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When the Medication Inflames the Pancreas: Acute Pancreatitis Under Sulfasalazine

A. Alaoui[1*], Z. Ztati[1], S. Sbihi[1], H. Aouroud[1,2], O. Nacir[1], F. Lairani[1], A. Ait Errami[1], S. Oubaha[1,2], Z. Samlani[1], K. Krati[1]

[1]Gastroenterology Department, Mohammed VI University Hospital, Marrakech, Morocco
[2]Physiology Laboratory, Faculty of Medicine and Pharmacy of Marrakech, Morocco

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***Corresponding author:** A. Alaoui
Gastroenterology Department, Mohammed VI University Hospital, Marrakech, Morocco

Abstract **Case Report**

Drug involvement in triggering acute pancreatitis (AP) is a rare occurrence (less than 2%) [1] but not exceptional in adults [2]. We report here a case of recurrent acute pancreatitis secondary to 5-aminosalicylic acid, with a positive provocation test.

Keywords: pancreatitis, aminosalicylate derivatives, sulfasalazine, inflammatory bowel disease, Case report.

INTRODUCTION

Acute pancreatitis is a serious disease with high mortality [3], the most common etiologies of which are gallstones, alcoholism, and metabolic causes [4-5]. Drug-induced pancreatitis is a rare entity, the prevalence of which remains difficult to assess due to challenges in determining causality [6]. Aminosalicylate (5-ASA) derivatives, including sulfasalazine, represent a safe and effective treatment for patients with inflammatory bowel disease (IBD). Pancreatitis has been described as a complication of 5-ASA treatment in patients with IBD. In a critical review, Mallory and Kern suggested that the sulfonamide component of sulfasalazine was responsible for this adverse effect due to the structural similarity of sulfonamides to thiazide diuretics, which are a well-known cause of drug-induced pancreatitis. Sulfasalazine comprises sulfapyridine linked by an azo bond to 5-aminosalicylate (5-ASA), which is the active ingredient.

OBSERVATION

We report the case of a 29-year-old female patient who underwent cholecystectomy for a multilithiasic gallbladder. She was being treated for ulcerative colitis of undetermined extent, which presented with a moderate flare-up. She was treated with sulfasalazine 4g/day and initially responded well. Two weeks later, the patient presented to the emergency department with severe epigastric pain radiating to the back, relieved by leaning forward. She was also experiencing vomiting that had been present for three days prior to admission. Her lipase level was 17 times the upper limit of normal. The patient was then admitted to the gastroenterology department for further management.

During her hospitalization, the patient underwent an abdominal ultrasound which showed a normal-appearing pancreas and normal-caliber bile ducts without any evidence of gallstones. Acute pancreatitis was classified as stage C according to the Balthazar CT scan score, with no abnormalities of the pancreatic duct or bile ducts. Laboratory tests revealed normal electrolytes, liver enzymes, calcium, and triglycerides.

Other etiologies have been ruled out, including infectious (HBV, HCV serologies) or tuberculosis (tuberculin skin test, Gene-Expert in sputum and negative QuantiFERON) and autoimmune (ANA, IgG4 levels).

Therapeutically, 5-ASA was stopped, with cessation of feeding and parenteral rehydration leading to complete resolution of symptoms.

After resolution of symptoms, a provocation test was performed with the patient's consent. After receiving 2g/day of Sulfasalazine, typical pancreatic pain reappeared with a lipase level of 11N and an ultrasound showing a swollen pancreas. Therefore, 5-ASA was discontinued with complete resolution of symptoms.

For maintenance treatment, the patient was started on azathioprine at a dose of 2.5 mg/kg/day with good progress.

DISCUSSION

Crohn 's disease with duodenal involvement [2]. Persistent asymptomatic hyperamylasemia has been described in patients with Crohn 's disease, likely reflecting pancreatic duct strictures.

Epidemiological data suggest that the risk of pancreatitis is highest with mesalazine (hazard ratio [HR] 3.5), azathioprine (HR 2.5), and simvastatin (HR 1.8) [9] . Drug-induced pancreatitis is classified (class Ia, Ib , II, III, IV) based on the number of reported cases, the demonstration of a consistent latency period (time between drug initiation and the development of pancreatitis), and the response to a provocation test [10].

Class 1 drugs were subdivided into Ia and Ib. Class Ia includes at least one documented case after re-exposure, excluding all other causes such as alcohol, gallstones, hypertriglyceridemia, and other medications. Class Ib drugs are similar to Class Ia. However, in this case, the potential causes of panthrosis were neither ruled out nor clearly present. Class II drugs include at least four cases reported in the literature as belonging to this group, where latency persists in at least 75% or more of the reported cases. Class I and II drugs have the highest potential to cause panthrosis. Class III drugs are weaker than the two previous classes and lack consistent latency or retest data. Finally, Class IV drugs include those that do not fit into the other classes mentioned and have a single case report published in the medical literature, without retest data. Latency periods were classified as short (latency <24 hours), intermediate (latency 1 to 30 days), and long (latency >30 days) [10 , 11]. Symptoms must resolve after drug discontinuation to classify the episode of acute pancreatitis as drug- induced [10 , 12] .

Finally, cases of PAA have been reported after both short-term and long-term exposure to 5-ASA derivatives. One study reported two cases of panthesia. Proven pro-inflammatory bowel disease (PID) occurring 2 and 14 days, respectively, after the initiation of oral 5-aminosalicylic acid therapy for inflammatory bowel disease [13]. Other studies have reported that PID can also occur after prolonged exposure to 5-ASA derivatives in some cases [14] .

This patient clearly developed acute symptoms of pancreatitis on two separate occasions temporally related to 5-ASA treatment. Serum lipase was elevated in both cases, and the pancreas was found to be inflamed by ultrasound. The symptoms and elevated serum lipase were reproduced by 5-ASA administration. A small but significant amount of 5-ASA is absorbed in the terminal ileum and colon. Previously reported episodes of sulfasalazine- induced pancreatitis have been attributed to sulfapyridine, due to the much higher blood levels of sulfapyridine compared to other 5-ASAs. However, we suggest that 5-ASA could indeed be responsible for acute pancreatitis, despite low circulating levels.

CONCLUSION

5-ASA is a first-line treatment for ulcerative colitis. Acute pancreatitis (AP) secondary to this treatment remains rare compared to thiopurines [8]. This diagnosis should be considered in any patient receiving this type of treatment who presents with pancreatic-type epigastric pain after ruling out other causes of AP.

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