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Spontaneous Gall Bladder Rupture in a Patient with Decompensated Cirrhosis Identified at time of Simultaneous Liver and Kidney Transplantation

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Rupture of Gall bladder can occur in less than 5% patients with acute cholecystitis. The incidence in cirrhosis is not known but may be lower due to gall bladder wall thickening and adhesions. Predisposing factors in cirrhosis include underlying gallstones & prior Trans Arterial Chemo embolization (TACE). We present a case of spontaneous ruptured gall bladder detected during Simultaneous Liver Kidney Transplant in a middle-agedpatient.

Case Report

We report the case of ruptured gall bladder detected during Simultaneous Liver Kidney Transplantin a 58-year male with Non-Alcoholic Fatty Liver Disease (NAFLD) cirrhosis & IgA nephropathy) with chronic kidney disease-stage 5 on hemodialysis. Hisprior decompensations requiring admissions included episodes of hepatic encephalopathy, (SBP) and GI Bleed. He was recently admitted with an episode of hypotension with anemia 4 weeks prior to transplant with a diagnosis of SBP and sepsis. Ascitic tap was hemorrhagic with drop in hemoglobin of more than 1.5 gm. During that admission for hypovolemia and anemia, he underwent CT angiography of abdomen and pelvis where no active bleeding /hematoma was identified. Diagnostic upper gastro-intestinal (UGI)endoscopy and colonoscopy after resuscitation for bleeding also did not show evidence of active or recent bleeding. Traumatic paracentesisor spontaneous ruptured abdominal wall varix was presumed to be responsible for the blood-stained ascites. The episode was managed conservatively with antibiotics and 2 units of packed red cell transfusion with resolution of symptoms.

While on the waiting list with a MELD Na of 31 he received an offer for Standard criteria brain-dead donor liver and kidney with Kidney Donor Profile Index (KDPI) below 20. During laparotomy,a gall bladder perforated at fundus with blood and bile-stained ascites was found. (**Figure 1**) There were no stones identified at laparotomy or in the removed gall bladder. He had an uneventful

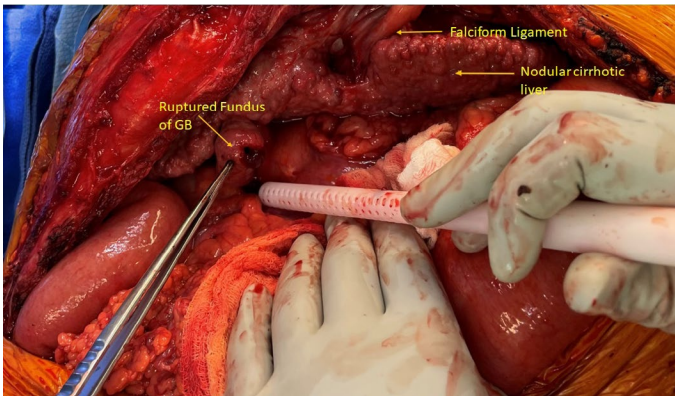

Figure 1: Intra operative photograph showing perforated fundus in the thick-walled gall bladder.

SLK with deceased donor organs and was discharged on POD11.

Discussion

Rupture of GB, which can occur in up to 4 to5 % patients with acute cholecystitis, is classified based on Niemier's classification depending on free, walled off or intrahepatic rupture.[1,2] Although the true incidence of rupture due to acute cholecystitis in cirrhosis is not known, it may be lower due to chronic gall bladder wall thickening, edema and adhesions.[3] Predisposing factors in cirrhosis include underlying gallstones with acute cholecystitis and prior TACE which may lead to compromise of blood flow due to embolization materialin the cystic artery. Our patient had no evidence of gallstones on multiple prior imaging or history of TACE.

Previous reports have reported cases of Gall bladder rupture in patients with cirrhosis.[3,4] Two recent reports of rupture also had underlying gall stones as the precipitating cause.[5,6]

Thus, although reported as spontaneous, they had either cholelithiasis or recent TACE. The presence of ascites, possibly, masks the clinical features of acute cholecystitis and the peritoneal irritation, is misdiagnosed as SBP or labelled as traumatic tap when ascitic fluid is bloody. The etiology for spontaneous rupture in our case, possibly the reason for emergent admission 4 week prior to transplant, is likely acalculous cholecystitis in other critical illness, with necrosis of fundus. This did not manifest with leukocytosis (due to hypersplenism) and thepain, and the paracentesis findings were interpreted as SBP. The presence of blood and the elevated serum bilirubin likely masked the bile staining on visual inspection of aspirated ascites.

Conclusion

Extraluminal gall stone on imagingin a patient with hemoperitoneum or hemorrhagic ascites without obvious traumatic tap or recent TACE should warrant appropriate imaging and analysis of ascites for bile and may aid in diagnosis. Optimal management depends on clinical picture and presentation. Ongoing bleeding may need angioembolization (may not work for gall bladder wall varix bleed). In patients where bleeding stops, conservative management is safer till transplant. Surgical hemostasis with drainage or cholecystectomy is likely to have high mortality and morbidity due to underlying cirrhosis and portal hypertension. Rupture identified at surgery in the absence of infected collection has no significant consequence and the transplant can proceed as planned.

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