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Cocaine as a Rare Cause of Cholecystitis

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Cocaine as a Rare Cause of Cholecystitis

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Abstract

Cholecystitis, inflammation of the gallbladder, is diagnosed in approximately 200,000 people in the US each year. Cocaine is an illicit drug that is wildly abused in the US, GI complications such as acute pancreatitis, colitis, bowel perforation, mesenteric ischemia, and biliary tree obstruction have been reported in limited studies. To the best of our knowledge, there are no reported cases of cocaine-induced cholecystitis and no established treatment plans to date, however, we strongly believe that our patient's cocaine use resulted in cholecystitis. Hence, we wanted to shed some light on the possible side effects of cocaine on the gall bladder.

Keywords

Cocaine Use, Acute Cholecystitis, Ultrasound Findings

Conflict of Interest Statement

We have no conflict of interest to declare

CASE REPORT

Cocaine as a Rare Cause of Cholecystitis

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Abstract

Cholecystitis, inflammation of the gallbladder, is diagnosed in approximately 200,000 people in the US each year. Cocaine is an illicit drug that is wildly abused in the US, GI complications such as acute pancreatitis, colitis, bowel perforation, mesenteric ischemia, and biliary tree obstruction have been reported in limited studies. To the best of our knowledge, there are no reported cases of cocaine-induced cholecystitis and no established treatment plans to date, however, we strongly believe that our patient's cocaine use resulted in cholecystitis. Hence, we wanted to shed some light on the possible side effects of cocaine on the gall bladder.

Keywords: Cocaine use, Acute cholecystitis, Ultrasound findings

1. Introduction

Cholecystitis, inflammation of the gallbladder, is diagnosed in approximately 200,000 people in the USA each year.[1] Gallstones are the leading cause of cholecystitis (contributing to 90−95% of cases). However, only 1−3% of people with gallstones develop cholecystitis.[2] Cholecystitis not caused by gallstones is termed acalculous and is diagnosed based on clinical features including ultrasound findings. Management of gallstone-induced and acalculous cholecystitis is similar. It involves treatment of symptoms, antibiotic therapy, and cholecystectomy or insertion of a percutaneous cholecystostomy tube.

In the U.S.A, cocaine is a widely abused illicit drug. It primarily exerts its effects on the nervous and cardiovascular systems. Gastrointestinal complications, such as acute pancreatitis, colitis, bowel perforation, mesenteric ischemia, and biliary tree obstruction, have been reported in a limited number of studies. Based on our review of available literature, there are no reported cases of cocaine-induced cholecystitis. Here we present a case of cholecystitis which was potentially precipitated by cocaine use.

2. Case report

A 30-year-old African American female presented to the hospital via emergency medical services (EMS) after being found down. She had past medical history pertinent for substance abuse and alcohol use disorder. Prior to admission, the patient was found on the floor unconscious at a party after having used cocaine, marijuana, and alcohol. EMS administered intravenous Naloxone, IV ondansetron and fluids during transport to the emergency department. The patient had altered mental status (GCS 10), vomiting, nausea, and abdominal pain on presentation to the hospital. She was hypotensive at 71/48 mmHg, afebrile at 37.2 °C (99 °F), and

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tachycardic at 121 bpm, respiratory rate of 20. On examination, the patient was in acute distress, ill-appearing, diaphoretic, and had dry mucous membranes. Abdominal examination revealed generalized tenderness, more prominent in the RUQ, and with a positive Murphy's sign. Labs showed elevated creatinine at 2.0 mg/dL, a negative BHCG test of <2 mIU/ml elevated transaminases with AST 896 U/L, ALT 871 U/L, alkaline phosphatase of 41 U/L, lipase 28 U/L, total bilirubin of 0.6 mg/dL, potassium of 8.1 mmol/L, lactic acid level of 7.5 mmol/L, leukocytosis with a white blood cell count of 25,700 (neutrophil predominance), platelet count of 312,000, and elevated troponins of 565 pg/ml with a positive delta of 19 pg/ml. Electrocardiogram showed hyperacute T-waves. Urine toxicology was just positive for cocaine. Computed tomography (CT) scan of the abdomen and pelvis showed that the gallbladder was distended and demonstrated tubular hyperdense abnormality centrally. It was unclear if the findings represented biliary sludge within the gallbladder or a contracted gallbladder with marked wall edema and an ultrasound was recommended. Subsequent ultrasound of the right upper quadrant showed an extremely thickened gallbladder wall with surrounding edema and a positive sonographic Murphy's sign. As there were no visible gallstones, these findings were consistent with acute acalculous cholecystitis (Fig. 1). The patient was treated with empiric antibiotics, including vancomycin and piperacillin-tazobactam, and was given septic bolus fluids with ringer lactate. Cultures of blood and urine resulted negative. By day 2, the patient's altered mental status had returned to baseline, and creatinine, transaminases, and WBC count continued to trend downward. The patient still complained of RUQ abdominal pain and tenderness, and the exam showed additional suprapubic and hypogastric regional tenderness. However, on day 3, the patient's symptoms continued to improve with supportive care and follow-up ultrasound revealed resolution of acute cholecystitis (Fig. 2). The patient was then discharged home on amoxicillin-clavulanate, aspirin and scheduled for outpatient cardiac magnetic resonance imaging.

3. Discussion

Cocaine induces vasoconstriction and thrombosis of mesenteric vessels, which is thought to contribute to gastrointestinal complications such as acute pancreatitis, colitis, bowel perforation, mesenteric ischemia, and biliary tree obstruction have been reported in limited studies, the existing clinical evidence is scarce.[3] However, such a clinical correlation is speculative at best, and supporting literature is scarce in quantity and strength.

Cocaine acts as a norepinephrine, dopamine, and serotonin reuptake inhibitor, leading to psychosomatic effects. The cocaine used by most individuals has been adulterated with different substances, including both pharmacologically active and inactive chemicals. Levamisole is the most commonly adulterant used as it potentiates the effect of cocaine 3. According to the statistics of United States Dawn Abuse Warning Network (DAWN) cocaine use had the highest rates of emergency department visits in 2011 compared to other drugs (162.1 per 100,000 population).[4] A few studies have highlighted the association between cocaine use and ischemic events in the pancreas, colon, and small intestine. A case report noted the reoccurrence of acute pancreatitis

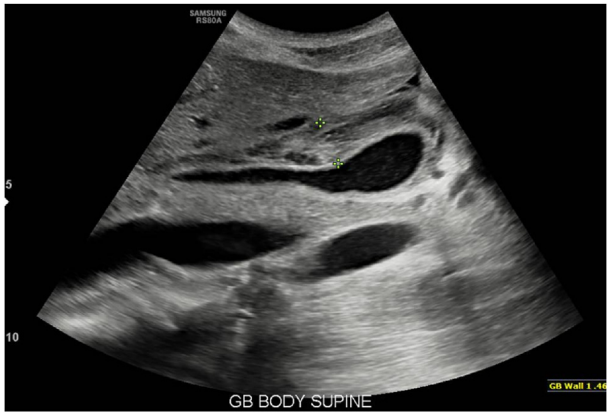

Fig. 1. Ultrasonography showing extremely thickened gallbladder wall and surrounding edema finding specific for acute cholecystitis.

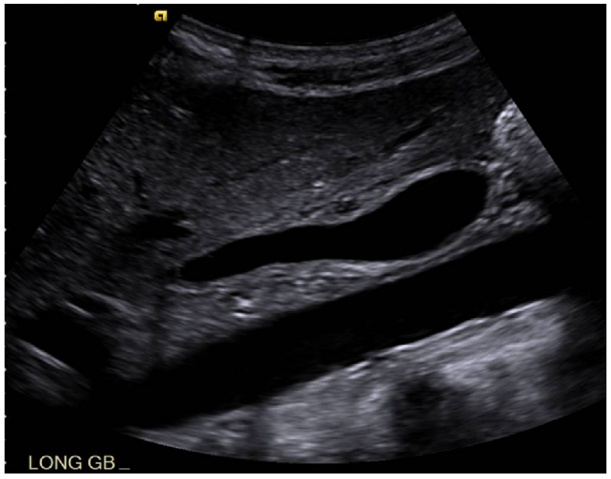

Fig. 2. Showing ultrasound done 72 h after the first one with no changes in the gallbladder wall and surrounding tissue. A sign of resolved cholecystitis.

after cocaine re-exposure. Vasoconstriction and thrombotic microangiopathy was speculated to cause pancreatitis, as was evident from reduced pancreatic perfusion following cocaine use.[5] A case report noted edema and congestion of gall bladder wall on gross examination in addition to multiple micro vascular thrombi in a patient after cocaine use.[6]

Acute acalculous cholecystitis often results due to gallbladder stasis or ischemia. Our case presents a rare incidence of acute acalculous cholecystitis occurring potentially due to cocaine use. Other risk factors include acute myeloid leukemia, acquired immunodeficiency syndrome, ampullary stenosis, bone marrow transplant, burns, cardiopulmonary resuscitation (CPR), childbirth, choledochal cyst, cholesterol emboli, diabetes mellitus, cystic duct obstruction, end-stage renal disease, heart failure, haemobilia, immunosuppression, infections, major trauma, mechanical ventilation, medications (e.g., opiates), transfusions, sepsis/hypotension, non-biliary surgery, total parenteral nutrition, vasculitis. Our patient did not have any significant medical or surgical history and had not received any recent transfusions, considering the above-mentioned risk factors. She did not require cardiopulmonary resuscitation or mechanical ventilation. She was also not intubated during her hospitalization. She was not on any immunosuppressive medication regimens. She had only tested positive for cocaine in her toxicology screen and was negative for opioids. Her CT scan and ultrasound ruled out any anatomical risk factors. She was found to have normal BHcg in labs, ruling out pregnancy, and had no recent history of severe burns. The exact etiology could not be identified in our patient and a potential causal relationship between cocaine and cholecystitis was considered.

There is no established treatment and management plan for such cases. Our case was managed conservatively with intravenous antibiotics, fluids, and antiemetics which resulted in significant improvement in the patient's condition with the discharge home 72 h after initial presentation to our ED. The close temporal association between cocaine use and symptom onset followed by clinical and biochemical improvement after conservative management suggests that cocaine may have induced the episode of cholecystitis.

Since cocaine is a widely used illicit entertainment drug, awareness should be raised regarding the possible association of cocaine with acute acalculous cholecystitis, and this should be considered a differential when tackling such cases in the future and when etiology is in doubt. In addition screening for drugs needs to be initiated in young adult patients presenting with symptoms of cholecystitis. Moreover, research should be initiated to study this association.

Conflicts of interest

None.

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