esophageal sphincter. This could lead to reflux esophagitis and precipitate formation of recurrent stricture.1

Since these strictures are generally long, these patients are treated with esophageal-colicoplasty in our institution.

References

Bile Ascites following Percutaneous Trucut Needle Liver Biopsy

Sir,

We would like to report an unusual complication, namely, asymptomatic bile ascites, following a percutaneous trucut needle liver biopsy in a 5 year old boy with underlying chronic liver disease. No surgical intervention was needed.

A 5 year old boy was admitted with jaundice of 8 weeks' duration. Examination revealed features of chronic liver disease and a moderate degree of firm hepatosplenomegaly. Liver function tests revealed a total bilirubin of 204 μmol/L, and conjugated fraction of 136 μmol/L, SGOT and SGPT 86 IU (1.43 μmol/L) and 80 IU (1.33 μmol/L), serum albumin 3.2 g/dl (32 g/L) and globulin 3.8 g/dl (38 g/L). Alkaline phosphatase was 15 KAU (1.77 μmol/L). Prothrombin time index was 60%. Hepatitis B antigen was negative by ELISA method, as was the workup for Wilson's disease, autoimmune hepatitis and – antitrypsin deficiency. Not bleeding grade I esophageal varicos were demonstrated on upper gastrointestinal endoscopy. An ultrasound of the abdomen excluded extrahepatic biliary tract obstruction and revealed altered hepatic echotexture and no dilatation of intrahepatic radicles.

The patient underwent a percutaneous liver biopsy with a trucut needle. The procedure was uneventful. Ascites was noticed on the fourth day after the biopsy. The patient was otherwise asymptomatic. Abdominal paracentesis revealed bilious fluid which was bacteriologically sterile and had a protein content of 2.2 g/dl (22 g/L). Ascitic fluid bilirubin and repeat serum bilirubin levels done at the same time were 119 μmol/L and 85 μmol/L respectively. A repeat ultrasound showed ascites and did not reveal any additional abnormality. As the ascites resolved within 4 weeks without surgical intervention, and the patient was completely asymptomatic, cholangiographic studies were not done. The liver biopsy suggested features of cirrhosis.

Our case illustrates an unusual complication of percutaneous needle biopsy of the liver. The incidence of bile ascites complicating liver biopsy has been reported to be 0.04% to 0.09%.12 The indolent course in our patient could be ascribed to a slow leak of sterile bile, probably from rupture of one of the small intrahepatic ducts.

It would be unlikely that a puncture of the gallbladder resulted in this complication.3 This is supported by the complete recovery without surgical intervention: cholecystectomy has been recommended as the treatment for gallbladder injury.4 In fact, surgical exploration is necessary to repair the leak in a majority of cases with bile ascites.5 Besides, there was no gallbladder tissue demonstrable histopathologically in the biopsy specimen and the patient had no symptoms. An isotope hepatobiliary scan would have been helpful in demonstrating the site of leak, but unfortunately was not available to us then.

Schoor6 reported a similar case following fine needle biopsy of the liver. Piccino et al7 reported a higher complication rate of bile peritonitis with trucut needle as compared to Menghini needle. Bile leak leading to
bile ascites may not require surgical intervention, but the progress of the patient needs monitoring.

References

Ultrasound Demonstration of Diminished Respiratory Caliber Variation in Portal Vasculature: A Sure Sign of Portal Hypertension

Sir,

Previous workers\(^1\) considered absence of respiratory caliber variation in portal vessels to be a specific sign of portal hypertension (PH). However, they did not define a lower limit of the respiratory variation below which the diagnosis of PH could be predicted with certainty. Kurok et al.\(^4\) observed quantitative respiratory variations in normal subjects, via mean respiratory variations of 40%, 150%, and 80% respectively in the portal (PV), splenic (SV) and superior mesenteric (SMV) vein diameters, but they did not correlate the value in patients with PH.

This prospective study involved 100 normal healthy subjects (78 men, 22 women; mean age 32 years, range 17-50) and 50 patients with PH (39 men, 11 women; mean age 36 years, range 16-63). The diagnosis of PH was based on the clinical features and the documentation of esophageal varices by esophagoscopy. The normal subjects and the patients were examined with real-time linear-array ultrasound scanner (EUB-24F) using a 3.0 MHz transducer. To avoid the effects of meal and posture,\(^5\) the subjects were studied after an overnight fast, and all measurements were taken in the supine position. The portal vessels were displayed in longitudinal section and the diameters were measured at their broadest at the same point during deep inspiration (I) and deep expiration (E). The respiratory caliber variation in percentage was calculated in each case as (I-E) \times 100/E; the mean ± SD values were compared using Student’s t test.

Sonography could visualize the PV, SV and SMV in 100%, 98% and 96% of healthy volunteers, and in 100%, 92% and 82% of the patients. The mean ± SD values and the range of percentage increase in caliber with inspiration in the PV, SV and SMV were 36.5 ± 10.2 (22.2-58.3), 67.8 ± 12.0 (36.7-100.0) and 62.7 ± 12.4 (40.0-100.0) in normal subjects; while these values in patients were 0.5 ± 3.4 (0.0-7.1), 0.7 ± 2.8 (0.0-8.4) and 0.7 ± 2.3 (0.1-7.7). The respiratory variation was statistically significant (p < 0.001) in normal subjects, but insignificant (p > 0.1) in patients.

Hemodynamic and morphologic changes in the normal portal vasculature during respiration (an increase in the diameters on inspiration and decrease on expiration) have been shown on pathophysiologic as well as ultrasonic studies.\(^6\) Bolondi et al.\(^7\) observed a significant respiratory caliber variation in normal SV and SMV, but not in PV. The present study shows a significant caliber variation in all the three veins, and thus confirms and extends the findings of Kurok et al.\(^4\) On quantitative estimation, we did not encounter in any case a respiratory variation less than 22%, 36% and 40% in the PV, SV and SMV; the respective values in an earlier study\(^8\) were 12%, 32% and 29%. Previous workers\(^1\) attached importance to the absence of caliber variation as being specific of PH; this was seen in 78-89% of their patients. In our study, the respiratory difference in the diameters of the PV, SV and SMV was absent in 98%, 94% and 90% of the patients respectively; in the remaining cases too, the difference was insignificant. Furthermore, in no case did the respiratory difference exceed the lower limit of caliber variation seen in normal subjects.

Hence, a diminished (rather than absent) respiratory caliber variation in the PV, SV and SMV of less than 22%, 36% and 40% respectively could be considered a specific, sensitive and accurate ultrasound diagnostic index for patients with suspected portal hypertension. Furthermore, in contrast to previous studies,\(^1\) we attach greater significance to the portal vein caliber variation because this vein could be traced out easily in all cases.

References