

Case Reports: Nonvisualization of the Liver with Tc-99m Sulfur Colloid

Virgilio A. Valdez*, Ramon N. Kranwinkel, Thomas W. Crucitti, and Nilo E. Herrera

Danbury Hospital, Danbury, Connecticut

Two cases with no accumulation of Tc-99m sulfur colloid in the liver are presented. Our first case describes a patient with a history of cirrhosis and a porto-caval shunt. The second case is a woman with acute alcoholic hepatitis. In both cases, however, the liver was visualized with I-131 rose bengal. A brief review of the reported cases of localized hepatic reticuloendothelial failure is included.

The cells forming the reticuloendothelial system (RES), which are mainly localized in the liver, spleen, and bone marrow, are known to be capable of phagocytizing colloid particulate matter from the blood stream. Because the colloidal particles are accumulated mainly in the Kupffer cells of the liver, Tc-99m sulfur colloid is widely used in the evaluation of liver disease. It is clear that hepatocellular damage might produce different degrees of Kupffer cell dysfunction.

Case Reports:

Case No. 1: A 55-year-old white man was admitted in 1968 for evaluation of ascites and swelling of both lower extremities. He admitted to drinking 6 to 10 oz of alcohol daily for many years. Liver function tests revealed a bilirubin of 3 mg/dl; alkaline phosphatase—12.4 K.A. (normal: up to 20); LDH—380 units (normal: up to 350); total protein—6.2 g; albumin—1.9 g/dl; and globulin—4.3 g/dl. Upper gastrointestinal series showed varices in the distal esophagus. An Au-198 colloid liver scan revealed an enlarged organ with irregular distribution of the radionuclide. A diagnosis of cirrhosis of the liver was made. Following treatment, the patient made an uneventful improvement.

In 1969 and 1973, he was admitted because of iron deficiency anemia secondary to bleeding esophageal varices. In November, 1973, an end-to-side porto-caval shunt was done. At the same time a liver biopsy was performed and revealed nutritional type of cirrhosis. Postoperatively, the patient did well.

He was readmitted in January, 1978, with the chief complaint of increasing fluid in his legs. On physical examination he was jaundiced, and had gynecomastia, multiple small spider nevi, hepatosplenomegaly, scrotal edema, and 3+ pitting edema of both lower extremities. Laboratory studies revealed: bilirubin—15 mg/dl; alkaline phosphatase—116 I.U. (normal: 20–90); SGOT—41 I.U./l (normal: 3–20); LDH—453 I.U./l (normal: 130–290); total protein—6.4; albumin—1.9 g/dl; Hct—27.5% and Hgb—9.4 g/dl. A Tc-99m sulfur colloid liver-spleen scan showed nonvisualization of the liver and an enlarged spleen (Fig. 1). In addition,

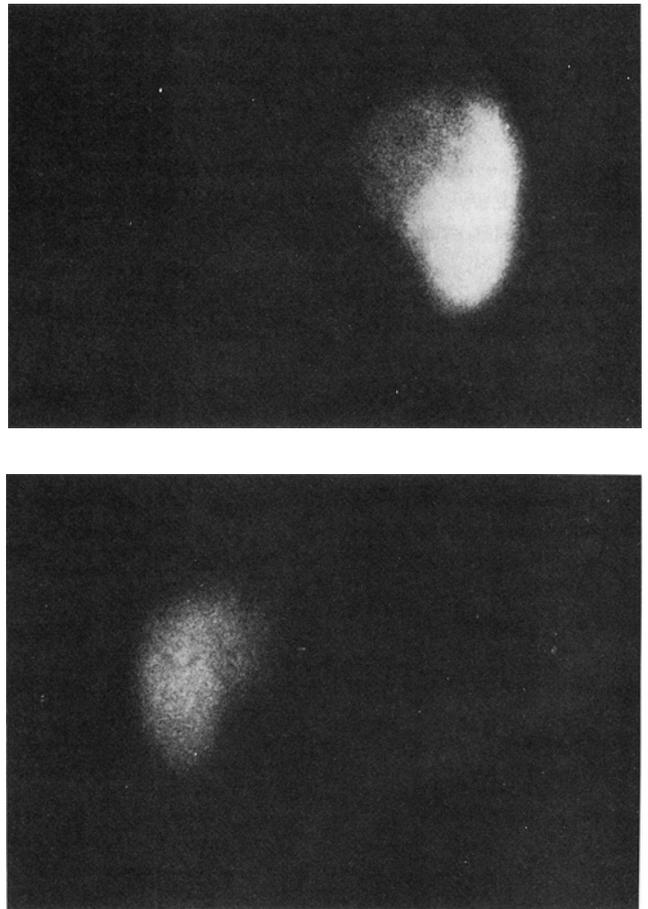


FIG. 1. Tc-99m sulfur colloid study shows anterior (top) and posterior (bottom) views. The liver is not visualized; note the enlarged spleen.

For reprints contact: Nilo E. Herrera, Director of Laboratories, Danbury Hospital, Danbury CT 06810.

* Present address: Div. of Nuclear Medicine, Cornell Medical Center, The New York Hospital, 568 E. 68 St., New York, NY 10021.

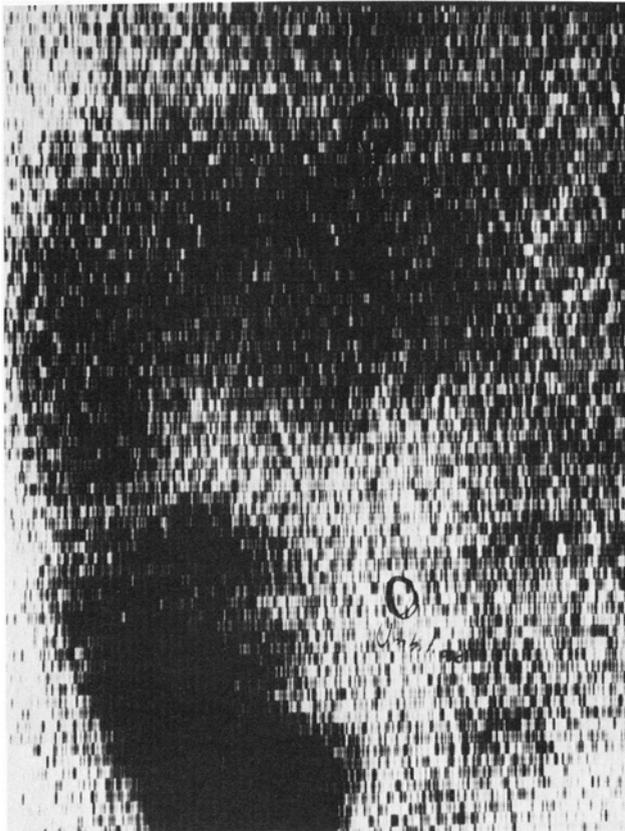


FIG. 2 Anterior rectilinear scan 4 hr after administration of I-131 rose bengal. The enlarged liver shows irregular uptake in the right lobe; note activity within the bowel.

some of the views showed uptake along the bone marrow of the skeleton of the trunk. An I-131 rose bengal liver scan showed an enlarged organ with areas of decreased uptake in the right lobe. There was excretion of the radionuclide into the intestine (Fig. 2). Following the administration of 3.2 mCi of Ga-67, views of the hepatic region were obtained at 24, 48, and 72 hr. The uptake in the liver was minimal. After adequate therapy, the patient improved and was discharged.

Case No. 2: A 39-year-old woman was admitted to the hospital with a one-week history of jaundice and increasing abdominal girth. The patient admitted to be a daily drinker of 1 to 1 1/2 gal of wine for years. Pertinent physical findings included icteric sclera and distended abdomen with ascites; the liver was palpable 5 cm below the right costal margin. Laboratory studies included: Hgb—12.8 gm/dl; Hct—38.2%; sed rate—50mm/hr; total protein—5.5 g; protein electrophoresis revealed low albumin; HAA—negative; bilirubin—10.8 mg/dl; direct bilirubin—6.1 mg/dl; alkaline phosphatase—248 I.U. (normal: 20–90); SGOT—72 I.U.; LDH—389 I.U.; and SGPT—16 I.U./l.

A Tc-99m sulfur colloid liver-spleen scan showed virtually no uptake in the liver. The spleen was enlarged and there was diffuse increased uptake in the bone

marrow of the spinal column and ribs (Fig. 3). An I-131 rose bengal scan was performed. Curves obtained after placing probes over the precordium and hepatic regions revealed significant delay in the pattern of plasma clearance and hepatic uptake. The static images showed an enlarged liver. There was excretion of the radionuclide into the intestine (Fig. 4). After adequate treatment, the patient improved.

The clinical diagnosis was acute alcoholic hepatitis. No liver biopsy was obtained.

Discussion

Uneven distribution of radioactivity within the liver may accompany diffuse parenchymal disease from many causes (1). The failure of the hepatic reticuloendothelial cells to accumulate radiocolloid has been reported in the past. Among the reported causes are: familial erythrophagocytosis (2); intestinal bypass (3); localized

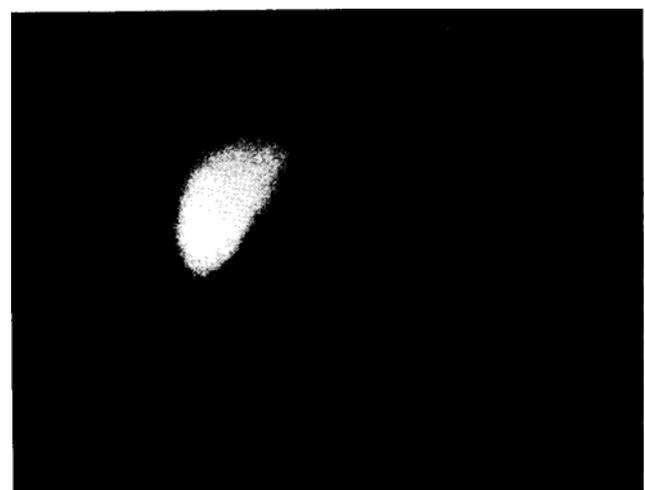
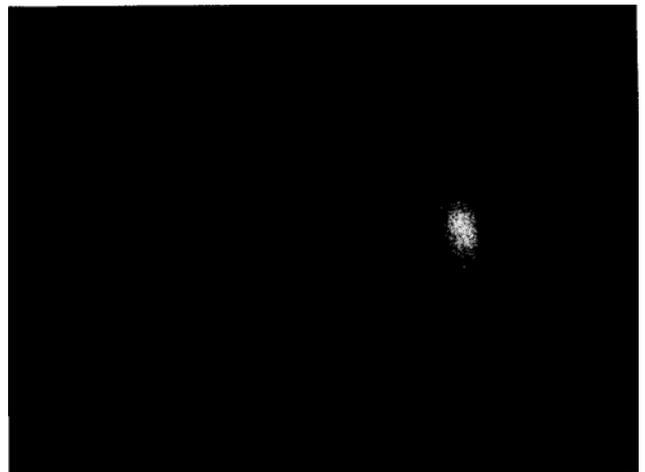


FIG. 3. Anterior (top) and posterior (bottom) views following the administration of Tc-99m sulfur colloid. There is little uptake in the liver. The spleen is enlarged and prominent; note increased uptake in ribs and spinal column.

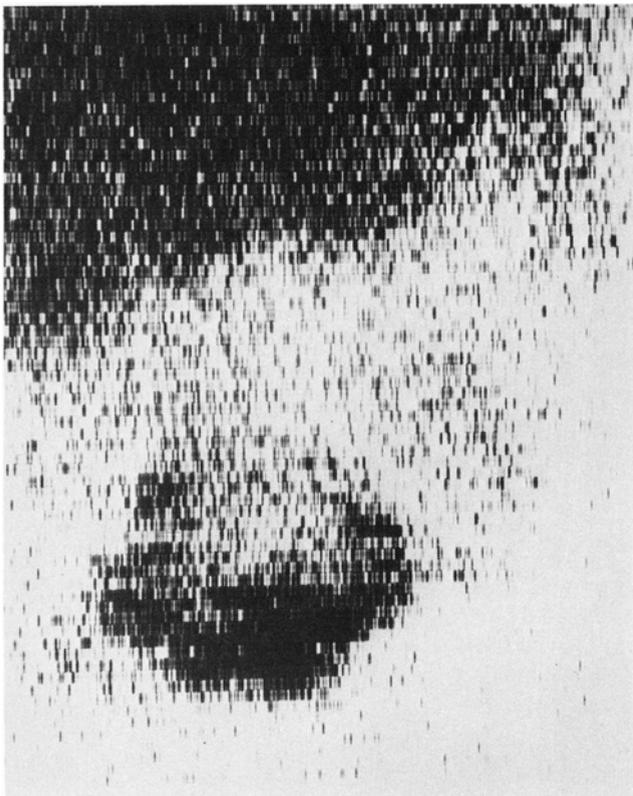


FIG. 4. Anterior rectilinear view 4 hr after administration of I-131 rose bengal. The liver is visualized and there is activity within the bowel.

hepatic irradiation (4); *Schistosoma mansoni* infestation (5); and advanced cirrhosis (6).

When the reticuloendothelial function of the liver is severely impaired, there is compensatory hyperactivity of the rest of the RES, especially in the spleen and bone marrow (1). It has been demonstrated that when the Kupffer cells fail to accumulate Tc-99m sulfur colloid, the use of other radiopharmaceuticals is of great value in the evaluation of liver function and morphology (5).

Chronic alcoholic liver disease generally produces nonuniform accumulation of the radionuclide with so-called "mottled" appearance and increased uptake in spleen and bone marrow. Our first case, in addition to the history of cirrhosis, had a porto-caval shunt. Hepatic blood flow is considerably reduced after a portal systemic shunt. The amount of reduction of the flow depends on the type of shunt as well as the severity of the liver disease

(7). In our second case, although no biopsy was obtained, the clinical picture was consistent with acute alcoholic hepatitis. This can be added to the list of conditions that may cause hepatic reticuloendothelial failure. Acute alcoholic hepatitis generally produces enlargement of the organ. It has been reported that marked sclerosis of the centrilobular sinusoids and veins may be present and lead to partial obliteration of the venous flow (8). This may be postulated as a mechanism for the nonvisualization of the liver in this case.

It has been theorized that hepatic arterial inflow could increase to compensate for the reduction in the portal circulation, but to what extent this occurs is unknown (7). Another factor to consider in both cases is that the I-131 rose bengal is accumulated by the hepatocytes, a function that is independent of the reticuloendothelial system. Therefore, when imaging the liver using I-131 rose bengal, there is little competition for this radiopharmaceutical, whereas there is competition for the colloid by the liver, spleen, and bone marrow.

Acknowledgment

Appreciation is extended to the staff of the nuclear medicine department and to Mrs. Joan Ackert, secretary.

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