

Cocaine-Induced Acute Pancreatitis

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ABSTRACT

Cocaine use is prevalent worldwide and affects multiple organ systems. Ischemia of the esophagus and small bowel are examples of its gastrointestinal complications. Cocaine-induced pancreatitis is a rare entity. Only 8 cases of cocaine-induced pancreatitis have been described in the literature. We present a rare case of a 61-year-old man cocaine user who presented with his first episode of acute pancreatitis (AP) in which common etiologies of AP were excluded. In addition, we explore the pathophysiology of cocaine-induced AP.

INTRODUCTION

Acute coronary syndromes, chest pain, agitation, and ischemic and hemorrhagic strokes are some of the effects of cocaine on the cardiovascular and central nervous system.¹ Gastrointestinal complications of cocaine use, such as esophageal and bowel ischemia and related perforation, are less common.² Cocaine-induced pancreatitis is a rare entity, and only 8 cases have been described in the literature.^{3–11} Acute pancreatitis (AP) is diagnosed when 2 of 3 criteria are met: abdominal pain typical of AP, amylase or lipase elevation greater than 3 times the upper limit of normal, and imaging findings consistent with pancreatic inflammation.¹² The most common causes are gallstones and chronic alcohol use. To the best of our knowledge, we describe the ninth case of cocaine-induced AP.

CASE REPORT

A 61-year-old man with a medical history of heart failure with reduced ejection fraction (HFrEF) of 35%, pulmonary sarcoid, chronic obstructive pulmonary disease, a cerebrovascular accident a few months earlier, and active crack-cocaine use presented with 3-day severe epigastric pain radiating to the back with associated nausea and vomiting. He denied chest pain, cough, shortness of breath, fever, or chills. He was a former 8-pack-year smoker with remote alcohol use history. Patient reported regular use of intranasal crack cocaine with last use 3 days before presentation. His home medication included aspirin, atorvastatin, lisinopril, and metoprolol tartrate. He denied new medication or recent steroid use.

His vital signs were within normal limits. The physical examination was remarkable for tenderness to epigastric palpation. He had normal breath sounds and no lower extremity edema. He was noted to have an elevated lipase of 2,300 IU/dL (11–82), total bilirubin 0.4 mg/dL, AST 30 mg/dL, ALT 16 mg/dL, WBC 5,700 cells/mL, triglycerides 90 mg/dL, calcium 9.3 mg/dL, and IgG4 levels of 18 mg/dL (1–123). Urine toxicology was positive for cocaine only. Electrocardiogram showed more pronounced T wave inversions in inferolateral leads compared with prior. Thoracic x-ray showed lung base scarring and mild central pulmonary vascular congestion. Abdominal ultrasound showed a heterogeneously hypoechoic pancreas consistent with pancreatitis, normal-sized common bile duct, and a distended gallbladder without gallstones. An abdominal computed tomography (CT) scan with intravenous contrast showed edematous enlargement of the pancreas, peripancreatic fat stranding and free fluid, and enlarged subcentimeter peripancreatic lymph nodes, consistent with AP.

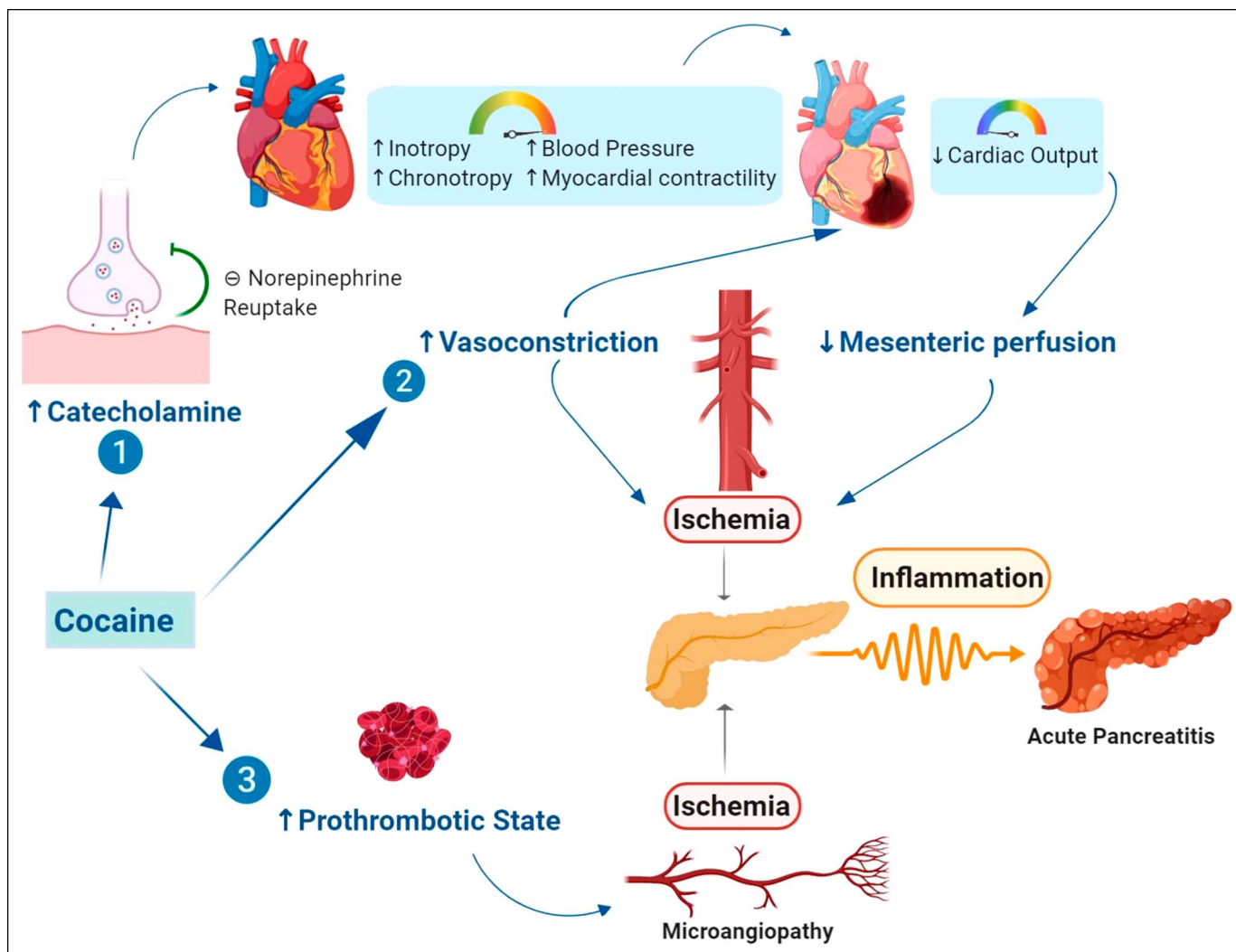


Figure 1. Underlying pathophysiology of cocaine-induced acute pancreatitis.

The patient received intravenous fluids in a goal-directed fashion¹³ with 1-L Lactated Ringer bolus in the emergency department and 200 mL/h for maintenance. On hospital day 2, fluids were decreased and oral intake initiated, with full tolerance of solids on day 3. Patient's pain was controlled well with oral acetaminophen and immediate release oral morphine on day 2. He remained afebrile and hemodynamically stable. He was discharged home on hospital day 4 with counseling and cessation resources for cocaine use and recommendations to continue to abstain from alcohol and tobacco.

DISCUSSION

This patient met diagnostic criteria for AP, which was attributed to crack-cocaine use based on the lack of other identifiable causative factors on history, physical examination, laboratory testing, and imaging. Abdominal ultrasound and CT scan showed no gallstones or structural pancreatic abnormalities with normal liver enzymes and total bilirubin.

Normal IgG4 levels, absence of previous AP episodes and of IgG4-related extrapancreatic involvement except the reactive lymphadenopathy, made autoimmune pancreatitis less likely. Hypertriglyceridemia, hypercalcemia, and new medications were ruled out. Lisinopril¹⁴ rarely causes AP. This patient has been on it for years and remained on it without AP recurrence after initial AP attack. His history of smoking and alcohol use were also remote; the patient was forthright in admitting to his crack-cocaine use, and given the timing of symptom onset, his AP was attributed to recent crack-cocaine use.

Eight total cases of cocaine-induced AP were reported in the literature: 7 were men, and 7 were younger than 30 years.^{3–11} This patient is the most senior among the reported cases, where the oldest was 53 years old. AP developed within 48 hours of cocaine use in 7 patients, while our patient's was within 72 hours. Three patients had a history of alcohol use without active use before the onset of AP, like our patient. No further AP was noted after cocaine cessation in all 5 patients

for whom data were available, which was the case of our patient on initial follow-up and a year after hospitalization.

Cocaine inhibits the reuptake of norepinephrine and dopamine. In the GI tract, it causes vasoconstriction and thrombosis of mesenteric vessels.⁶ The pathophysiology of cocaine-induced pancreatitis is postulated to be related to vasoconstriction and thrombotic microangiopathy.^{5,6} Our patient was using crack-cocaine, which does not undergo first-pass hepatic metabolism. This can lead to a rapid rise in plasma concentration. In addition, it is possible that it might have been mixed or contaminated with other compounds. A common contaminant to consider is levamisole, an immunomodulatory agent whose side effects include vasculitis and agranulocytosis. We cannot rule out the possibility that this could have contributed to our patient's presentation.¹⁵ Concomitant beta-blocker use can result in unopposed alpha receptor activity and worsening of the vasospasm, although this is debated in the literature.¹⁶ We postulate that our patient had decreased pancreatic perfusion due to vasoconstriction and thrombotic microangiopathy of mesenteric vessels from cocaine use and atherosclerosis as well as decreased cardiac output secondary to his HFrEF and myocardial ischemia with resultant pancreatic ischemia leading to an inflammatory response with subsequent AP^{1,17,18} (Figure 1).

The pancreas is highly susceptible to ischemic and reperfusion injury, with AP resulting from shock or decreased perfusion.^{17,18} We also hypothesize that previous alcohol and tobacco use may have led to underlying parenchymal disease, which may have predisposed our patient to AP. Cocaine-induced pancreatitis is not common but is possibly underreported, especially in patients with underlying predisposing factors such as a history of heavy alcohol or tobacco use given the increased prevalence of cocaine use. Cocaine-induced pancreatitis should be on the list of differential diagnoses when treating a patient with abdominal pain and recent cocaine use.

DISCLOSURES

Author contributions: All authors contributed equally to this manuscript. J. Strzepka is the article guarantor.

Previous presentation: This case was presented at the American College of Gastroenterology Annual Scientific Meeting; October 25-30, 2019; San Antonio, Texas, and was also presented at the University of Illinois College of Medicine Research Forum; November 22; Chicago, Illinois.

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