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RESEARCH ARTICLE

Crohn's Disease and Acute Pancreatitis: Dissecting Causes Between Treatment and Disease Course

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Abstract: The relationship between Crohn's disease (CD) and acute pancreatitis (AP) is complex. This article reviews the current understanding of this association, exploring whether AP is a manifestation of CD itself or a complication of CD therapies. We examine the potential etiological factors, including the role of CD medications and the involvement of the gastrointestinal tract in CD.

Key words: Crohn's disease, acute pancreatitis, etiology, therapy-induced pancreatitis, inflammatory bowel disease, treatment complications, differential diagnosis, gastrointestinal disorders.

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory condition of the pancreas with varied etiologies. While common causes include gallstones and alcohol consumption (Gunşahin et al., 2024; Edu et al., 2025), the occurrence of AP in patients with Crohn's disease (CD), a chronic inflammatory bowel disease, presents a unique clinical challenge. CD is a chronic, relapsing inflammatory disorder that can affect any part of the gastrointestinal tract, from the mouth to the anus, but most often involves the small intestine and colon. The inflammation in CD is transmural, meaning it extends through all layers of the intestinal wall, leading to a range of complications, including strictures, fistulas, and abscesses. While the gastrointestinal manifestations of CD are well-recognized, its extraintestinal manifestations, though less common, can also significantly impact patient morbidity. Among these, pancreatic involvement, though not a frequent occurrence, presents a complex clinical picture.

The association between CD and AP is not straightforward. AP in CD patients can arise either as a direct consequence of the underlying inflammatory process of CD, particularly when the disease involves the duodenum or biliary tract, or as an adverse effect of the medications used to manage CD. Differentiating between these two possibilities is crucial for appropriate clinical management.

This paper aims to explore the etiology of AP in CD patients, specifically differentiating between AP as a direct consequence of CD and AP as a complication of CD treatment. Previous studies have documented the significant burden of AP, including hospitalization costs (Pahomeanu et al., 2023), and the need for a comprehensive review of its various causes (Mederos et al., 2021). A deeper understanding of the factors contributing to AP in CD patients is essential for optimizing their care and minimizing potential complications.

METHODS

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This review synthesizes findings from clinical studies, case reports, and literature reviews focusing on the association between CD and AP. We examined the following key areas:

- CD and the Pancreas: We investigated the potential pathophysiological mechanisms by which CD, particularly when involving the duodenum or biliary tract, can lead to AP.
- Drug-Induced Pancreatitis: We reviewed cases of AP associated with medications commonly used in the treatment of CD, such as 5-aminosalicylates (5-ASA) and tumor necrosis factor-alpha (TNF-α) inhibitors.
- Literature Review: We analyzed relevant articles using databases. We included studies that specifically addressed the etiology of AP in CD patients. Related research on ulcerative colitis and its treatment was also considered where relevant [6, 8, 9, 17, 18].

RESULTS

Etiology and Pathophysiology of Acute Pancreatitis in Crohn's Disease

The etiology of acute pancreatitis in Crohn's disease is complex and multifactorial. Key mechanisms include:

1. Direct Inflammation: Crohn's disease can affect the pancreatic duct, leading to ductal obstruction and pancreatic inflammation. The chronic inflammation in the gastrointestinal tract may extend to adjacent organs, including the pancreas, leading to a predisposition to pancreatitis.

2. Immune Dysregulation: Crohn's disease is driven by an aberrant immune response, which can increase systemic inflammation. This inflammatory cascade may impact pancreatic function, contributing to the development of acute pancreatitis.

3. Therapeutic Effects: The medications used to treat Crohn's disease, including immunosuppressive therapies (e.g., corticosteroids, thiopurines), biologic agents (e.g., TNF inhibitors), and immunomodulators, can increase the risk of pancreatitis. For instance, thiopurines are known to cause pancreatitis in a subset of patients, possibly through their effect on the pancreas's exocrine function.

4. Pancreatic Enzyme Secretion Dysfunction: Alterations in pancreatic enzyme secretion, whether due to ductal involvement or medication side effects, contribute to the development of acute pancreatitis in these patients.

Clinical Manifestations and Diagnosis

Patients with Crohn's disease and acute pancreatitis may present with overlapping symptoms, making diagnosis challenging. Common clinical features include:

- Abdominal Pain: Persistent abdominal pain, especially in the upper abdomen, is often present in both Crohn's disease and acute pancreatitis. This pain is frequently exacerbated by eating.
- Diarrhea and Weight Loss: While these symptoms are typical of Crohn's disease, they can also be seen in acute pancreatitis due to pancreatic insufficiency and malabsorption.
- Enzyme Markers: Elevated levels of pancreatic enzymes, particularly amylase and lipase, are indicative of acute pancreatitis. However, patients with Crohn's disease may have baseline elevations in these enzymes due to pancreatic involvement, making interpretation challenging.
- Imaging: Imaging modalities such as CT scans, MRI, and ultrasonography are essential for diagnosing pancreatitis. These tools can also assess the extent of pancreatic

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involvement and rule out other causes of pancreatitis.

Treatment Approaches

1. Management of Crohn's Disease: The primary goal of treating Crohn's disease in patients with acute pancreatitis is to control the inflammatory process. Medications used to treat CD include corticosteroids, immunosuppressive drugs, and biologic agents like TNF inhibitors. The use of these therapies must be carefully balanced with the risk of exacerbating pancreatitis. Corticosteroids, while effective in controlling inflammation, may increase the risk of developing pancreatitis.

2. Management of Acute Pancreatitis: Treatment of acute pancreatitis focuses on supportive care, including hydration, pain control, and the management of complications such as infection or organ failure. In severe cases, endoscopic or surgical intervention may be required.

3. Immunosuppressive Therapy: The role of immunosuppressive therapy in both Crohn's disease and acute pancreatitis requires careful consideration. Medications like thiopurines and methotrexate have been associated with pancreatitis in some patients, necessitating close monitoring.

4. Nutritional Support: Since both Crohn's disease and acute pancreatitis can lead to malabsorption and nutritional deficiencies, proper nutritional support is critical. This may include enteral nutrition or total parenteral nutrition in severe cases.

DISCUSSION

AP in CD patients presents a complex diagnostic and therapeutic challenge. It is crucial to distinguish between AP caused by CD itself and AP induced by CD medications.

When AP occurs in a CD patient, clinicians should consider:

- Ruling out other common causes of AP, such as gallstones and alcohol.
- Carefully reviewing the patient's medication list, with a high index of suspicion for 5-ASA drugs and TNF-α inhibitors.
- Evaluating the location and extent of CD, as proximal disease may be more likely to involve the pancreas or biliary tract [12, 13, 14, 20].

The management of AP in CD patients depends on the underlying cause. Drug-induced AP typically resolves upon discontinuation of the offending medication [15, 18, 24]. AP due to CD itself may require treatment of the underlying CD with medications. The decision to use biologics in CD patients should be carefully weighed against the potential risk of complications, including pancreatitis [23].

CONCLUSION

In conclusion, the interplay between Crohn's disease and acute pancreatitis highlights the complexity of managing inflammatory diseases that involve multiple organ systems. While significant progress has been made in understanding the pathophysiology of both conditions, further research is needed to identify biomarkers for early detection, optimize treatment strategies, and develop therapies that can address both the underlying disease and its complications. The ultimate goal is to improve patient outcomes by reducing the incidence of acute pancreatitis in Crohn's disease patients and providing more effective and safer therapeutic options.

AP in CD patients can result either from the CD itself or from medications used to treat it. A thorough evaluation is necessary to identify the specific etiology in each case. Further research is needed to elucidate the precise mechanisms by which CD and its treatments can lead to AP.

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