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Case Report

Adalimumab-Associated Acute Pancreatitis in a Patient with Ulcerative Colitis: A Case Report

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Abstract

Background: Drug-induced acute pancreatitis (DIAP) is an uncommon etiology of acute pancreatitis and remains a diagnosis of exclusion. Tumor necrosis factor-alpha (TNF-α) inhibitors are widely used in inflammatory bowel disease, but acute pancreatitis has only rarely been reported with these agents.

Case Presentation: We describe a 30-year-old man with pancolonic ulcerative colitis treated with adalimumab who developed acute pancreatitis during maintenance therapy. The patient presented with intermittent epigastric pain associated with a marked elevation of serum lipase. Cross-sectional imaging and magnetic resonance cholangiopancreatography showed no pancreatic or biliary abnormalities. A comprehensive etiological workup excluded gallstone disease, alcohol use, metabolic disorders, and autoimmune pancreatitis. Clinical and biochemical improvement occurred after discontinuation of adalimumab.

Conclusion: This case supports adalimumab as a rare but possible cause of acute pancreatitis. Awareness of this adverse event and a structured causality assessment are essential to guide management and prevent recurrence.

Keywords: Ulcerative Colitis; Adalimumab; Anti-TNF; Drug-induced pancreatitis

Introduction

Acute pancreatitis is one of the most frequent gastrointestinal emergencies worldwide, with gallstones and alcohol accounting for the majority of cases. Drug-induced acute pancreatitis (DIAP) is considerably less common, representing less than 5% of reported cases, but its true incidence is likely underestimated due to diagnostic difficulties and underreporting [1]. The absence of specific clinical or radiological features makes DIAP a challenging diagnosis, particularly in patients with complex medical histories [1,2].

The diagnosis of DIAP relies on several key principles: a compatible temporal relationship between drug exposure and symptom onset, exclusion of other established etiologies, objective confirmation of pancreatitis, and improvement after drug withdrawal. Recurrence after rechallenge, although rarely performed, provides the strongest evidence for causality [2].

Tumor necrosis factor-alpha (TNF-α) inhibitors are widely used in the treatment of inflammatory bowel disease and have significantly improved disease outcomes. Adalimumab, a fully human monoclonal antibody targeting TNF-α, is approved

for moderate to severe ulcerative colitis and is generally considered safe. However, rare cases of acute pancreatitis associated with anti–TNF-α therapy have been reported [3,4].

We present a case of adalimumab-induced acute pancreatitis occurring during maintenance therapy.

Case Presentation

A 30-year-old patient was diagnosed 1 year ago with pancolonic ulcerative colitis (Montreal classification E3) after presenting with more than 10 bloody and mucus-containing stools per day, diffuse abdominal pain, and rectal tenesmus. He had no past medical history, no family history of inflammatory bowel disease or pancreatitis, and no history of tobacco, alcohol, or recreational drug use. His body mass index was 17 kg/m^2, indicative of undernutrition linked to the illness.

Initial investigations revealed a markedly elevated fecal calprotectin level (1003 µg/g). Colonoscopy revealed inflammatory colitis consistent with ulcerative colitis, and histopathological examination showed ulcerated congestive inflammatory colitis with mild epithelial abnormalities.

The patient was initially treated with azathioprine, which was discontinued after one year because of hematologic toxicity . Therapy was therefore escalated to adalimumab, following the standard induction regimen (160 mg subcutaneously at week 0 ; 80 mg at week 2 ; then 40 mg every 2 weeks as maintenance therapy).

Three months later, the patient was hospitalized for a severe acute flare of ulcerative colitis while receiving adalimumab, requiring intravenous antibiotics and high-dose corticosteroid therapy. Clinical and biological improvement was rapidly achieved.

While receiving maintenance adalimumab therapy, three days after the most recent injection, the patient developed severe epigastric pain radiating to the back. Serum lipase was markedly elevated at 1417 U/L on day 1 and subsequently decreased to 811 U/L on day 2 more than three times the normal value . Contrast-enhanced abdominal computed tomography (Figure 1) showed a normal pancreatic morphology without peripancreatic inflammation or fluid collections, corresponding to Balthazar stage A acute pancreatitis. No biliary duct dilation was observed.

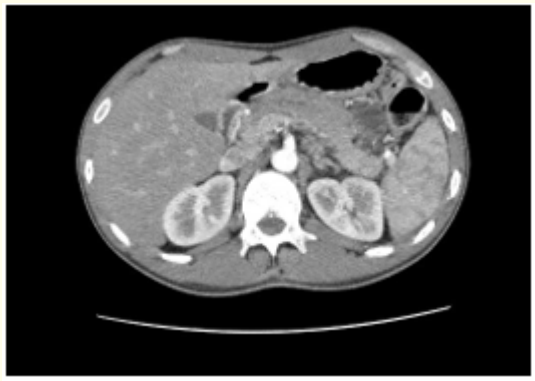

Figure 1: Axial contrast-enhanced computed tomography image showing a normal pancreatic morphology without peripancreatic fat stranding or fluid collections, consistent with Balthazar stage A acute pancreatitis.

A comprehensive etiological workup was unremarkable. There was no alcohol consumption. Liver function tests were non-cholestatic (total bilirubin 3.4 mg/L, AST 19 U/L, ALT 12 U/L). Serum triglyceride (0.69 g/L) and calcium levels were within normal ranges. Autoimmune testing, including serum IgG4 (0.320 g/L), was negative. Pancreatic magnetic resonance imaging was performed and showed no biliary or pancreatic abnormalities (Figure 2).

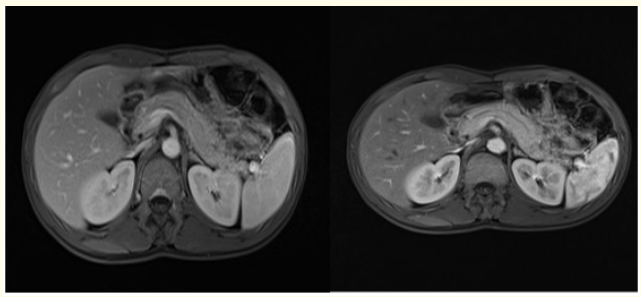

Figure 2: Axial T2-weighted abdominal magnetic resonance image showing a normal pancreatic parenchyma normal pancreatic ducts, and no evidence of gallstones or biliary obstruction.

Abdominal pain gradually subsided once adalimumab was stopped. There was no symptom recurrence since the patient did not receive the planned follow-up injection.

Following a multidisciplinary inflammatory bowel disease team discussion, the decision was made not to reintroduce adalimumab and to switch to a biologic agent with a different mechanism of action. The patient was subsequently started on ustekinumab, and no adverse effects, including epigastric pain, were reported after the first injection, with no recurrence of acute pancreatitis during follow-up.

Discussion

Drug-induced acute pancreatitis (DIAP) has been recognized for several decades as an uncommon cause of pancreatic inflammation . Since the first reports in the mid-20th century, an increasing number of commonly prescribed medications across various therapeutic classes have been implicated in pancreatic injury. Although drug-induced acute pancreatitis is considered uncommon, its reported incidence ranges from approximately 0.1% to 2% of all acute pancreatitis cases, and the true burden is likely underestimated due to underreporting and diagnostic challenges [5,6].

There are more than 120 drugs that have been implicated in causing AP with different mechanism of injury reported in the literature [2].

Furthermore, pharmacovigilance data from the World Health Organization list over 500 drugs for which acute pancreatitis has been reported as a potential adverse effect [7].

Drug-induced AP (DIAP) is a rare entity that often is challenging for clinicians [2]. It is based on confirming acute pancreatitis, primarily through clinical features and elevated serum lipase. Then , common etiologies such as gallstones and alcohol must be excluded through careful review of the patient's history, medication exposure, laboratory tests (liver enzymes, triglycerides, and calcium), and contrast-enhanced abdominal computed tomography, which is essential to rule out biliary obstruction or structural pancreatic disease. In the absence of clinical, biochemical, or radiological evidence of choledocholithiasis, invasive procedures such as ERCP are not indicated [8].

An association between pancreatic disorders and inflammatory bowel disease (IBD) has long been described, with early observations reported as early as the 1950s and 1960s. Although autoimmune pancreatitis (AIP) remains rare among patients with IBD, its prevalence is higher than in the general population and appears to be more strongly associated with ulcerative colitis than with Crohn's disease [9,10]. For this reason, AIP should be considered as a differential diagnosis in patients with ulcerative colitis presenting with acute pancreatitis.

In our patient, acute pancreatitis was most likely related to exposure to tumor necrosis factor-alpha (TNF-α) inhibitors after exclusion of common etiologies, including biliary disease, alcohol consumption, hypertriglyceridemia, and hypercalcemia. Autoimmune pancreatitis was considered but deemed unlikely based on several elements: serum IgG4 levels were within normal limits, cross-sectional imaging did not demonstrate features suggestive of autoimmune pancreatitis, and pancreatic symptoms resolved rapidly without the need for corticosteroid therapy. Taken together, these findings strongly argue against both type 1 and type 2 autoimmune pancreatitis [9].

Anti–TNF-α agents have transformed the management of inflammatory bowel disease but are associated with rare immune-mediated adverse events [4]. Acute pancreatitis has been reported with TNF-α inhibitors, including infliximab and adalimumab, although the number of published cases remains limited [3,4,11].

The pathophysiological mechanisms underlying this association are not fully understood and are likely multifactorial. Proposed mechanisms include idiosyncratic immune reactions, pancreatic duct constriction, microvascular injury such as arteriolar thrombosis, direct or indirect toxic effects on pancreatic tissue, and localized pancreatic angioedema [2,6].

Our observation is comparable to the case reported by Werlang., *et al*. who described acute pancreatitis induced by infliximab in a patient with ulcerative colitis, with recurrence after switching to adalimumab, suggesting a class effect among TNF-α inhibitors [4]. Similarly, El Ouardi., *et al*. a case of adalimumab-induced acute pancreatitis, further supporting the association between anti–TNF-α therapy and pancreatic injury [11].

These reports suggest that switching from one TNF-α inhibitor to another may expose patients to a risk of recurrent acute pancreatitis. From a therapeutic standpoint, recognition of

TNF-α inhibitor–associated pancreatitis therefore has important implications. Rather than reintroducing another agent within the same class, changing to a biologic therapy with a different mechanism of action appears to be a safer strategy. In our case, we opted to switch directly to a different therapeutic class, which was associated with a favorable outcome and no recurrence of pancreatitis during follow-up.

Conclusion

Acute pancreatitis represents a rare adverse event associated with anti–TNF-α therapy. In this case, the close temporal relationship between adalimumab exposure and symptom onset, the exclusion of alternative etiologies, and the sustained clinical remission after drug withdrawal and switching to a different biologic class support a drug-induced mechanism. Early recognition and prompt discontinuation of the offending agent are essential to prevent recurrence and to guide safe therapeutic alternatives.

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