



Acute Alcohol-Induced Pancreatitis After Transcoronary Alcohol Ablation

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ABSTRACT

Alcohol-induced pancreatitis typically presents as acute nausea, vomiting, and abdominal pain, typically seen as a complication of chronic alcoholism or binge drinking alcoholic beverages. Here, we present a case of alcohol-induced pancreatitis from an unusual source: alcohol used in a catheter ablation for atrial fibrillation. This is the first case in current literature that has identified this adverse effect of alcohol catheter ablation.

KEYWORDS: pancreatitis; necrotizing pancreatitis; alcohol ablation; alcohol

INTRODUCTION

Alcohol is one of the most common etiologies of acute pancreatitis, accounting for approximately 17%–25% of all cases of acute pancreatitis worldwide. It is typically associated with chronic alcohol consumption or binge drinking alcoholic beverages. Alcohol causes pancreatic injury through various mechanisms including increasing oxidative stress, increasing inflammatory cytokines, and inhibiting apoptosis and enhancing necrosis. Here, we present a patient who developed acute alcohol-induced pancreatitis from an unexpected source.

CASE REPORT

A 48-year-old man with a medical history of persistent atrial fibrillation (with prior cavotricuspid isthmus radiofrequency ablation in 2019) leading to nonischemic cardiomyopathy initially presented from home for a planned cardiac ablation for recurrent atrial fibrillation. The patient received monitored anesthesia care and was given 700 mL of normal saline and 200 mg of intravenous propofol during the procedure, as well as 3 transcoronary infusions each of 3 mL 99% dehydrated alcohol for chemical ablation by the cardiologist. The procedure went without issues, and the patient was discharged home. The next morning, the patient developed severe epigastric abdominal pain with radiation toward his back prompting hospital admission. The patient was afebrile, nontachycardic, O₂ saturation at 100% with a physical examination significant for tenderness to palpation of the epigastrium without rebound or guarding. Initial laboratory work was significant for leukocytosis to 16,000 with neutrophil predominance. Lipase was greater than 30,000. An abdominal and pelvic computed tomography scan with oral contrast was obtained, which demonstrated acute interstitial edematous pancreatitis with prominent peripancreatic fluid tracking caudally without loculation and no evidence of pancreatic parenchymal necrosis. A right upper quadrant ultrasound did not demonstrate cholelithiasis or sludge. The patient was diagnosed with acute interstitial pancreatitis and admitted to the medical floors for continuous intravenous fluids. A thorough work-up was initiated to determine the patient's cause of pancreatitis. The patient had no personal or family history of pancreatitis. The patient had no prior outpatient, emergency, or inpatient visits for abdominal pain or any other gastrointestinal complaints in the past. He endorsed lifetime abstinence from alcohol or tobacco use, corroborated by his family members. The patient had no recent travel history and no recent viral illness. The patient's medication list was reviewed and included metoprolol and apixaban; thus, medication-induced pancreatitis was ruled out. Triglycerides were 84, and his IgG4 level was normal. A magnetic resonance cholangiopancreatography was obtained, which did not demonstrate any pancreaticobiliary pathology that could have explained the patient's acute pancreatitis. Initially, it was believed that pancreatitis was caused by the propofol infusion, but during his prior ablations in 2019 and 2020, he received similar amounts of propofol without any complications. On the other hand, this was his first alcohol ablation, where he received 2–3 times the standard

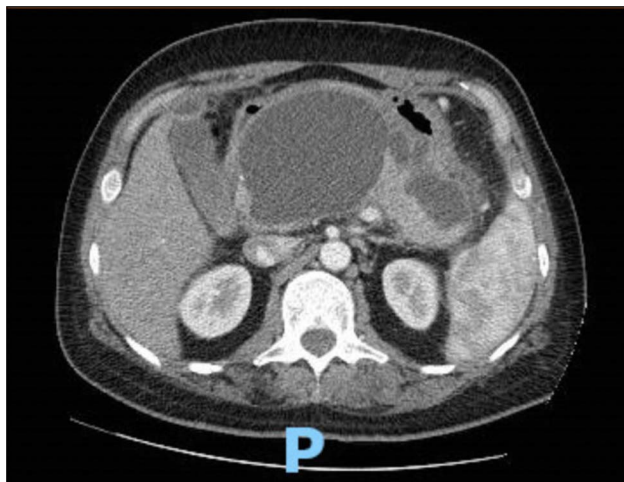


Figure 1. CT imaging demonstrating pancreatic necrosis from the neck to the body where there are a large 10 × 8 cm pancreatic collection and a smaller multiloculated 7 × 4 cm pancreatic collection. CT, computed tomography.

amount of alcohol due to persistent fibrillation, which was ultimately felt to be the cause of the patient's pancreatitis. His hospital course was complicated by persistent abdominal pain and new fevers, with an abdominal and pelvic repeat computed tomography scan demonstrating new necrosis extending from the pancreatic neck to mid-body with extensive liponecrosis of the central small bowel mesenteric root (Figure 1). A week later, after the collections were confirmed to have a well-formed wall on repeat imaging, the patient underwent endoscopic ultrasound-guided cystgastrostomy with a 10 × 20 mm hot lumen apposing metal stent placement, followed by endoscopic necrosectomy 1 week later (Figure 2). The collection was successfully debrided and after 1 session appeared resolved, and the stent was removed. The patient's symptoms had completely resolved.

DISCUSSION

We present a patient who presented with acute pancreatitis complicated by walled off pancreatic necrosis requiring endoscopic cystgastrostomy and necrosectomy. The etiology of his pancreatitis was not initially clear because the usual work-up for common causes was negative. However, the timing of pancreatitis after his cardiac ablation prompted deeper investigation into the medications received during this procedure. Two potential causative factors were identified: propofol and 99% dehydrated ethanol. Propofol is an extremely rare class III/IV agent only documented in 18 case reports in the published literature.^{1,2} Among these cases, propofol-induced pancreatitis was most commonly associated with long infusions for maintenance of sedation (average greater than 24 hours) and with patients without any prior exposure to propofol. In our case, the patient had only received 1 relatively small bolus of propofol at the beginning of the procedure and had tolerated similar doses of propofol during prior procedures, thus making propofol-induced pancreatitis unlikely.



Figure 2. Pancreatic fluid collection seen on endoscopic ultrasound.

However, this was the patient's first experience with transcatheter alcohol infusion. His prior ablation in 2019 was a radiofrequency ablation. In addition, the patient received significantly more alcohol than typically used during this procedure. Typically, only a total of 0.5–3 mL of pure alcohol is used for transcatheter ablation.³ In the landmark Vein of Marshall Ethanol for Untreated Persistent Atrial Fibrillation trial that evaluated the efficacy of Vein of Marshall alcohol infusion for atrial fibrillation, 155 patients successfully underwent alcohol ablations.³ Among all of these patients, approximately 1 mL of ethanol was used in the ablation, with up to 4 ablations performed per procedure. However, in this patient's procedure, 9 mL of pure ethanol, or 2–3 times the recommended volume, was used because of the patient's persistent atrial fibrillation and failed prior ablations. When converted to the blood alcohol level, the amount of alcohol delivered to the patient is approximately the same as the level in an average man after 8–9 drinks!

Animal studies have theorized that alcohol-induced pancreatitis occurs because of mechanisms including increasing oxidative stress, increasing transcription of proinflammatory cytokines, and inhibiting apoptosis and enhancing necrosis.⁴ We can surmise that the significant amount of pure ethyl alcohol delivered directly to the patient's cardiovascular system led to pancreatic inflammation and damage ultimately causing acute pancreatitis.

This is the first documented case of alcoholic pancreatitis from a cardiac ablation. We hope to bring light a rare but potentially serious adverse event that can result from transcatheter alcohol ablation.

DISCLOSURES

Author contributions: All authors provided substantial contributions to the creation of the manuscript, drafting and revising content, approving final approval for publication, and agreement to be accountable for all aspects of the work. K. Raphael is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received July 13, 2023; Accepted November 2, 2023

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