A Rare Case of Cocaine Induced Biliary Tree Constriction in a Middle Aged Male

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Abstract

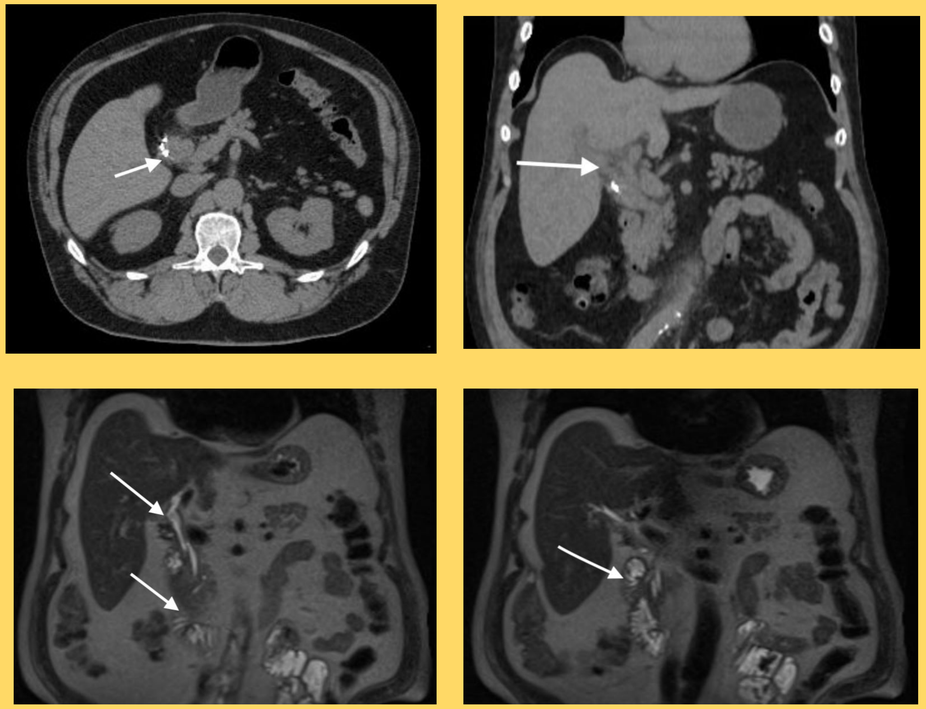
This is a case of a 62 year old male with a history of extensive cocaine use and cholecystectomy that presented to the emergency department with abdominal pain, urobilia, nausea, and vomiting. The patient had elevated total bilirubin (5.2) and direct bilirubin (3.7). CTA subsequently showed a mass in the gallbladder fossa measuring 3.1 x 2.8 cm. MRCP showed a 4.9 x 3.0 cm mass at the porta hilium with proximal biliary ducts and abrupt transition at the porta hilium. ERCP was performed with stent placement and brush biopsy which showed predominantly benign ductal epithelium with rare atypical cells. Fluid ductal cytology was also negative for CA 19-9 levels at this time were 94.3. He subsequently had internal-external biliary drain placed. The patient was subsequently discharged and told to obtain endoscopic ultrasound as an outpatient. The patient was subsequently re-admitted and discharged for other issues several times and received EUS at one of the surrounding hospitals, results were inconclusive and the test needed to be repeated.. On last admission to the hospital CT scan showed no biliary tree obstruction which was further confirmed with MRCP, and removal internal external biliary drain. We suspect that the patient's initial symptoms and radiographic findings of biliary tree constriction may have been induced by cocaine use. Evidence in the literature states that cocaine use can precipitate Sphincter of Oddi dysfunction leading to obstructive jaundice. This is a rare case that has not been described in the literature. At this present time no direct correlation has not linked cocaine usage to biliary tree constriction. We highly suspect that this patient's obstructive jaundice and biliary tree constriction was caused by underlying cocaine use.

Introduction

Cocaine is an illicit drug used by millions of people in the United States every day. The drug is extracted from the leaves of the Coca plant and processed into benzoylmethyl ecgonine. Once the drugs enters into the body. It serves as a norepinephrine, dopamine, and serotonin reuptake inhibitor causing psychosomatic effects. It also behaves as a monoamine oxidase inhibitor and stimulates alpha receptors. A more potent form of the drug is “crack cocaine” which is the smokable version of the drug. This form of is cocaine hydrochloride, it has a more potent effect due to the fact that it does not undergo first pass hepatic metabolism. Primarily cocaine main side effects are centered on the cardiovascular system and nervous system. It is well known and documented that cocaine use can lead to myocardial infarction, life threatening arrhythmia’s, cerebrovascular accident, seizures. It has been documented to cause pulmonary complications such as pulmonary hypertension, pulmonary edema, pneumothorax, pneumomediastinum, pulmonary fibrosis, interstitial pneumonitis, and barotrauma. Other complication includes retroperitoneal fibrosis, placental abruption, spontaneous abortion, and drug induced psychosis. To a lesser extent, the effect of cocaine on the gastrointestinal tract have been studied as well but have been limited to case reports and are rarer. There is documented case of cocaine induced gastric and duodenal ulcers perforation, pancreatitis, intestinal ischemia, mesenteric ischemia, ischemic colitis, as well as small and large bowel perforation. To the best of our knowledge, there are no documented cases of cocaine induced biliary tree constriction however, we strongly believe that due to our patients use of cocaine within 48 hours of presenting to the hospital initial, and subsequent resolution of biliary stricture pathology upon returning to the hospital months later.

Case Presentation

- 62 year old male presented to the ED with dark urine, abdominal pain, nausea, and vomiting.
- Upon arrival the patient's electrolytes were within normal limits, lipase was 27. total bilirubin 5.06 which rose to 5.2 and Direct bilirubin of 3.6 which rose to 3.7. He had positive UDS for cocaine on admission
- Abdominal U/S was ordered which initially showed 1.5 cm dilated common bile duct with a surgically absent gallbladder indicating prior cholecystectomy.
- CTA Ab/Pel showed a showed a mass in the gallbladder fossa measuring 3.8 x 2.1 cm possibly representing cholangiocarcinoma.
- MRCP showed a 4.9 x 3.0 cm mass at the porta hilium with proximal biliary ducts and abrupt transition at the porta hilium.
- Gastroenterology was consulted and performed ERCP with stent placement and Brush Biopsy , which were negative.
- Interventional Radiology was consulted for internal-external drain biliary drain placement.
- Hematology and Oncology was consulted as well given, his imaging results they recommended obtaining EUS and outpatient follow-up based on the results of the EUS.
- His T. Bili and D. Bili trended down to normal levels and he was subsequently discharged with instructions and prescription to obtain EUS on the outpatient basis and to follow up with our team and Heme/Onc
- The patient had many social issues such as homelessness, drug addiction, and psychiatric issues.
- He was able to get EUS performed and results were inconclusive, requiring a repeat.
- He never followed up outpatient to receive repeat EUS and was admitted and discharged from our hospital several times over the span of 6 months.
- During his last admission at around six months time, a repeat CT ab/pel showed no ductal dilation or mass.
- MRCP was performed which showed no ductal dilation and mass as well.
- Interventional Radiology was consulted to perform ductal cholangiogram and internal-external drain removal which showed no masses or dilation of biliary tree.
- The patient was subsequently discharged in stable condition.



A. CT Ab/Pel Axial section showing 3.1 x 2.8 cm mass. **B.** CT Ab/Pel Coronal View Showing 3.1 x 2.8 cm mass possibly representing cholangiocarcinoma. **C.** IR biliary ductal cholangiogram showing contrast flow into the duodenum with no evidence of biliary tree obstruction or masses. **D.** IR biliary ductal cholangiogram with contrast present in the duodenum no evidence of biliary tree obstruction or masses.

Discussions

- Cocaine can cause many different pathophysiological processes including myocardial infarction, arrhythmias, pulmonary edema, pulmonary fibrosis, renal fibrosis, vasoconstriction, and intestinal ischemia.
- In our patient we highly suspect that the patient’s initial imaging results and elevated total and direct bilirubin were a results of cocaine use within less than 48 hours on arrival to the hospital
- At the time his initial lipase was 27, ruling out pancreatitis.
- Another possibility could be our patients history of cholecystectomy which is a cause of benign biliary tree structure but more commonly than not it does not resolve on its own. As in our case.
- There have been a few case reports that have stated cocaine use induced pancreatitis.
- We suspect that via similar mechanism of vasoconstriction to vessel supplying the biliary tree leading to ischemia, spasm and stricture.
- Another theory is that the patient could have possibly had sphincter of oddi dysfunction which could possibly been exacerbated by cocaine use.
- The patient could have possibly had biliary lymphadenopathy causing biliary tree obstruction as well.
- Overall, given the patient’s use of cocaine within 48 hours of admission and ruling out many other different types of pathology. We believe strongly that the patient in question biliary tree constriction which was assumed to be cholangiocarcinoma at the time, was due to cocaine use.
- So far to the best of our knowledge this is the only report of cocaine induced biliary tree constriction.

Conclusion

The case presented here is a very interesting. We have strong belief that our patients initial presenting pathology was induced by cocaine use. But there is no real way to definitively prove this. However, this case can be a good learning tool and review for the many different side effects and pathology that cocaine usage can elicit. The patient in question had many variable factors that affected our care and ability to more specifically rule out cholangiocarcinoma. In conclusion, we do not think there is anything we could have done to change the outcome in this case scenario and we believe we provided the patient with the best possible care that we could have.

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