specific which include an irregularly shaped, hypodense, hypovascular mass with peripheral enhancement; areas of central enhancement that can give a multiloculated appearance with nearby necrotic or non-necrotic lymphadenopathy. These characteristics mimic those of pancreatic cystic lesions that are inflammatory or neoplastic[5]. A clearly defined mass that typically appears in the pancreatic head and exhibits heterogenous enhancement are MRI findings of focal pancreatic TB. On fat-suppressed T1-weighted images, these lesions are typically hypointense, and on T2-weighted images, they exhibit a mixture of hypo/hyperintensity[2].

Biliary obstruction may develop as a result of external compression or direct ductal compression by infected nodes, along with periductal inflammation and stricture[1]. With a success rate ranging from 50 to 62%, fine needle aspiration cytology/biopsy has been used to diagnose a small number of patients[2]. High-resolution imaging with EUS enables the detection of abdominal and mediastinal lymphadenopathy as well as the differentiation of pancreatic and peripancreatic tumors. For diagnostic reasons, fine-needle biopsy (FNB) or EUS-guided fine-needle aspiration (FNA) can be used to investigate lymphadenopathy or pancreatic masses. It can detect pancreatic cancer up to 95% of the time and pancreatic tuberculosis up to 76% of the time[6]. Samples acquired could be sent for histology and microbiology utilizing acid-fast bacilli culture, Ziehl-Neelsen staining, and polymerase chain reaction assay[2]. According to reported literature, anti-tuberculous drug therapy is advised for 6-12 months[5].

Endoscopic intervention is required in patients who develop obstructive jaundice due to biliary stricture, which may be progressive despite initiation of anti-tuberculous therapy. This intervention should be carried out early on in the course of treatment[5]. The progression or resolution of the disease can be guided by CT imaging[6]. In this case series, one patient needed biliary intervention, and all three patients showed clinical and biochemical response to anti-tuberculous therapy on follow up.

Conclusion

Large peripancreatic lymphnodes compressing the pancreas and surrounding adjacent vasculature can mimic pancreatic masses and may be mistaken for malignancy. In endemic nations, tuberculosis should be considered, especially in young patients in whom pancreatic mass is reported on imaging.

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Double Gall Bladder Mimicking Choledochal Cyst Type 6

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Congenital anomalies of the biliary tree are frequently encountered in patients undergoing surgery for gallbladder stones and are a common cause of postoperative complications in these patients. Vesica fellea duplex or double gall bladder is a rare congenital malformation that

results from embryonic dysgenesis of the hepatic antrum.[1] Autopsy studies show an incidence of 1 in 4000 births, however, symptomatic cases are rare.[1] Due to the rarity of the disease, these are often misdiagnosed preoperatively. We report a case of double gall bladder which was diagnosed as choledochal cyst type 6 on imaging and was found out to be a double gall bladder intraoperatively.

Case Report

A 67-year-old female with no known comorbidities presented to the outpatient clinic with complaints of pain right upper abdomen for the last 2 days. There was no associated history of fever/nausea/vomiting. Abdominal examination was unremarkable. Ultrasound abdomen revealed gall bladder stones with thickened walls suggestive of acute cholecystitis. Her biochemical profile showed leucocytosisand liver functions showed raised alkaline phosphatase (220 U/L) with normal bilirubin.

Because of elevated alkaline phosphatase, magnetic resonance cholangiopancreatography (MRCP) was performed which was reported as distended gallbladder filled with multiple stones and another cystic outpouching related to the cystic duct suggestive of type 6 choledochal cyst. The rest of the biliary anatomy was normal. Given these findings, patient was planned for laparoscopy and to proceed with cholecystectomyor excision of cyst/common bile duct with Roux En-Y hepaticojejunostomy.

On laparoscopy, the common bile duct was delineated and found to be normal. The gall bladder was unusually distended. Dissection was performed at the Calot's triangle and to our surprisetwo separate cystic ducts were identified running parallel to each other accompanying a single cystic artery. (**Figure 2**) Both cystic ducts and artery were clipped separately and divided. Gall bladder was then dissected off the cystic plate using hook electrocautery. The cut section of the specimen showed two separate lumens of gallbladder covered in a common peritoneal lining with separate cystic ducts entering into each moiety (Type 2 double gall bladder). (**Figure 2**) After the surgery, the operative findings were discussed with the radiologist. The MRCP was reviewed again, which showed a likelihood of two cystic ducts, each entering into the distended gall bladder

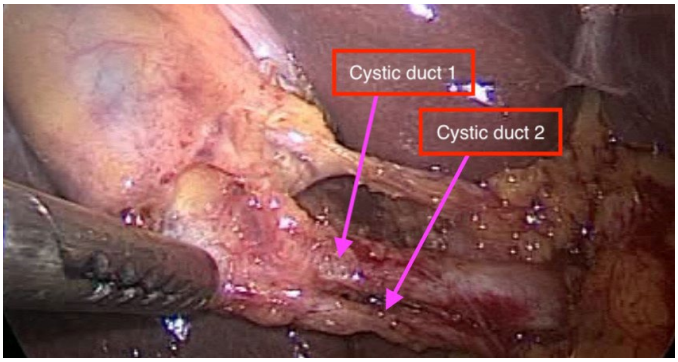

Figure 1: Intraoperative image showing double cystic duct after delineation of Calot's triangle anatomy (one duct clipped).

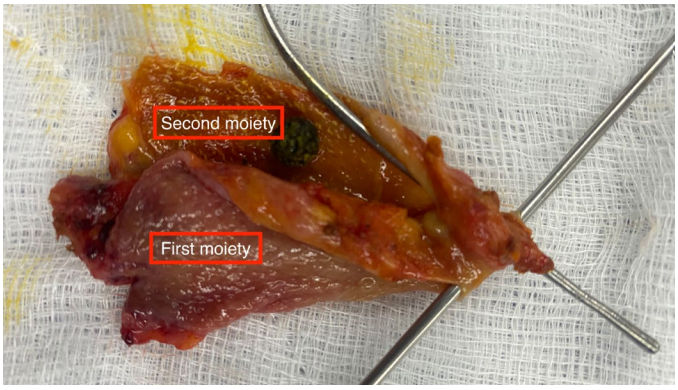

Figure 2: Cut section of gall bladder, showing two separate gall bladder lumen with separate cystic duct (probe passed from cystic duct opening).

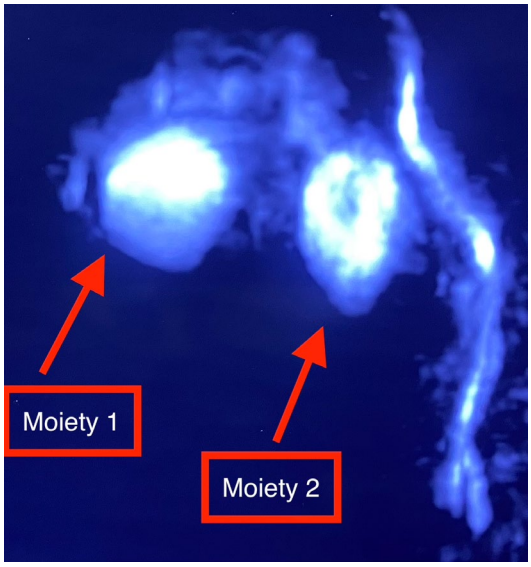

Figure 3: MRCP image representing double gall bladder.

and the juxta gallbladder cystic outpouching suggestive of a double gallbladder .(**Figure 3**) The post-operative course was uneventful and the patient was discharged on postoperative day 1. Histopathology was suggestive of cholecystitis.

Discussion

Congenital malformations of the gall bladder can be either morphological or positional abnormalities. Double gall bladder is a rare morphological abnormality that results from incorrect differentiation of embryonic organs during the 5th to 6th week of gestation, when an additional bud arises from the hepatic diverticulum, leading to the formation of two separate gallbladders.[2]

The earliest description of a double gall bladder dates back to 31 BC, in a sacrificial victim of Emperor Augustus. Sherren in 1911, documented the first double gall bladder in a living human.[3]

Multiple classification systems for double gallbladder have been proposed based on anatomical features and the presence of associated anomalies. The first classification was given by Boyden et al in 1929, based on 20 reported cases.[3] The two main groups included – Group 1 included bilobed gall bladders with single cystic duct – vesica fellea divisa and Group 2 included true gall bladder duplication – vesica fellea duplex.

Harlaftis et al in 1977 again classified duplex gall bladder into three groups as per the embryogenesis. Type 1 or split primordial group includes a single cystic duct draining into common bile duct. These are further divided into V-shaped or Y-shaped. Type 2 or accessory gall bladder group has more than one cystic duct into the common bile duct. They can be either H type or right and left trabecular type. Type 3 is a rare anomaly like triple gall bladder. Our case is a type 2 H type double gall bladder.

The anatomical location of the two moieties can vary. Most gallbladders share a common peritoneal coat and are usually adjacent to each other. However, completely separate gall bladders in different locations – intrahepatic or subhepatic have also been reported in the literature.

As per a report by Gadour et al. in 2021, there are around 57 cases of double gallbladder reported to date. There are a few reported cases of triple gallbladder also.[4]

There are no specific signs and symptoms associated with just presence of a double gall bladder. Symptoms are usually secondary to gallstones, cholecystitis or biliary pancreatitis. The differential diagnosis includes focal adenomyomatosis, Phrygian cap, choledochal cyst and GB diverticulum.

The finding may be commonly missed on a routine transabdominal ultrasound due to the rarity of disease and ultrasound being an operator-dependent investigation. Congenital anomalies of the gall bladder and biliary tract are associated with a higher incidence of biliary injuries post cholecystectomy and hence biliary anatomy should be properly defined in patients with high suspicion of such findings on an ultrasound. MRCP or CT cholangiography is the investigation of choice for the identification of double gall bladder preoperatively.5 We reviewed the MRCP retrospectively where two ducts entering into the gallbladder were identified which was missed by the radiologist. This clinical error can happen in such rare cases due to a lack of anatomical familiarity. An intraoperative cholangiogram or a laparoscopic ultrasound may also be used in cases of doubt intraoperatively.

The indications for surgery are similar to as in the presence of a single gall bladder with surgery reserved for patients with symptoms. Laparoscopic cholecystectomy is the treatment of choice, however, a low threshold for conversion to open surgery is advised in case of doubt.

Conclusion

Duplication of gall bladder although a rare congenital anomaly, requires special attention to the biliary and the vascular anatomy in cases of doubt. Surgeons should consider these anomalies while performing cholecystectomy to avoid post-cholecystectomy biliary injuries. A laparoscopic approach to double gall bladder is feasible and safe, however, should be performed with caution by a high-volume hepatobiliary surgeon.

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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

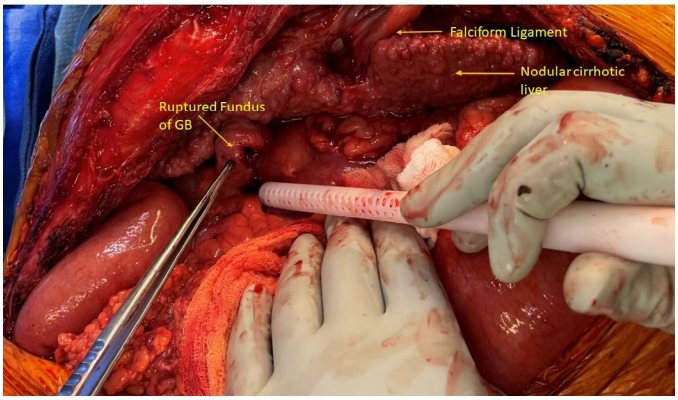

Figure 1: Intra operative photograph showing perforated fundus in the thick-walled gall bladder.