jnm/case Report

FALSE-POSITIVE LIVER SCAN CAUSED BY DILATED SPLENIC VEIN

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A 61-year-old patient with splenomegaly exhibited a horizontal defect on liver scan. Hepatic angiography revealed a dilated splenic vein occupying the site of the defect. Other radiographic studies ruled out other etiologies for the scan finding.

In the interpretation of liver scans, knowledge of the anatomy and physiology of the liver and its surroundings may often permit a definitive diagnosis of the abnormality seen. However, not all the "intrahepatic" abnormalities are truly within the liver, and the observing physician should be alerted by findings that appear to defy known anatomic relationships. The following is such an example.

CASE REPORT

A 61-year-old asymptomatic black woman was referred for a liver scan to evaluate her clinically diagnosed hepatomegaly. Splenomegaly was not apparent on the admitting physical examination and the patient had no history of alcohol abuse, congestive heart failure, or hepatitis. The plain films of the abdomen revealed calcification in the left upper quadrant in a linear configuration. The upper GI series and oral cholecystogram were normal. The liver scan, done with ^{99m}Tc-sulfur colloid on a Nuclear-Chicago Pho/Gamma III HP scintillation camera with a high-resolution, low-energy collimator, demonstrated hepatosplenomegaly with a prominent, elongated, horizontal defect in the left hepatic lobe extending into the medial portion of the right lobe. The location of this lesion and its shape are unusual since intrahepatic lesions such as primary or metastatic tumors, abscesses, and vascular abnormalities have a predominately rounded shape. The overall distribution of radiocolloid was diffusely irregular, and these findings were considered consistent with cirrhosis accompanied by portal venous hypertension. Insufficient data were available at that time to ascertain the origin of the horizontal defect.

To define further this abnormality and to ascertain its vascularity, a hepatic perfusion study using sodium pertechnetate was performed the next day. Serial pictures were obtained on Kodak 2495 RAR film in a Nikon 35-mm motor-driven camera with

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Anterior Spleen

18Sec Flow Image

FIG. 1. Anterior liver view with ^{90m}Tc-sulfur colloid shows linear defect in liver. Right lateral liver view demonstrates defect being posteriorly situated. Anterior view of spleen demonstrates splenomegaly and hepatic defect. Flow image demonstrates region of hyperfusion (arrows).

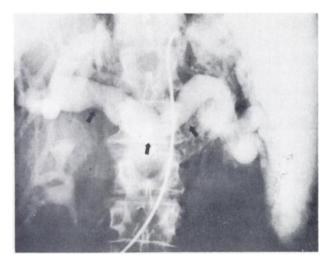


FIG. 2. Abdominal arteriogram shows large spleen and dilated, tortuous splenic vein (arrows).

exposures every 2.5 sec. During the later stages in the "venous phase", 18 sec after aortic visualization, increased perfusion was seen in the same region as above but was somewhat obscured by surrounding hepatic activity. As a result of the above characteristics, these studies were interpreted as showing a vascular mass. Because this was a single lesion, vascular tumors were considered to be the most likely possibility, and it was suggested that it might represent a venous angioma, hepatoma, or possibly a highly vascular metastatic deposit.

Selective superior mesenteric, celiac, and hepatic arterial angiographic injections were performed which showed a dilated tortuous splenic vein exiting the massively enlarged spleen. The intrahepatic vasculature showed changes consistent with cirrhosis. No gastric varices were evident and no tumor vascularity was found.

The dilated tortuous splenic vein follows the same

course as the horizontal vascular defect demonstrated on the sulfur colloid scan. In retrospect both the defect and the perfusion blush exhibited a vaguely serpentine shape corresponding to the tortuous splenic vein.

DISCUSSION

This is an example of an extrinsic cause of an apparently intrahepatic defect. The defect does not correlate with commonly seen phenomena nor with normal intrahepatic anatomical structures. Its appearance in the latter stages of the perfusion study would suggest that it was fed, as is the normal liver, from the portal venous system; however, without the angiographic demonstration of the splenic vein, this would have been construed as being an intrahepatic vascular mass of unknown etiology. Other more common extrahepatic causes for hepatic defects are known: the right kidney, intrahepatic gallbladder, spinal impression, dilated hepatic vein in congestive failure impressing the hepatic dome, the right colic flexure, and the impression of the rib cage in individuals with a large or descended liver accompanied by an obese, flaccid abdomen.

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