Image in EUS

Portal Hypertensive Biliopathy Developing After Acute Severe Pancreatitis

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Rana SS, Bhasin DK, Rao C, Singh K. Portal Hypertensive Biliopathy Developing After Acute Severe Pancreatitis. *Endosc Ultrasound* 2013; 2(4): 228-229.

A 46-year-old female patient was presented with a severe epigastric pain and vomiting of 3 days duration. Her serum amylase was 630 (70-200) U/L and serum bilirubin was 0.5 (0.3-1.3) mg/dL. She had normal hemogram, renal function tests, calcium and lipid profile and intact parathormone levels. There were no gall stones on ultrasound of the abdomen. Contrast enhanced computerized tomography (CT) showed grade E acute pancreatitis (AP) with a CT severity index of 8. She was managed conservatively and improved. An endoscopic ultrasound (EUS) evaluation performed 1 month later revealed normal gall bladder and common bile duct and she was diagnosed as a case of idiopathic AP.

On follow-up, 9 months later, she was found to be having elevated serum alkaline phosphatase 356 (N: 42-128) U/L with normal aminotransferases. Ultrasound of the abdomen was revealed prominent central intrahepatic biliary radicles with thrombosed portal and splenic vein. EUS revealed a large number of intra-abdominal venous collaterals. These collaterals were prominent around the gastro-esophageal junction, porta and peri pancreatic location (Fig. 1). The common bile duct was found to be prominent (8 mm) with extensive venous collaterals compressing it from outside as well as intra choledochal collaterals were noted (Figs. 2-4). The pancreatic body and tail were atrophied and main pancreatic duct was not dilated. A diagnosis portal hypertensive biliopathy (PHB) secondary to segmental portal hypertension was made. There was no jaundice and there are no esophagogastric varices. She has been advised regular follow-up and she is asymptomatic at 6 months follow-up.

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E-mail: drsurinderrana@yahoo.co.in
Received: 2012-10-25; Accepted: 2013-03-10
DOI: 10.4103/2303-9027.121244

PHB is usually secondary to extra-hepatic portal vein obstruction, non-cirrhotic portal fibrosis or liver cirrhosis. It is due to the biliary obstruction or ischemia caused by the adjacent porto-portal collaterals formed secondary to long standing portal vein obstruction.[1] Left sided portal hypertension secondary to thrombosis of spleno-portal axis occurs in up to 15% of chronic pancreatitis (CP) and 22% of AP patients.[2,3] Occurrence of PHB is rare with CP and has been rarely reported.[4] PHB has also been rarely reported to occur following AP and this may be because the vascular thrombosis in AP may resolve in at least one-third cases after recovery and also the portal hypertension is of shorter duration, probably giving no time for the formation of collaterals.[5,6] Choledochal varices in these patients can be documented by using EUS or intraductal ultrasonography.[7]

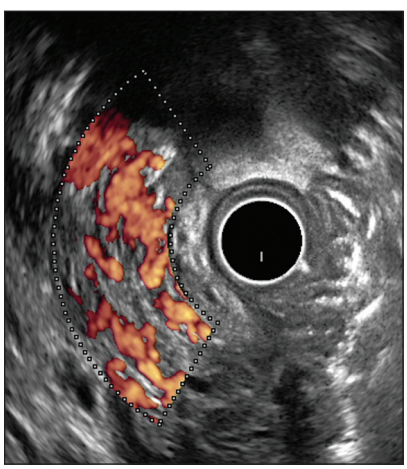

Figure 1. Endoscopic ultrasound showing extensive peri pancreatic collaterals

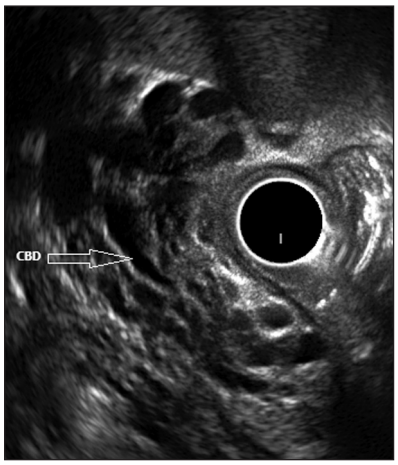

Figure 2. Endoscopic ultrasound showing prominent common bile duct with multiple anechoic channels around it

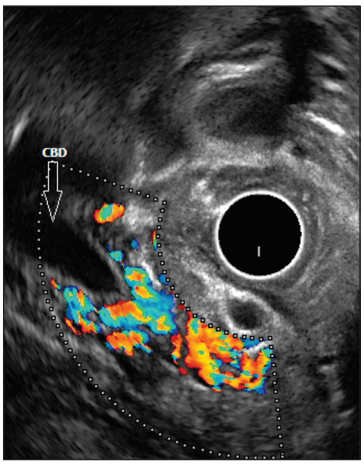

Figure 3. Color Doppler showing multiple collaterals around the common bile duct

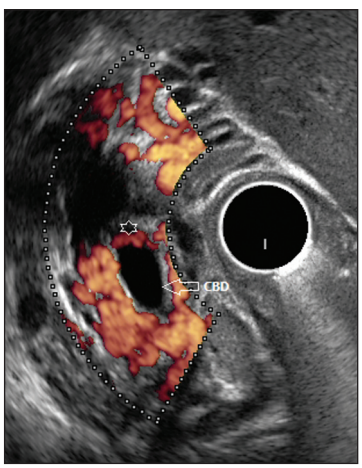

Figure 4. Power Doppler showing multiple collaterals around the common bile duct. An intra-choledochal collateral is also noted (*)

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