

CASE REPORT



Why now? Delayed drug-induced pancreatitis due to dapsons for dermatitis herpetiformis

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ABSTRACT

Drug-induced pancreatitis can be caused by a wide array of medications. In fact, the diagnosis is likely commonly missed due to the difficulty in diagnosing one agent as the sole cause. We present a case of dapsons-induced pancreatitis in a 75-year-old male with history of celiac disease. He presented with abdominal pain and was found to have acute pancreatitis. Interestingly, he had been on dapsons for 5 years and had no other recent medication changes, significant alcohol use, or gallbladder disease. It was determined this was an episode of delayed acute pancreatitis due to dapsons. This is a rarely addressed entity in the literature and is the first case in which pancreatitis occurred so late in a patient's treatment course on dapsons.

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1. Introduction

Acute pancreatitis accounts for over \$2 billion in healthcare costs annually [1]. While the majority of cases are caused by gallstones or alcohol, a subset are caused by drugs. Drug-induced pancreatitis is an under-recognized entity, with reported incidence only accounting for 0.1–2% of all pancreatitis cases [2]. The difficulty of diagnosing drug-induced pancreatitis cannot be explained solely by the need to rule out more common causes, but also because the list of drugs that can cause acute pancreatitis is so vast. Furthermore, the mechanism by which drug-induced pancreatitis occurs is controversial. In fact, the mechanism may differ depending on the offending agent. We present a case of dapsons-induced pancreatitis, which has only been previously described a few times in the literature.

2. Case

A 75-year-old man with history of celiac disease, dermatitis herpetiformis, hypertension, and hyperlipidemia presented to the emergency department with sharp upper abdominal pain after eating a fatty meal. He had intermittent abdominal pain for a few weeks. A few days prior to presentation, he was diagnosed with acute pancreatitis (lipase 1149) as an outpatient. Due to increased pain and anorexia, he was sent to the hospital for further evaluation. Other history included uncomplicated umbilical hernia repair 6 months prior to admission. His medications included aspirin, dapsons, furosemide 10 mg once daily, losartan, ranitidine,

and simvastatin. He had been taking dapsons for over 5 years for dermatitis herpetiformis, and his dose was increased 4 weeks prior to presentation, coinciding with his history of intermittent abdominal pain. His furosemide dose has been unchanged for years. He consumed 1–2 alcoholic beverages monthly.

On presentation, he had epigastric pain, nausea, and anorexia. He denied any vomiting, fever, or change in bowel movements. Physical exam revealed mild epigastric tenderness with hypoactive bowel sounds. Labs showed down-trending lipase and leukocytosis. Computed tomography abdomen showed moderate peripancreatic stranding consistent with pancreatitis. Triglyceride levels were normal. Right upper quadrant ultrasound revealed no cholelithiasis and no sign of cholecystitis.

The patient was managed with intravenous fluids. His home medications were continued including dapsons. On day 2, his pain and anorexia were not improving. Dapsons was discontinued. The patient clinically improved the next day and was discharged with plans to follow a strict gluten-free diet while off of dapsons. He is being closely monitored for any recurring dermatitis herpetiformis.

3. Discussion

Dapsons is a nonsulfonamide antibiotic that works similarly to a sulfonamide. It is used for a variety of disorders, including dermatitis herpetiformis, malaria, brown recluse spider bite, and *Pneumocystis jiroveci* pneumonia prophylaxis. There are three

searchable case reports of adult dapsone-induced pancreatitis in the literature. All three cases report pancreatitis within 4 months from starting dapsone [3–5]. One case described a patient who developed acute pancreatitis soon after starting dapsone for dermatitis herpetiformis. The drug was discontinued, but then resumed after the pancreatitis resolved. The patient then had a recurrence of pancreatitis [3]. A second case described a patient developing acute pancreatitis as part of a ‘sulfone syndrome’ hypersensitivity reaction [4]. The third case described a case of diabetic ketoacidosis in the setting of dapsone-induced acute pancreatitis [5].

All three cases previously described in the literature are similar in that dapsone-induced pancreatitis occurred within a couple months of initiating the drug. However, our patient had been taking dapsone for over 5 years, with the dose increased a few weeks prior to presentation, potentially triggering pancreatitis. Therefore, a proposed mechanism of cause in our patient could have been a dose-dependent reaction which has not been previously described. Our patient was also taking furosemide, which is also known to cause pancreatitis. However, the symptoms resolved with cessation of dapsone and continued use of furosemide. It is generally accepted that drug-induced pancreatitis occurs soon after the offending agent is started. However, our case illustrates delayed drug-induced pancreatitis, possibly indicating a dose-dependent reaction.

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