

# Medical rehabilitation of patients with chronic pancreatitis complicated by irritable bowel syndrome at inpatient stage

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## ABSTRACT

Pancreatic abnormalities are common in inflammatory bowel disease (IBD) patients and represent a heterogeneous group of conditions that include acute pancreatitis, chronic pancreatitis, autoimmune pancreatitis and asymptomatic abnormalities. We sought to review the available evidence concerning the aetiology, clinical presentation, diagnosis and treatment of pancreatic conditions in IBD patients. A PubMed/Medline query was conducted addressing pancreatic disorders in IBD. Reference lists from studies selected were manually searched to identify further relevant reports. Relevant manuscripts about pancreatic disorders in patients with IBD were selected and reviewed. Thiopurines and gallstones are the most frequent causes of acute pancreatitis in IBD patients. Thiopurine-induced acute pancreatitis is usually uncomplicated and self-limited. Some evidence suggests that chronic pancreatitis may be more common in IBD. Most cases are idiopathic, affecting young males and patients with ulcerative colitis. Autoimmune pancreatitis is a relatively newly recognized disease and is increasingly diagnosed in IBD, particularly for type 2 autoimmune pancreatitis in ulcerative colitis patients. Asymptomatic exocrine insufficiency, pancreatic duct abnormalities and hyperamylasaemia have been identified in up to 18% of IBD patients, although their clinical significance and relationship with IBD remain undefined. The wide spectrum of pancreatic manifestations in IBD is growing and may represent a challenge to the clinician. A collaborative approach with a pancreas specialist may be the most productive route to determine aetiology, guide additional diagnostic workup, illuminate the aetiology and define the treatment and follow-up of these patients.



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## 1. Introduction

Inflammatory bowel disease (IBD) is an idiopathic chronic and recurrent condition that comprises Crohn's disease (CD) and ulcerative colitis (UC). Its pathogenesis involves a complex interaction between genetic susceptibility traits, environmental factors and intestinal microflora, which leads to an abnormal and

excessive immune response, compromised epithelial barrier function and, ultimately, gastrointestinal tract inflammation and tissue damage [1]. Being a multisystemic disease, it may affect many organs. Extraintestinal manifestations, defined as complications occurring distant from the bowel, are reported in 21–47% of IBD patients [2] and will be extensively reviewed in the upcoming First European Evidence-Based Consensus on Extra-Intestinal Manifestations in Inflammatory Bowel Disease, to be published in this journal. Pancreatic abnormalities in IBD patients are common and represent a heterogeneous group of conditions [3], [4] including acute pancreatitis (AP), chronic pancreatitis (CP), autoimmune pancreatitis (AIP), asymptomatic exocrine insufficiency, enzyme elevations and imaging abnormalities [5–7]. Since the last review on IBD and pancreatitis was published in 2010 [6] new information has emerged, particularly regarding thiopurine-associated pancreatitis and AIP. Hence, we sought to prepare an updated review of the spectrum of pancreatic disorders in patients with IBD.

## 2. Methods

**Methods** We performed a broad literature search to identify relevant studies addressing pancreatic disorders in IBD. PubMed and Medline were searched up to May 2015, using the keywords ‘IBD’, ‘UC’ and ‘CD’ combined with ‘AIP’, ‘AP’, ‘CP’, ‘idiopathic pancreatitis’, ‘druginduced pancreatitis’, ‘exocrine pancreas insufficiency’ or ‘pancreatic autoantibodies’. Articles in English, French, Portuguese and Spanish were reviewed. Articles reporting on the clinical presentation, diagnosis, treatment and outcome of pancreatic diseases and silent pancreatic abnormalities in IBD were selected and reviewed. Moreover, a manual search of the reference list of initially selected articles was conducted. Articles published only as abstracts were excluded.

## 3. Conclusions

Acute and chronic pancreatitis may complicate the course of IBD. The most common causes of AP are thiopurines and gallstones. The course of thiopurine-induced AP is usually uncomplicated and self-limited. New genetic markers for thiopurine-induced pancreatitis are being identified and in the future may prove to be a useful tool in selecting patients for this therapy. Most cases of CP are idiopathic and some authors have suggested that this condition may be an extraintestinal manifestation of IBD. However, the evidence is scarce and restricted to case reports and case series. In addition, exocrine dysfunction and pancreatic duct abnormalities have been identified in up to 18% of asymptomatic IBD patients. Although exocrine pancreatic insufficiency seems to be the most common pancreatic manifestation in IBD, its clinical significance remains undefined. Autoimmune pancreatitis is a relatively recently recognized entity that should be considered in the differential diagnosis of pancreatitis, especially among IBD patients, since up to 27% of AIP patients also have IBD, mostly UC. The wide spectrum of pancreatic manifestations and severity in patients with IBD may represent a challenge to the clinician, particularly in the setting of asymptomatic abnormalities and CP, idiopathic or recurrent pancreatitis. In these situations a collaborative approach with a pancreas specialist may be the most productive way to decide additional diagnostic workup, illuminate the aetiology and define the follow-up of these patients.

## 4. References

[1] Xavier RJ, Podolsky DK. Unravelling the pathogenesis of inflammatory bowel disease. *Nature* 2007; 448:427–34.

[2] Ott C, Schölmerich J. Extraintestinal manifestations and complications in IBD. *Nat Rev Gastroenterol Hepatol* 2013; 10:585–95.

[3] Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*

2013; 144:1252–61.

[4] Ueki T, Kawamoto K, Otsuka Y, et al. Prevalence and clinicopathological features of autoimmune pancreatitis in Japanese patients with inflammatory bowel disease. *Pancreas* 2015; 44:434–40.

[5] Navaneethan U, Shen B. Hepatopancreatobiliary manifestations and complications associated with inflammatory bowel disease. *Inflamm Bowel Dis* 2010; 16:1598–619.

[6] Pitchumoni CS, Rubin A, Das K. Pancreatitis in inflammatory bowel diseases. *J Clin Gastroenterol* 2010; 44:246–53.

[7] Jasdanwala S, Babyatsky M. Crohn's disease and acute pancreatitis. A review of literature. *J Pancreas* 2015; 16:136–42.

[8] Bradley EL 3rd. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. *Arch Surg* 1993; 128:586–90.

[9] Braganza JM, Lee SH, McCloy RF, McMahon MJ. Chronic pancreatitis. *Lancet* 2011; 377:1184–97.

[10] Yadav D, Whitcomb DC. The role of alcohol and smoking in pancreatitis. *Nat Rev Gastroenterol Hepatol* 2010; 7:131–45.

[11] Hamada S, Masamune A, Kikuta K, Hirota M, Tsuji I, Simosegawa T. Nationwide epidemiological survey of autoimmune pancreatitis in Japan. *Pancreas* 2014; 43:1244–8.

[12] Shen HN, Lu CL, Li CY. Epidemiology of first-attack acute pancreatitis in Taiwan from 2000 through 2009. *Pancreas* 2012; 41:696–702.