Hepatolithiasis, A Rare Cause of Secondary Biliary Cirrhosis

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Hepatolithiasis is a rare disease in India. Here we report a patient with extensive hepatolithiasis with biliary obstruction. She presented with secondary biliary cirrhosis, developed rapidly progressive liver failure and died.

Case Report

A 53-year-old lady presented with jaundice, ascites and hepatic encephalopathy. There was no history of fever or abdominal pain. Her ultrasound abdomen confirmed liver cirrhosis with dilation of intra-hepatic biliary radicles with filling defects. Hepatitis B surface antigen and anti HCV antibody was non-reactive. Her computed tomography of abdomen confirmed extensive hepatolithisis with ascites (**Figure 1 and 2**). A diagnosis of secondary biliary cirrhosis was made. Magnetic resonance cholangiopancreatography ruled out common bile duct stones. Arterial ammonia was 182 (normal range 11 to 51 micro mol per litre) she had high SAAG (2.15) ascites and no spontaneous bacterial peritoninits.

Serum bilirubin 17.97 mg per dl (reference 0-1), direct bilirubin 15.8 mg per dl (reference 0-0.3), alkaline phosphatase 143 units per liter (reference 35-105), aspartate aminotransferase 87 units per liter (reference 0-40), alanine aminotransferase 56 units per liter (reference 0-40), serum albumin 3.22 mg per dl (reference 3.5-5.2) to INR 1.25. There was no history of fever, abdominal pain in past. There was no prior history suggestive of liver disease.

Unfortunately, patient has a rapidly progressive course of illness and could not be referred for liver transplantation and died.

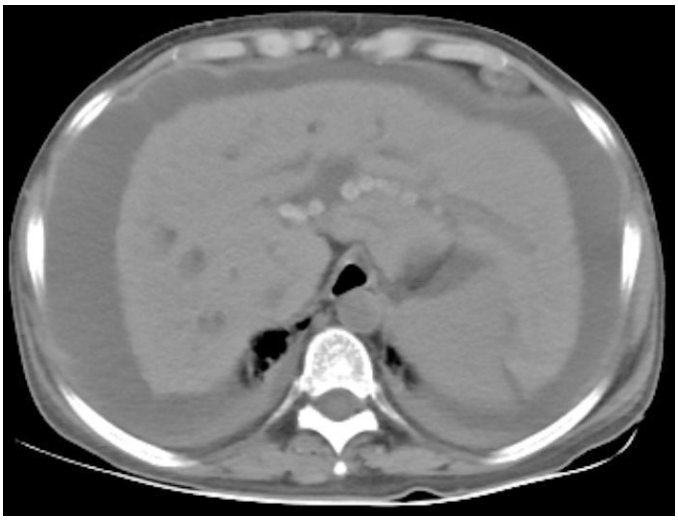

Figure 1: Stones in left intrahepatic duct with ascites and irregular liver contour.

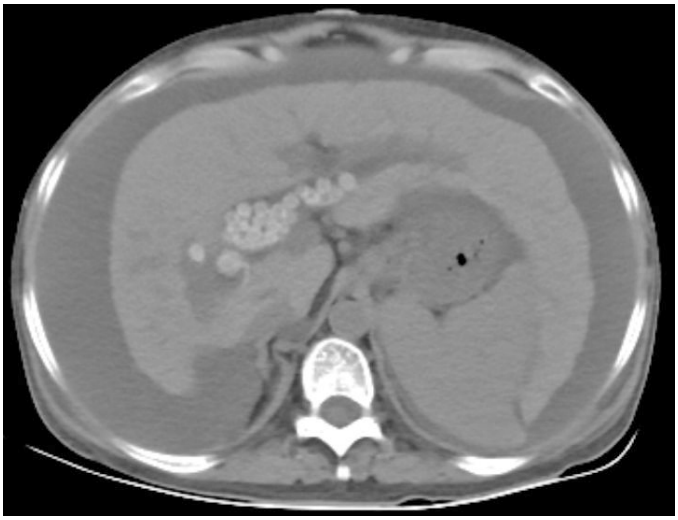

Figure 2: Stones in right intrahepatic ducts and biliary confluence with ascites and irregular liver contour.

Discussion

Our patient had hepatic encephalopathy as substantiated by features of liver cirrhosis and ascites on computed tomography. She developed rapidly evolving liver failure. Although, she did not have symptoms of biliary obstruction or cholangitis in past. Hepatolithiasis involves presence of gallstones proximal to the confluence of left and right hepatic ducts, irrespective of presence or absence of common bile duct stones.[1] Hepatolithiasis is frequently encountered in East Asian countries and is rare in other

parts of the world.[2] Cause of hepatolithiaisis is unknown, parasitic infestations, bacterial infections, dietry factors, environmental and genetic causes have been implicated.[3] Hepatolithiaisis may cause in recurrent cholangitis, secondary biliary cirrhosis and cholangicarcinoma.[1]

Feng *et al*. proposed Dong's classification for hepatolithiasis, in this classification diffusely distributed stones with bilary cirrhosis were classified as type IIc and liver transplant is recommended in such patients. On the other hand, patients with localized stones or diffuse stones without cirrhosis (Types 1 and IIa and IIb), are best managed with hepatectomy.[4]

References

1. Sakpal SV, Babel N, Chamberlain RS. Surgicalmanagement of hepatolithiasis. HPB (Oxford). 2009;11:194-202.
2. Tazuma S. Gallstone disease: Epidemiology, pathogenesis, andclassifcation of biliary stones (common bile duct and intrahepatic).Best Pract Res Clin Gastroenterol 2006; 20: 1075-1083
3. Kim HJ, Kim JS, Joo MK, Lee BJ, Kim JH, Yeon JE, *et al*. Hepatolithiasis and intrahepatic cholangiocarcinoma: A review. World J Gastroenterol. 2015;21:13418–13431.
4. Feng X, Zheng S, Xia F, Ma K, Wang S, Bie P, Dong J.Classification and management of hepatolithiasis: A highvolume, single-center's experience. Intractable Rare Dis Res. 2012; 1:151-156

Post-ERCP Hepatic Hematoma: Case Report and Review of Literature

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Endoscopic retrograde cholangiopancreatography (ERCP) is commonly performed minimally invasive therapeutic procedure for pancreaticobiliary disorders.[1] Even though considered as relatively safe procedure, ERCP is associated with high potential complications requiring prolonged hospital stay, invasive intervention, morbidity and risk of mortality.

Risk of complications associated with ERCP ranges from 3-10% with mortality risk of 0.33% and commonly reported complications are post-ERCP pancreatitis, bleeding, perforation, infection and sedation related events.[2] Less common complication like hepatic hematoma have been reported as case reports and its true incidence is not known.[3] Hepatic hematoma even though rare, carries significant morbidity and mortality risk of around 10%.[4] We report a case of hepatic sub capsular and parenchymal hematoma, developed post ERCP for choledocholithiasis, which was successfully managed conservatively without any complications.

Case Report

68 year old male, known case of abdominal aortic aneurysm, post aortic stenting in 2016, ischemic heart disease, post percutaneous transluminal coronary angioplasty (PTCA) in 2019, diagnosed as acute calculous cholecystitis in 2019 planned for interval cholecystectomy but deferred due to poor cardiac function, presented with history of sudden onset of upper abdominal pain worsening with food intake for 2 days. On examination, he was hemodynamically stable, febrile and had mild tenderness in epigastric and right hypochondrium. On evaluation his hemogram showed mild leucocytosis, normal hematocrit. His liver function tests showed alanine transaminase (ALT) 311.0 IU/L, aspartate transaminase (AST) 625 IU/L, alkaline phosphatase (ALP) 352IU/ml and serum bilirubin 2.7 mg/dl. His serum amylase level was 1061 U/ml and serum lipase was >300 U/L and renal function tests were normal. His ultrasound abdomen showed cholecystolithiasis with normal proximal bile duct and lower bile duct obscured by bowel gas. In view of clinical suspicion of biliary pancreatitis, MRCP was done which showed cholecystolithiasis, choledocholithiais with dilated common bile duct with central IHBRD with focal acute pancreatitis. Based on clinical, laboratory and radiological parameters, clinical diagnosis of biliary