CASE REPORTS

Tc-99m RBC Blood-Pool Imaging Demonstrates Umbilical Vein in Portosystemic Shunt

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Dynamic hepatic scintigraphy in a patient with longstanding ethanolism revealed hepatofugal drainage of the liver area. That this pathway represented the umbilical vein was shown by subsequent blood-pool images. The umbilical vein drained the left portal vein into venous channels in or near the anterior abdominal wall. These channels proceeded through epigastric vessels into the right femoral/iliac venous system. The umbilical vein may serve as a shunt from the portal system to the systemic circulation in cases of portal hypertension. Blood-pool imaging has a potential role in clarifying possible venous channels identified by hepatic scintiangiography, and can suggest the presence of portal hypertension when these vessels are visualized.

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Constriction of hepatic venous channels, with subsequent obstruction to normal portal (venous) blood flow, will elevate portal venous pressure. Collaterals between portal and systemic circulations develop in response to portal hypertension. One pathway for collateral circulation involves the embryologic, probe-patent umbilical vein (*1*,*2*). Aggard and associates concluded that recanalization of the umbilical vein often occurs in hepatic cirrhosis and portal hypertension (*3*). Demonstration of the umbilical vein during hepatic scintigraphy is rare, with only three reported cases; this may be due in part to lack of routine dynamic hepatic scintiangiography (*2*,*4*,*5*). We describe another case of the umbilical-vein pathway demonstrated by dynamic hepatic scintiangiography, and the first in which an intravascular label was subsequently used to define the vessel(s) involved.

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CASE REPORT

A 40-yr-old male with a long-standing history of ethanolism and liver disease was admitted to the hospital with a 2-wk history of abdominal pain, bloating, weakness, and increasing jaundice. Significant laboratory values included markedly elevated bilirubin (total of 20 mg/dl with a direct of 11.6 mg/dl) increased alkaline phosphatase (142, with normal = 12–68), mildly increased SGOT (55, with normal = 7–21), normal SGPT (14, with expected range 6–19), and an increased GGT (263, with normal = 15–85 IU/l).

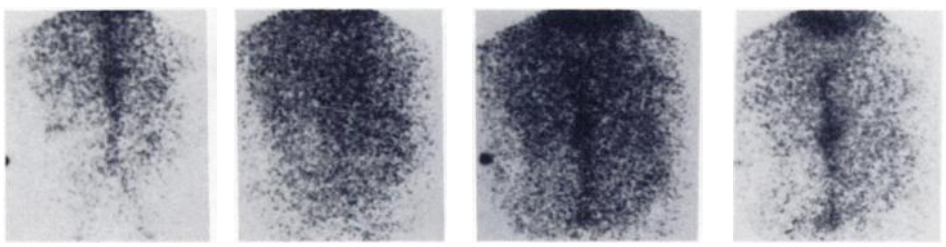

FIG. 1. Consecutive images (3 sec each) from hepatic dynamic study. Left-hand view reveals late perfusion, with intrahepatic activity demonstrable ("dot" marker is on patient's right side). Second image shows diffuse intraorgan "blush." On next two images, hepatofugal activity is apparent, traversing a dilated and tortuous pathway.

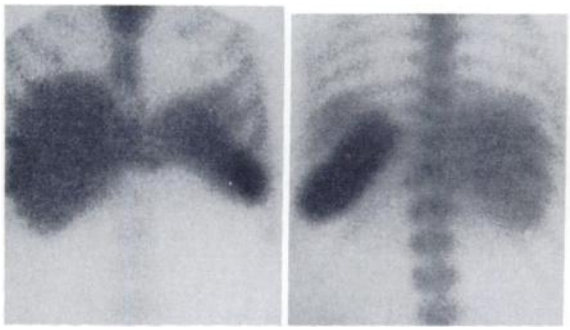

FIG. 2. Anterior (left) and posterior (right) images obtained after administration of Tc-99m sulfur colloid. Liver was 16 cm in length, with uneven radiocolloid distribution. Anterior view shows radioactivity in sternum and ribs. In posterior image, ribs, vertebrae, and tips of scapulae contain radioactivity. Spleen was 14 cm in length (enlarged) and showed major uptake of radiocolloid.

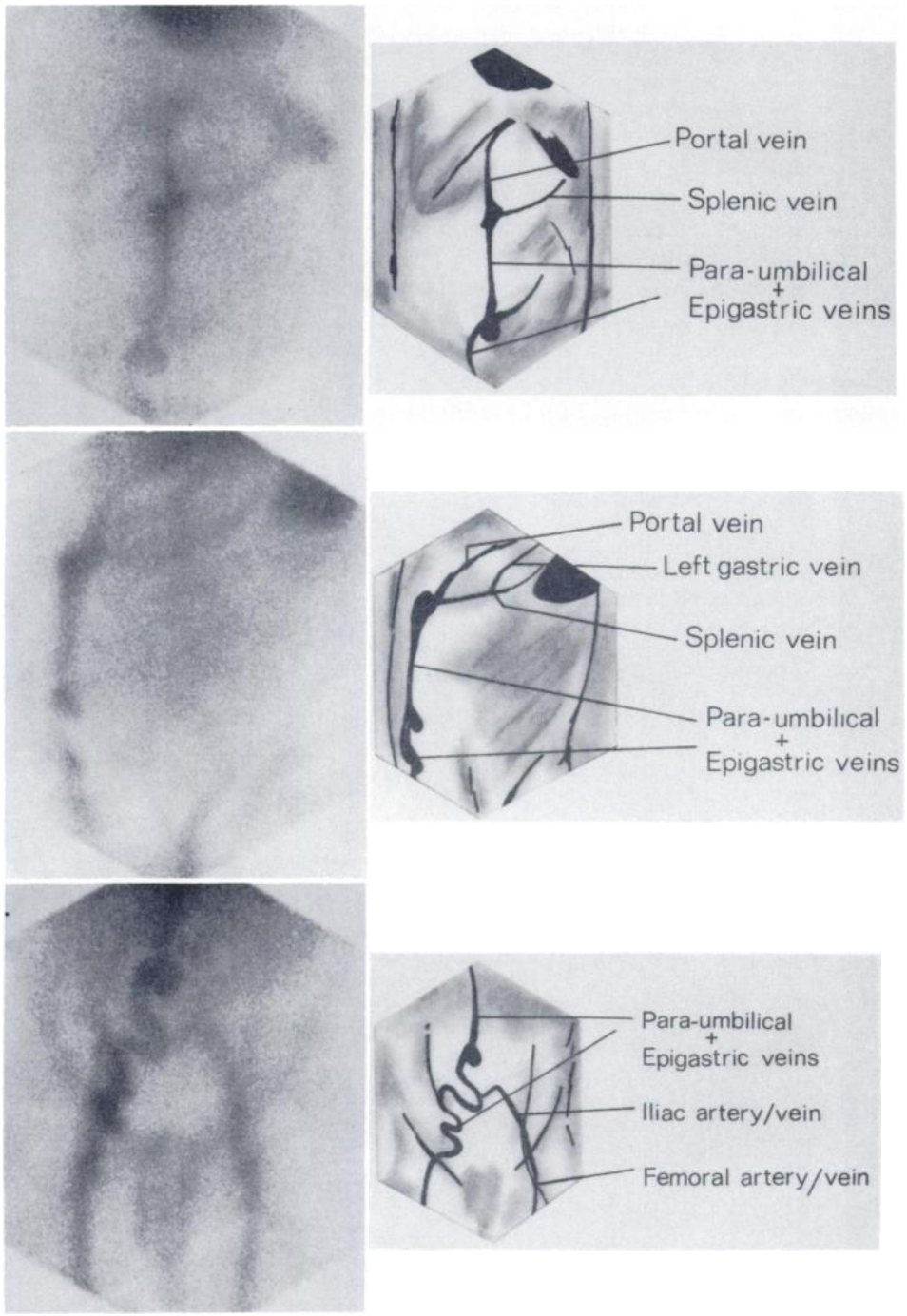

FIG. 3. Blood pool images, with corresponding diagrams of anatomy. Top: Anterior view of abdomen. Middle: Left anterior oblique image of abdomen. Bottom: Anterior view of pelvic region.

Physical examination revealed significant jaundice, marked clubbing of the hands and feet, and ascites. Engorged vessels could be seen on the abdominal wall.

Hepatic scintiangiography was performed in the anterior view after bolus intravenous administration of 6 mCi of Tc-99m sulfur colloid. Images were obtained at intervals of 3 sec. Four consecutive views are shown in Fig. 1. Following hepatic perfusion, a pathway leaving the liver was apparent. This appeared dilated and tortuous; because of its clarity, it was thought to be located far anteriorly. Subsequent static images were compatible with severe hepatocellular disease (Fig. 2). That is, there was uneven hepatic extraction of radiocolloid and a massive shift of radiocolloid to the bone marrow and spleen. Splenomegaly was also present. Sonography confirmed the presence of ascites in the hepatorenal space.

In order to demonstrate conclusively that the aberrant pathway involved the umbilical vein, the patient was given a blood-pool study. Following intravenous administration of stannous pyrophosphate, pertechnetate was injected in order to label the red blood cells. Images showed that the venous pathway was located in or near the anterior abdominal wall, ultimately draining through the right femoral or iliac veins. These views are shown in Fig. 3, along with corresponding anatomical sketches.

DISCUSSION

A patent collateral venous circulation (CVC) has been demonstrated by other imaging modalities, the majority of which are more invasive than hepatic scintigraphy. Visualization of CVC most often indicates the presence of portal hypertension. Other causes include obstruction of the superior or inferior vena cava, superficial venous thrombosis, and emaciation. Such causes can usually be excluded clinically. Burchell and associates found functional umbilical-venous channels in less than 10% of splenoportograms (*6*). The use of percutaneous transhepatic portography by Aagaard and co-workers revealed patent umbilical veins in approximately 25% of studies (*3*). Both of these examinations carry significant potential morbidity and cannot be performed routinely to evaluate the presence of portal hypertension. Dokmeci and associates, using ultrasound (*7*), and Ishikawa's group, using contrast-enhanced CT (*8*), have also been successful in demonstrating CVC in patients with portal hypertension.

The use of the dynamic sequence during hepatic scintigraphy can be useful in identifying CVC. Due to the reduction of activity from CVC with time (when a radiocolloid is used) delayed static hepatic scintigraphy would not display these pathways. Patients with a history of hepatocellular disease are often imaged at least 15 min after injection, because of delayed clearance of radiocolloid from the circulation. If dynamic hepatic scintiangiography is not performed, visualization of CVC would diminish as the radiocolloid is cleared from the circulation by the liver, spleen, and bone marrow. Routine dynamic hepatic scintiangiography could therefore increase the likelihood for the detection of CVC. The addition of imaging with an intravascular label (blood-pool study) could be used effectively to aid in the identification of abnormal vascular channels.

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