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Single Case

Pancreatitis Induced by Cocaine

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Keywords

Pancreatitis · Cocaine · Management

Abstract

Pancreatitis is one of the commonest diseases of the gastrointestinal tract, characterized by epigastric pain of moderate to severe intensity, which radiates to the back, elevation of pancreatic lipase and amylase enzymes, and changes in pancreatic parenchyma in imaging methods. The most common etiologies vary, generally the most frequent being biliary lithiasis and alcohol, followed by hypertriglyceridemia. Among the less frequent causes is drug-induced pancreatitis. We report a case of acute pancreatitis caused by cocaine, rarely described in literature.

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Introduction

Pancreatitis is one of the commonest diseases of the gastrointestinal tract. The diagnosis is made with 2 of the following 3 criteria, according to several scientific societies [1–4]:

1. Characteristic persistent epigastric pain of moderate to severe intensity, which classically radiates to the back.
2. Alterations in laboratory values (elevation of lipase and amylase pancreatic enzymes higher than 3 times).
3. Changes in pancreatic parenchyma in imaging methods (abdominal CT, MRI, or abdominal ultrasound).

It has different clinical presentations related to its severity, from patients that require few days of hospitalization in a general room to severe cases characterized by multi-organ failure with requirements of a complex vital support. The most common etiologies vary, depending on the population studied, the most frequent being biliary lithiasis and alcohol, followed by hypertriglyceridemia. Among the less frequent causes is drug-induced pancreatitis [5–8], and in this subgroup, very few reports of cases induced by cocaine can be found [9–12].

Case Report

A male patient, 23 years old with no relevant medical history, was admitted to another medical center due to shock and multi-organ dysfunction syndrome after drug abuse. He showed impaired sensorium, hemodynamic instability, rhabdomyolysis, renal and liver failure, associated with thrombocytopenia and coagulopathy. He was intubated and connected to mechanical ventilation, and vasoactive drugs were initiated. The urine toxicological study was positive for marijuana, cocaine, amphetamines, and dioxin (methamphetamines). At that point, he was referred to our center looking for a hospital of high-level complexity.

He was under midazolam and fentanyl sedation, and norepinephrine infusion requirements of 0.15 µg/kg/min. The initial laboratory results were: hematocrit 38%, WBCs 10,200 cells/mL, TGO 1,411 IU/dL, TGP 2,277 IU/dL, total bilirubin 4.1 mg/dL, lipase 2,954 IU/dL, amylase 652 IU/dL, triglycerides 169 mg/dL (Table 1). Physical examination showed no abdominal distention, and positive bowel sounds. Pain was not possible to evaluate due to sedation. Abdominal ultrasound reported normal liver size, intra- and extrahepatic bile ducts were not dilated, gallbladder was not distended, there was no evidence of gallstones, pancreas was normal, and there was minimum free abdominal fluid.

At 72 h of the onset of symptoms and following the improvement of clinical conditions and various organ failures, we decided to extubate the patient and progressively reduced the sedation and analgesic administration. Afterwards, enteral nutrition was initiated by nasogastric tube and the patient reported pain in the upper abdomen irradiating to the back associated with the persistence of elevated pancreatic enzymes, while the rest of all laboratory parameters had returned to normal. After suspending enteral feeding, parenteral nutrition was started with improvement of symptoms. Contrasted tomography of the abdomen evidenced edematous pancreas, without any alteration of the peripancreatic fat or evidence of necrosis (Fig. 1, Fig. 2). As there were no local complications and he remained without abdominal pain, he started oral nutrition 72 h later, showing good tolerance. Daily levels of lipase and amylase are expressed in Figure 3 and Figure 4.

Discussion

Pancreatitis is a gastrointestinal disease commonly caused by gallstones and alcohol. Cocaine is a rare etiology, with few cases reported in the literature. Given the increase in cocaine abuse, we must remember that within the spectrum of gastrointestinal complications associated, one of them could be pancreatitis.

The first discussion we initially faced when analyzing this case was whether the patient actually had pancreatitis or whether we just faced an additional organ failure of its syndrome associated with cocaine. We believe that, since the patient had 2 of the 3 criteria (characteristic pain and elevated pancreatic enzymes) as stated by the different medical associations [1–4], he definitely had diagnosis of pancreatitis, even more when the pain was associated with improvement of the rest of the laboratory tests and with the initiation of enteral feeding tolerance. However, to confirm pancreatitis, we requested an abdominal tomography demonstrating pancreatic edema.

Other entities can also cause elevation of serum lipase in the absence of pancreatitis. These causes can be divided into those associated with a decreased clearance of the enzyme (impaired renal function), intra-abdominal non-traumatic causes, and miscellanea. Within the latter, critical illness deserves special mention, as patients with abdominal trauma may experience increased lipase, and similarly, there are described cases of septic shock and respiratory failure with isolated increased lipase [13]. However, our patient did not have any of these scenarios.

In turn, gallstone was ruled out by ultrasound and CT, alcoholic etiology was not taken into account because the patient was not consuming alcohol, and, finally, hypertriglyceridemia was dismissed because serum values were normal, besides that in the last 2 etiologies, patients usually present with normal pancreatic enzymes.

Cocaine is an alkaloid whose mechanism of action is based on inhibition of noradrenalin reuptake; this would produce vasoconstriction and thrombosis of the mesenteric vessels. The decrease in splanchnic flow would be the cause of the spectrum of gastrointestinal side effects, among which are intestinal ischemia, ischemic colitis, and hollow viscera perforation [14]. There are few reports of cocaine-induced pancreatitis in the literature [9, 10], some of which were associated with thrombotic microangiopathy as pathophysiologic mechanisms in 2 cases [11, 12].

Furthermore, methamphetamines are sympathomimetic drugs whose mechanism of action is increased release of monoamines in the sympathetic nervous system [15]. Acute consumption, as well as cocaine, is also associated with gastrointestinal complications. This spectrum of complications includes acute liver failure and intestinal ischemia. The pathophysiology would be similar to that associated with cocaine use, splanchnic vasoconstriction and necrotizing vasculitis [15, 16]. However, there are no reported cases of pancreatitis due to methamphetamines, which is why in our patient we ascribed the etiology of pancreatitis to cocaine.

Conclusion

Acute pancreatitis associated with cocaine use is a rare entity, so its diagnosis obliges to rule out other etiologies. The low number of cases does not allow elucidating the pathophysiology, which is probably related to vasoconstriction and microangiopathy. The management of these patients is generally complex because it is associated with multiple organ failure, cardiovascular collapse, rhabdomyolysis, and renal failure, and therefore, patients should be admitted to highly complex centers with multidisciplinary teams.

Statement of Ethics

The IRB reviewed the case and gave the approval statement for publication. It ruled that there is no need of informed consent.

Disclosure Statement

There is no conflict of interest.

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Fig. 1. CT showing edematous pancreas body.



Fig. 2. CT showing edematous pancreas head.

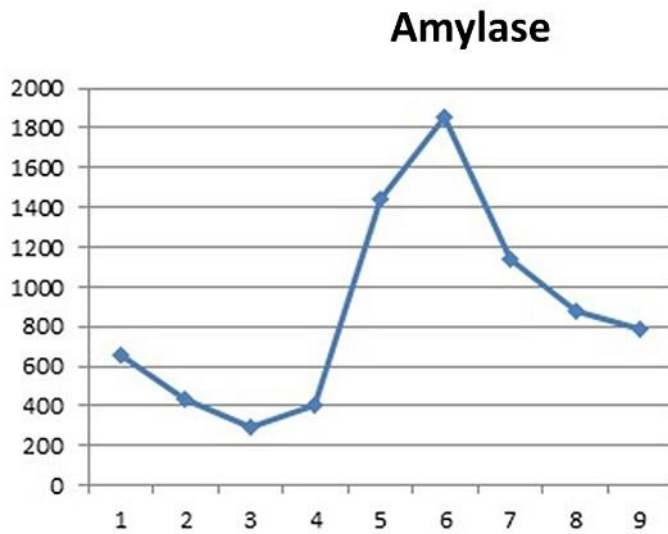


Fig. 3. Evolution of daily levels of amylase (IU/L). On the 3rd day, enteral nutrition was initiated and was suspended on the 6th day.

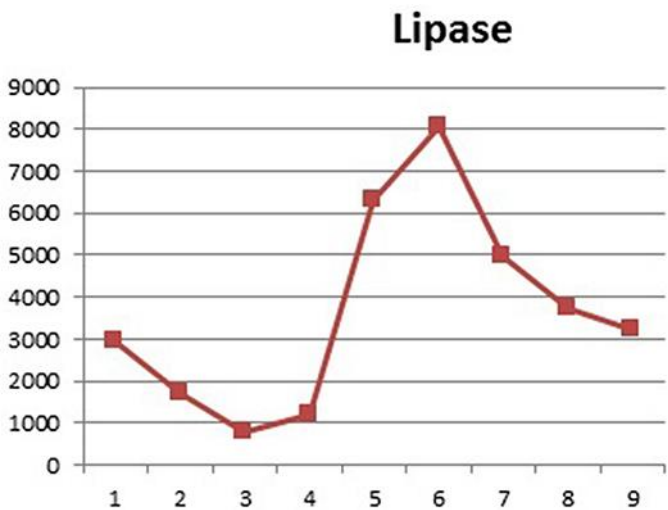


Fig. 4. Evolution of daily levels of lipase (IU/L). On the 3rd day, enteral nutrition was initiated and was suspended on the 6th day.

Table 1. Laboratory values on admission day

Hematocrit: 38%	WC: 10,200 cells/mL	PLAT: 41,000 cells/mL	Na: 141 mEq/dL	K: 5 mEq/dL	CL: 108 mEq/dL	CK: 21,640 IU/L	
Creatinine: 2.4 mg/dL	BUN: 62 mg/dL	Glycemia: 89 mg/dL	ASAT: 1,411 IU/L	ALAT: 2,277 IU/L	CHOL: 74 mg/dL	Alkaline phosphatase: 76 IU/L	Total bilirubin: 4.1 mg/dL
pH: 7.47	PO ₂ : 59 mm Hg	PCO ₂ : 30 mm Hg	HCO ₃ : 21 mEq/dL	Base excess: –1.4	SaO ₂ : 95%	PAFI: 120	Lactic acid: 24 mg/dL
Prothrombin time: 38%	aPTT: 1.64 seg	Amylase: 652 IU/L	Lipase: 2,954 IU/L	Triglycerides: 169 mg/dL	Ca: 7.5 mg/dL	Mg: 3.2 mg/dL	P: 3.9 mg/dL