology an aspecific, active inflammation.

Therapy was started with imuran 50 mg two times daily and budenofalk 3 mg three times a day. Diarrheal episodes decreased quickly and after two weeks of therapy the patient had no complaints any more.

During the follow up of 18 months after starting the therapy for Crohn's disease, the patient remained free of symptoms of acute pancreatitis and with normal pancreatic enzyme levels.

## Discussion

It is known that acute pancreatitis can be an extra-intestinal manifestation of Crohn's disease. The onset of pancreatitis can be before, during or after the diagnosis of IBD. However, the presentation of acute pancreatitis before the diagnosis of Crohn's disease is relatively rare (5,7-9). In our patient there were even post-hoc no specific signs of Crohn's disease on the computed tomography of the abdomen. The largest case series comes from Israel where 20 patients were retrospectively identified (5). Patients are usually young with a short and uneventful evolution of the pancreatitis. The time between the onset of the pancreatitis and the Crohn's disease varies between a few months and one year or more.

The pathogenesis of pancreatitis remains unexplained. The possibility of reflux of duodenal contents into the pancreatic ducts (due to ampullary or mucosal damage) as well as the presence of duodenal-pancreatic duct fistulas or flow obstruction (due to ampullary involvement) has been reasoned to account for the pancreatitis in cases with duodenal Crohn's disease (10).

Autoantibodies against the exocrine pancreas have been suggested to explain the occurrence of pancreatitis in cases without duodenal involvement (10). However the clinical significance of these autoantibodies in IBD is not clear. There is a prevalence of 27% to 39% for autoantibodies against exocrine pancreas in patients with Crohn's disease (1). Similarities of acute and chronic pancreatitis in Crohn's disease with the entity of autoimmune pancreatitis enhance a possible role for autoantibodies in initiating or potentiating pancreatic injury in patients with Crohn's disease (1). However, many studies have not found an association between autoantibodies against exocrine pancreas and pancreatitis in Crohn's disease (1).

There are some difficulties with the diagnosis and the management of pancreatitis in IBD patients which could lead to overestimation of pancreatitis. Pancreatic enzymes may be elevated in IBD patients without pancreatitis. Hyperamylasemia was found in 11% and hyperlipasemia in 7% in a cohort of 239 patients with IBD (11). High

levels of serum amylase were associated with extensive Crohn's disease and high histologic activity (11).

Furthermore, a lot of medications can induce pancreatitis and it is often difficult to establish a causal role for medications in the pathogenesis of pancreatitis (1).

Another difficult differential diagnosis is autoimmune pancreatitis (1). A biochemical hallmark of autoimmune pancreatitis is raised serum levels of IgG4 (12), but the sensitivity and specificity of this test is low (13). In our case the levels of IgG4 were normal, but in previous cases these levels were not always mentioned (5,7-9). However the differential diagnoses between pancreatitis in Crohn's disease and autoimmune pancreatitis type 2 especially is extremely difficult. There are also no raised levels of IgG4, so a definitive diagnosis can only be made by histology of the pancreas.

In our case, a mutation in the CFTR gene was not ruled out. However the clinical evolution of the patient argues against the presence of this mutation, due to the lack of recurrence after initiating the treatment of the IBD.

In conclusion, this case report supports the hypothesis that acute pancreatitis can precede the clinical manifestations and diagnosis of the underlying IBD.

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