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Cocaine-induced Hepatotoxicity: A Case Report

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Abstract: Cocaine is a tropane alkaloid that is obtained from the leaves of the coca plant. It has a small number of legitimate medical applications. Historically, it was useful as a topical anesthetic in eye and nasal surgery, although it is now predominantly used for nasal and lacrimal duct surgery. From numerous studies it is known that it causes irreversible structural changes on the brain, heart and other organs such as lungs and kidneys. Cocaine-induced acute liver injury has been described in animal models, although few cases have been reported in humans. Most of the direct hepatotoxic effects are mediated by oxidative stress and by mitochondrial dysfunction produced during the metabolism of noradrenaline or during the metabolism of norcocaina. We describe here the case of a 33-year-old man who developed acute liver injury after cocaine use. He presented to the emergency room with worsening abdominal pain and fatigue requiring hospitalization. Confounding liver insults included a history of untreated chronic hepatitis C and heavy alcohol use.
Keywords: Cocaine, Hepatotoxicity, Humans.

INTRODUCTION

Drug-related hepatotoxicity is a serious health problem and the most common cause of acute liver failure in the United States [1]. Cocaine is not a very common cause of hepatic injury and its prevalence has not been documented. Presentation can vary from subclinical elevation of liver enzymes to fulminant hepatic failure with necrosis of the majority of hepatocytes [2]. No previous studies have shown any association between dose of cocaine and hepatotoxicity in humans. Hepatotoxicity in man was first suggested in 1967 by Marks and Chapple [3], whose study of 89 heroin and cocaine users showed abnormal liver test results in approximately two thirds of the group. Another case of liver toxicity in a 24-year-old male was reported by Kanel GC *et al*. [4]. Wanless IR *et al*. reported 4 cases of cocaine-induced hepatotoxicity and described the histopathological findings of their liver [5].

CASE REPORT

A 33-year-old male presented to Stony Brook University Hospital with four days history of malaise, anorexia and epigastric/right upper quadrant abdominal pain. He denied fever, chills, weight loss, jaundice, pruritus, diarrhea or use of herbal supplementation. His past medical history consisted of chronic hepatitis C and gastritis. Since his diagnosis with hepatitis C, the patient had never received any treatment or followed with gastroenterology/hepatology. Patient is a heavy alcohol drinker of 6-pack-beer daily. He uses occasionally cocaine, once every month or every other month, and last use was 4 days before he presented to the hospital. He does not use any other medications. Family history was negative for chronic liver disease including Wilson's disease, α-1-antitrypsin deficiency and autoimmune hepatitis.

At the time of his admission, patient was afebrile with unremarkable vital signs. Physical exam revealed mild epigastric and right upper quadrant tenderness with no hepatomegaly, no jaundice, anicteric sclera, no pharyngeal erythema or exudate, supple neck, clear lungs, regular heart rate and rhythm, non-focal neurologic exam, and no rash. Complete blood count and chemistries were within normal limits. The rest of the patient's laboratory values were significant for transaminitis (Figure 1), and he was admitted secondary to a possible acute exacerbation of his underlying liver disease. The patient's chest x-ray was unremarkable and an abdominal ultrasound with Doppler did not show any liver or biliary tree pathology, ascites or portal vein thrombosis. Additional investigation included serologic testing for cytomegalovirus, Epstein-Barr virus, Herpes Simplex virus and hepatitis A, B, and C viruses; results of all the serologies were positive only for previous infection with hepatitis C virus but negative for any acute infection. Further evaluation also included testing his levels of acetaminophen, tissue transglutaminase antibody, HIV-1 antibody, urinalysis, amylase, and lipase. His liver-panel results were consistent with hepatocellular but not biliary injury. Patient's bilirubin,

serum albumin levels and prothrombin time were within normal levels throughout his hospital course. He was started on high doses of proton pump inhibitors for presumable alcoholic gastritis or peptic ulcer disease as a cause of his abdominal pain. After 48 hours of admission, the patient's laboratory values and symptoms improved, and there were no other complications during his hospital stay. Liver biopsy or any other additional testing was not performed at that point, as patient's overall health condition showed remarkable improvement. Patient was discharged on the third hospital day. He did not follow with his scheduled appointment with Gastroenterology as outpatient.

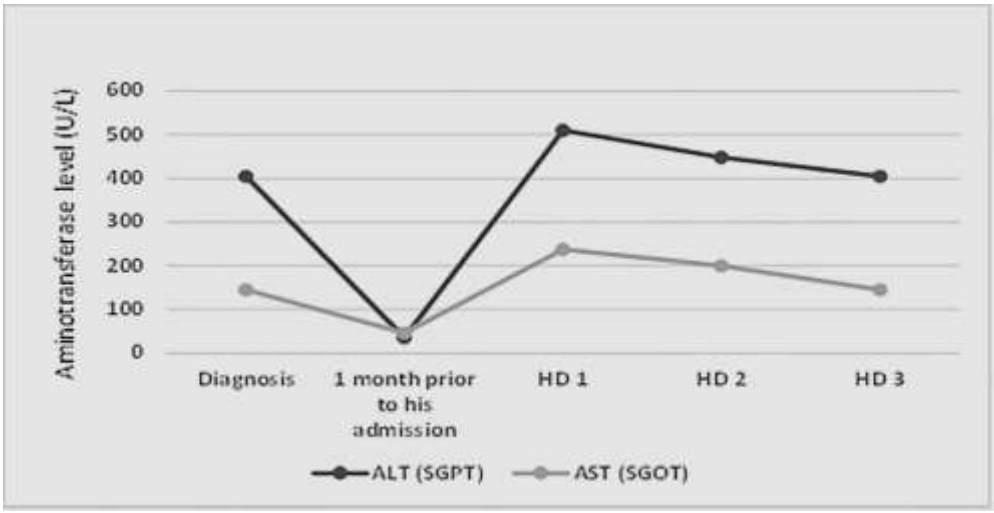

Fig. 1: Serum aminotransferase levels (HD: Hospital Day)

We arrived at a diagnosis of cocaine-induced acute liver injury after an evaluation for viral and metabolic conditions that failed to reveal a cause for hepatitis in this patient. His presentation, with laboratory findings emerging after administration of the drug and normalization shortly after withdrawing the drug, led to our conclusion that the hepatitis resulted from a reaction to cocaine. Of note, in August 2013, when patient was diagnosed with chronic hepatitis C, he admitted to cocaine use few days prior to his presentation as well. This pattern of elevated liver enzymes shortly after cocaine use strengthens our belief that cocaine is the ultimate culprit. According to the Naranjo scale, it is probable (score = 7) that this case of acute liver injury was a result of an adverse drug reaction [6].

DISCUSSION

Our case is one of the few cases in the medical literature that cocaine use has resulted in a significant elevation of transaminases. The mechanisms of cocaine-induced liver injury have been studied extensively in animal models for over three decades. In humans, post mortem liver pathology has shown that cocaine induced hepatotoxicity can lead to zonal and periportal coagulative necrosis with both macrovesicular and microvesicular fatty changes in the residual hepatocytes [4, 7-9].

Drug-induced liver disease can manifest as acute or chronic hepatitis, cholestasis, fibrosis/cirrhosis, vascular injury, and tumors. Hepatotoxicity can be dose dependent or idiosyncratic.

Drug history must include temporal relationship to symptoms, doses, and use of non-prescription or herbal drugs. Diagnosis of drug-induced liver injury, however, can only be made after exclusion of other causes of hepatotoxicity (infectious, autoimmune, vascular, metabolic etiologies etc.) [10]. Liver biopsy should be considered if the extent of liver damage or the etiology is in doubt. There is no treatment other than removal of the offending agent and supportive care. Patients should be considered for emergency liver transplantation on individual basis if the conservative measures fail.

In our case, we observed a hepatocellular liver injury possibly related to cocaine in a man who uses it occasionally. As discussed above, a year prior to this admission, the patient found to have elevated transaminases following cocaine use, similar to this episode. Between these two occasions, patient had normal levels of transaminases. The recurrence of the laboratory findings shortly after exposure to cocaine supports our hypothesis. One might suggest that the observed lab results could be attributed to the fluctuation of liver enzymes due to chronic hepatitis C. In chronic hepatitis C though, the elevation of the transaminases is usually mild [11].

CONCLUSION

Our case report emphasizes the fact that cocaine-induced liver injury can be under-reported or overlooked and should be included in the differential diagnosis in cases presenting with acute liver injury.

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