Unusual Liver-Spleen Scan Findings

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Radioisotope scintigraphy is a widely used procedure for the evaluation of various forms of liver disease. It provides information regarding the size, shape, and position of the liver, as well as identification of any space-occupying lesions of the liver or spleen (1-4,6-8). The amount of radiocolloid clearance in liver, spleen, and bone marrow depends on the distribution of radioactivity in reticuloendothelial (RE) tissue, the degree of liver disease, and its effect on relative blood flow (1,9,10). We present an unusual liver-spleen scan demonstrating delayed clearance of blood-pool activity due to portal hypertension and consequent severe RE shift to spleen and bone marrow (1,5,11-13).

CASE REPORT

A 56-yr-old female with a 15-yr history of alcohol abuse presented for a liver-spleen scan. The patient had a longstanding history of alcoholic liver cirrhosis and recent worsening of hepatic function. A liver-spleen scan was performed to evaluate the extent of liver disease.

Fifteen minutes postinjection of technetium-99m- (99mTc) labeled sulfur colloid, the anterior image of the abdomen demonstrated severe reticuloendothelial shift to spleen and bone marrow with minimum uptake of sulfur colloid in the liver and an unusual display of vascular structures, most probably representing the splenic and superior mesenteric veins (Fig. 1). Forty-minute delayed images showed clearance of the sulfur colloid from the blood pool (Fig. 2).

DISCUSSION

At our institution, we perform an average of three to five liver-spleen scans daily to evaluate the degree of liver disease in patients. About 40%-50% of these patients have alcoholic liver disease.

The early film shows vascular structures due to delayed blood clearance of radiocolloid by the reticuloendothelial system. This is a reflection of portal hypertension and demonstrates some of the collateral pathways of venous flow secondary to severe hepatic dysfunction.

Reticuloendothelial shift is defined as the shift in distribution of radiocolloid from Kupffer cells in the liver to the spleen and bone marrow due to shunting of blood (1,8,9,12). Liver fibrosis increases resistance to flow producing portal hypertension. The fibrosis replaces liver sinusoids, which results in decreased hepatic transit. Blood concentration of sulfur colloid is thereby elevated for a period, allowing the spleen and bone marrow to extract more sulfur colloid than usual. Splenomegaly develops due to portal hypertension (1,8,9).

REFERENCES

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