Pseudoparallel Channel Sign on Ultrasonography in Alcoholic Hepatitis

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A 27-year-old male presented with yellowish discoloration of eyes and urine for 1 month with a history of hematemesis 1 month back. He had history of chronic alcohol consumption, of around 80–100 g/day ethanol for last 8 years with last intake 20 days ago. Physical examination revealed pallor, deep icterus, hepatomegaly of 5 cm below right costal margin, smooth and mildly tender and a hepatic bruit on auscultation. There was no splenomegaly and free fluid in the abdomen. Hemoglobin was 8.7 g/dL, total leukocyte count 16,000/mm³, platelet count 174,000/mm³, and prothrombin time was 18 s prolonged with an INR 2.22. Liver function tests revealed total bilirubin of 15 with a conjugated fraction of 8 mg/dL with aspartate aminotransferase level of 223 IU/mL, alanine aminotransferase of 69 IU/mL, alkaline phosphatase of 135 IU/mL and serum albumin of 2.6 g/dL. His renal parameters were normal. His viral markers (hepatitis B surface antigen and antibody to hepatitis C virus) were negative. Upper gastrointestinal endoscopy showed grade II–III esophageal varices with cherry red spots for which variceal band ligation was performed. His Maddrey's discriminant score was 97.8 and model for end stage liver disease score was 27. His ultrasound showed large liver with irregular surface, heterogenous echotexture and splenomegaly. Ultrasonography also demonstrated the findings, which were suggestive of alcoholic hepatitis.

The findings are shown in Figures 1–4.

Ultrasound of liver showed parallel tubular structures adjacent to each other [pseudoparallel channel sign (PPCS), Figure 1]. Duplex Doppler ultrasound also demonstrated the color flow in each of the tubular structure. Dilated intrahepatic artery demonstrated red color, while portal venous branch showed blue color (Figure 2). Pulsed Doppler waveform was seen in the portal venous

branch (Figure 3) and in the left hepatic artery branch within the liver (Figure 4).

The sonographic findings of parallel tubular structures in alcoholic hepatitis have been observed in the past resembling the parallel channel sign seen in obstructive jaundice.^{1–3} This entity of intrahepatic artery dilation has been termed as PPCS by Sumino and coworkers.⁴ The parallel tubular structures in this entity are formed by a dilated hepatic arterial branch and an adjacent branch of the portal vein.

Hepatic arterial buffer response is an intrinsic regulatory mechanism of the liver to maintain total hepatic blood flow when portal perfusion decreases. Increased hepatic arterial blood flow mediated by adenosine washout in the portal triad compensates for the reduced portal blood flow. Histologic studies demonstrate the presence of dense sinusoidal collagen deposits in patients with acute alcoholic hepatitis. The sinusoidal collagen causes increased sinusoidal resistance, impeding blood flow through the hepatic sinusoids. Ultimately, sinusoidal pressure builds until portal blood flow is compromised in a retrograde manner, with marked reduction in portal venous perfusion of the sinusoids. The reciprocal response of hepatic arterial blood flow to reduction in portal venous perfusion is well

Figure 1 Ultrasound of liver showing parallel tubular structures adjacent to each other (pseudoparallel channel sign).

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Abbreviations: PPCS: pseudoparallel channel sign

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Figure 2 Duplex Doppler ultrasound shows the color flow in each of the tubular structure. Red color in the dilated intrahepatic artery and blue color in portal venous branch (pseudoparallel channel sign).

established. To preserve perfusion to the liver, compensatory dilation of the hepatic artery occurs with subsequent increase in hepatic artery flow. Angiography of hepatic arteries in alcoholic hepatitis demonstrate elongated, stretched and tortuous arteries.⁵ The marked increase and tortuosity of the hepatic artery is responsible for the characteristic systolic bruit heard on auscultation of the liver surface.⁶

A significant decrease in the hepatic artery resistive index is seen in acute alcoholic hepatitis compared to controls and chronic viral hepatitis. Ethanol itself may be responsible for hepatic artery dilatation as it has been seen in chronic alcoholic patients without liver damage.⁷

Figure 3 Pulsed Doppler waveform is seen in the portal venous branch within the liver.

Figure 4 Pulsed Doppler waveform is seen in the left hepatic artery branch within the.

However progression of alcoholic hepatitis to cirrhosis profoundly impairs the hepatic arterial responsiveness as a result of fibrosis with vascular distortion.⁷ However coefficients of interobserver and intraobserver variability ranged from 5.2% to 10.1% in this study.

Sumino et al observed this finding of PPCS in 90% of patients with acute alcoholic hepatitis and in 23% of patients with other than alcoholic liver disease. However it was not detected in nonalcoholic liver disease and healthy individuals. Liver biopsy was available in 51 patients with alcoholism and PPCS gave a sensitivity of 82%, specificity of 87% and diagnostic accuracy of 84% in diagnosing acute alcoholic hepatitis.⁴

Sumino et al studied this finding also in Japanese group of patients.⁸ Duplex Doppler ultrasound modality has been used to study the hemodynamics within the hepatic artery in patients with acute alcoholic hepatitis.⁹ The mean hepatic artery diameter is larger in patients with acute alcoholic hepatitis than in those with cirrhosis and healthy controls. Peak systolic velocity is higher and mean resistive index is lower in acute alcoholic hepatitis compared to cirrhosis and healthy controls.⁹ There was no correlation between the Duplex Doppler ultrasound parameters and the severity of liver disease as assessed by Maddrey's discriminant function. The clinical significance of this finding has not been studied however it does demonstrate the pathophysiological alterations in acute alcoholic hepatitis and may serve as a diagnostic sonographic finding in patients with alcoholic hepatitis.

CONFLICTS OF INTEREST

All authors have none to declare.

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