TECHNETIUM-99m-SULFUR COLLOID AND PERTECHNETATE BLOOD POOL SCANS IN HEPATIC VENO-OCCCLUSIVE DISEASE: CASE REPORT

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A liver scan on a patient with biopsy-proven hepatic veno-occlusive disease showed multiple areas of decreased sulfur colloid uptake which were not perfused on the blood pool phase of a pertechnetate bolus study.

This report describes the dynamic and static radionuclide findings in a patient with the rare condition of biopsy-proven hepatic veno-occlusive disease (1,2).

CASE REPORT

A 46-year-old woman who had spent the previous 25 years as a nurse in Asia under rather primitive conditions had had a rapidly increasing abdominal girth for 2 months prior to admission. Physical examination revealed a markedly enlarged liver that was firm, nodular, and nontender. Massive ascites was present. Abnormal laboratory findings included alkaline phosphatase 156 IU (normal 30–85 IU), SGOT 74 IU (normal 10–50 IU), lipase 1.8 IU (normal 0–1 IU), and protime 11.3 sec (control 9.6 sec). The chest radiograph was normal. Abdominal radiographs showed evidence of ascites; the liver shadow was prominent but no hepatic calcifications were detected. After administration of 2 mCi of 99mTc-sulfur colloid, scintillation camera images showed hepatomegaly with many large areas of focally absent uptake. The spleen was not enlarged (Fig. 1). During the blood pool phase a bolus injection of 10 mCi 99mTc failed to show any perfusion in the areas of absent sulfur colloid uptake (Fig. 2). Surgery demonstrated a massive ascites, a "somewhat knobby" omentum, and a mildly enlarged liver with a finely scarred surface. The gallbladder, common duct, and spleen appeared normal. Wedge biopsy of the liver revealed a normal architecture but

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FIG. 1. Technetium-99m sulfur colloid scan demonstrates hepatomegaly with multiple areas of absent colloid uptake (upper). Spleen is normal (lower). Linear decreased uptake on anterior liver view is lead marker on right costal margin.
marked acute congestion with dilated sinusoids and small blood lakes. The portal veins were normal. The central veins were dilated and the lumina were filled with a loose fibrous network in which there were small recanalized channels. The pathologic diagnosis was hepatic veno-occlusive disease.

DISCUSSION

The term hepatic veno-occlusive disease is properly applied to an acute type of hepatic vein occlusion involving initially only the small hepatic veins (1,3). The block is due to endothelial damage in the sinusoids and subterminal and terminal hepatic veins, with extravasation of red cells and cellular debris into Disse's space that causes secondary compression of the small veins. However, a loose fibrin network and not thrombosis is found in the involved veins (4). This lesion may be due to certain plant toxins (5), urethane poisoning (6), and radiation injury (7). Many reported cases of idiopathic Budd-Chiari syndrome may actually represent true hepatic veno-occlusive disease (8).

Veno-occlusive disease usually appears in childhood, but has been observed in adults. The common clinical presentation in the acute phase is similar to that of the Budd-Chiari syndrome, i.e., pain, hepatomegaly, and ascites (2,9). The patient may die, recover, or pass into a chronic phase that leads to cirrhosis and portal hypertension. Portacaval anastomosis has been used for relief of portal hypertension in the chronic stages. The diagnosis of veno-occlusive disease is based on the characteristic lesion found on liver biopsy.

In this case of primary veno-occlusive disease the hepatic radionuclide images are not specific (hepatomegaly, multiple large areas of decreased colloid uptake, and a normal spleen). Lack of perfusion in the colloid-deficient areas during the dynamic study decreases the probability of a vascular tumor and the normal-size spleen decreases the probability of portal hypertension. Veno-occlusive disease is often confused with other cases of the Budd-Chiari syndrome, but in the latter (9,10) nearly all patients showed a single large area of decreased colloid uptake in the right hepatic lobe, in contrast to the findings in this patient.

Veno-occlusive disease of the liver should be considered in the differential diagnosis of the liver scan that demonstrates hepatomegaly with multiple areas of decreased colloid uptake and a normal-size spleen.

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REFERENCES