trated through water absorption (*7*). Excess biliary excretion due to improper radiopharmaceutical preparation can be excluded as the cause since there was no visualization of the liver in the serial bone scan at any time and other bone scans performed on the same day showed a normal distribution of tracers.

REFERENCES

1. Vardy PA, Lebenthal E, Schwachman H. Intestinal lymphangiectasia: a reappraisal. *Pediatrics* 1975;55:842–851.
2. Puri AS, Aggarwal R, Gupta RK, et al. Intestinal lymphangiectasia: evaluation by CT and scintigraphy. *Gastrointest Radiol* 1992;17:119–121.
3. Fakhri A, Fishman EK, Jones B, Kuhajda F, Siegelman SS. Primary intestinal lymphangiectasia: clinical and CT findings. *J Comp Assist Tomog* 1985;9:767–770.
4. Soucy JP, Eybalin MC, Taillefer R, Levasseur A, Jobin G. Lymphoscintigraphic demonstration of intestinal lymphangiectasia. *Clin Nucl Med* 1983;8:535–537.
5. Mistilis SP, Skyring AP, Stephan DD. Intestinal lymphangiectasia: mechanism of enteric loss of plasma-protein and fat. *Lancet* 1965;1:77–79.
6. Divgi CR, Lisann NM, Yeh SDJ, Benua RS. Technetium-99m-albumin scintigraphy in the diagnosis of protein-losing enteropathy. *J Nucl Med* 1986;27:1710–1712.
7. Conway JJ, Weiss SC, Khentigan A, Tofe AJ, Thane TT. Gallbladder and bowel localization of bone imaging radiopharmaceuticals [Abstract]. *J Nucl Med* 1979;20: 622.

Fulminant Hepatic Failure Monitored by Technetium-99m-DTPA-Galactosyl-Human Serum Albumin Scintigraphy

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We describe a 43-yr-old woman with fulminant hepatic failure whose progress was monitored scintigraphically using ^{99m}Tc-galactosyl-human serum albumin (^{99m}Tc-GSA). On admission, the liver was atrophic and the heart was delineated distinctly by scintigraphy with ^{99m}Tc-GSA. The receptor index, calculated by dividing the radioactivity of the liver region of interest by the radioactivity of the liver plus heart regions of interest at 15 min post-tracer injection, was very low. As the patient's condition improved, the right lobe of the liver enlarged while the left lobe became atrophic; after 4 mo, the left lobe almost completely disappeared. Delineation of the heart gradually became less distinct, and the receptor index slowly increased. Hepatic receptor imaging with ^{99m}Tc-GSA can define both the hepatic functional reserve and morphological changes of the liver, so it is useful for the diagnosis and follow-up study of fulminant hepatic failure.

Key Words: fulminant hepatic failure; technetium-99m-GSA; hepatic receptor imaging

J Nucl Med 1996; 37:641–643

Technetium-99m-phytate and sulfur colloid, which have been used as liver imaging agents, are transported to the liver and taken up by Kupffer cells after intravenous injection (*1*). The hepatocyte-oriented radiotracer ^{99m}Tc galactosyl-neoglycoalbumin (^{99m}Tc-GSA), developed as a receptor-binding radiopharmaceutical for noninvasive assessment of liver function, is a synthetic radioligand to the asialoglycoprotein receptor (hepatic-binding protein), which resides on the plasma membrane of liver cells. Upon intravenous injection, ^{99m}Tc-GSA is directed to hepatocytes because of its chemical recognition and binding by a specific receptor of hepatic-binding protein. After binding, it is transferred to hepatic lysosomes by receptor-mediated endocytosis (*2,3*).

The use of ^{99m}Tc-GSA enables us not only to evaluate hepatic function in patients with diffuse liver diseases but also to assess morphological changes of the liver. We describe a patient with fulminant hepatic failure who was evaluated scintigraphically with ^{99m}Tc-GSA.

Received Mar. 3, 1995; revision accepted Jun. 29, 1995.

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CASE REPORT

A 43-yr-old woman consulted a physician because of general fatigue and clouding of consciousness. The results of clinical tests showed severe liver dysfunction. She was referred to our hospital for further examination and therapy. On admission, physical examination showed jaundice and ascites, and there was clouding of consciousness. Her white blood cell count was 10,100/mm^3, red blood cell count was 362 × 10^4/mm^3, total bilirubin was 13.2 mg/dl, aspartate aminotransferase was 109 IU/liter, alkaline phosphatase was 372 IU/liter, serum albumin was 2.8 g/dl, lactate dehydrogenase was 512 WU/liter, and the prothrombin time was 25%. Anti-hepatitis A antibody, hepatitis B surface antigen and hepatitis C virus antibody were not detected. Hepatic injury, caused by diclofenac sodium, was diagnosed by results of a lymphocyte stimulation test. On abdominal CT, the liver showed extensive low-density regions and atrophy of both lobes (Fig. 1).

The patient responded to intensive therapy including plasmapheresis. Hepatic receptor imaging with ^{99m}Tc-GSA was performed four times (on admission and after 1, 2 and 4 mo). One 185-MBq dose of ^{99m}Tc-GSA was injected intravenously and dynamic imaging was performed with the patient supine under a large field of view gamma camera with a low-energy, all-purpose parallel-hole collimator. Computer acquisition of the gamma camera data was started just before injection of ^{99m}Tc-GSA and was stopped 20 min later. Digital images (128 × 128 pixels) were acquired in the byte mode at a rate of 60 sec/frame. Accumulation images of the anterior abdominal view were obtained at 20 min after the injection. Time-activity curves for the heart and liver were generated from regions of interest (ROIs) for the whole liver and precordium. The receptor index (LHL15) was calculated by dividing the radioactivity of the liver ROI by the radioactivity of the liver plus heart ROIs at 15 min after the injection.

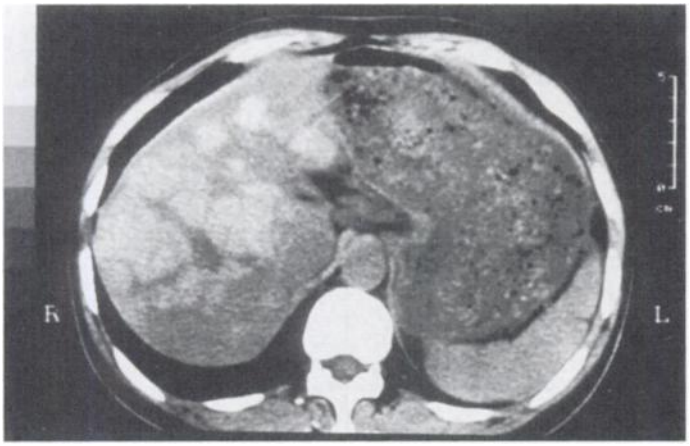

FIGURE 1. Abdominal CT shows extensive low-density regions and atrophy of both lobes in the liver.

When the patient was admitted to our hospital, both lobes of the liver were atrophic and the heart was delineated distinctly (Fig. 2). The LHL15 at that time was very low (Table 1). The range of LHL15 for six healthy subjects was 0.914–0.956, and the mean ± s.d. was 0.936 ± 0.015 (*3*). The patient's condition improved, and at the same time the right lobe of the liver enlarged while the left lobe became atrophic; after 4 mo, the left lobe almost completely disappeared (Fig. 2). Delineation of the heart gradually became less distinct and LHL15 slowly increased (Table 1).

DISCUSSION

In recent years, the survival rates in patients with fulminant hepatic failure have improved, although the mortality remains high (*4*). Various blood biochemical tests have been used for evaluation of the hepatic functional reserve (*5,6*), but it is not always possible to assess hepatic functional reserve in patients with fulminant hepatic failure as they often undergo plasmapheresis and blood product supplementation. Imaging techniques such as liver scintigraphy (*7–9*), abdominal CT scan (*10*) and abdominal ultrasonography (*11*) have been found useful in the diagnosis of diffuse hepatic disease such as fulminant hepatic failure. Liver scintigraphy with a radiocolloid agent is especially helpful in establishment of a diagnosis of fulminant hepatic failure as it facilitates understanding of the hepatic functional reserve. Waxman (*7*) noted in a review the presence of hepatomegaly in 6 (46%) of 13 survivors of fulminant hepatic failure and found no hepatomegaly among the 9 patients who died. Pulmonary uptake was not seen in any of the survivors; on the other hand, it was seen in 3 (33%) of the 9 nonsurvivors. Previously, we performed liver scintigraphy with ^{99m}Tc-phytate in 44 patients with acute hepatitis and 12 patients with fulminant hepatic failure and found that evidence of liver atrophy and redistribution of the radiocolloid to the bone marrow is useful in establishing a diagnosis of fulminant hepatic failure (*8*). Of the 12 patients with fulminant hepatic failure, pulmonary uptake was not seen in any of the 7 survivors but was seen in 3 of the 5 nonsurvivors.

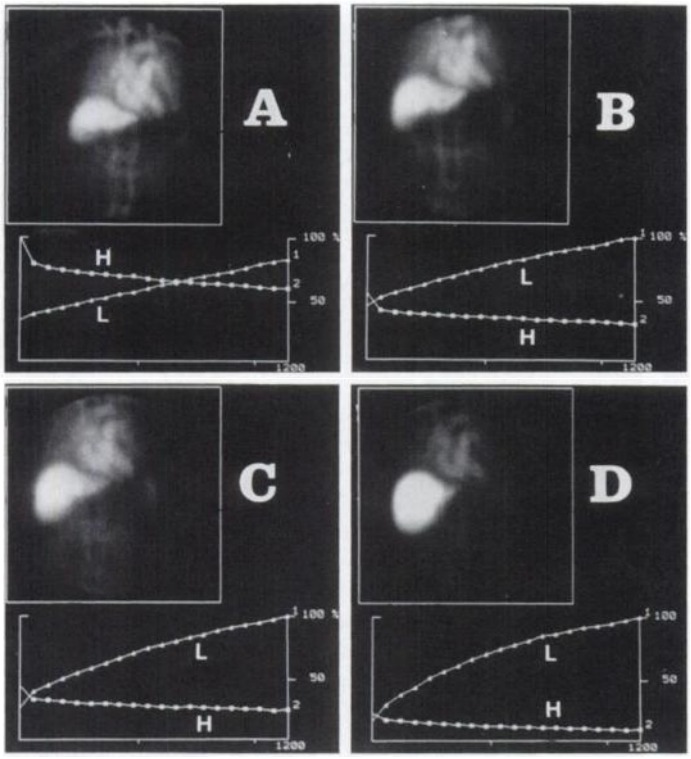

FIGURE 2. Summed images (20 min) and time-activity curves for the liver and heart after injection of ^{99m}Tc-GSA. (A) On admission, (B) after 1 mo, (C) after 2 mo and (D) after 4 mo. L = liver; H = heart.

TABLE 1
Changes in LHL15 Values and Laboratory Data

	On admission	1 mo	2 mo	3 mo
LHL15	0.58	0.70	0.77	0.88
PT (%)	25	40	52	82
Total bilirubin (mg/dl)	13.2	3.6	1.6	1.0

LHL15 is calculated by dividing the radioactivity of the liver plus heart ROIs at 15 min postinjection of ^{99m}Tc-GSA.
PT = prothrombin time.

CONCLUSION

Hepatic receptor imaging with ^{99m}Tc-GSA is an accepted new diagnostic approach for hepatic disease (*2,3*). The results of liver scintigraphy with a radiocolloid are affected by the activity of Kupffer cells. Therefore, more reduced uptake into the liver, a condition termed hepatic reticuloendothelial failure, may occasionally be detected among abusers of alcohol (*12,13*). Hepatic receptor imaging with ^{99m}Tc-GSA, however, is affected only by the function of hepatocytes and not by that of Kupffer cells. Furthermore, hepatic receptor imaging with ^{99m}Tc-GSA permits numerical evaluation of the hepatic functional reserve in terms of the LHL15. If analysis of a series of patients shows satisfactory results, this method might provide a basis for a more objective diagnosis than before. The technique also facilitates the diagnosis of morphological changes in the liver, as seen in our case. In view of these advantages, ^{99m}Tc-GSA scintigraphy of the liver may gain acceptance for use in the clinical diagnosis and follow-up of fulminant hepatic failure.

REFERENCES

1. Stein HS, McAfee JG, Subramanian G. Preparation, distribution and utilization of technetium-99m-sulfur colloid. *J Nucl Med* 1966;7:665–675.
2. Stadalnik RC, Vera DR, Woodle ES, et al. Technetium-99m GSA functional hepatic imaging: preliminary clinical experience. *J Nucl Med* 1985;26:1233–1242.
3. Kudo M, Todo A, Ikekubo K, Hino M. Receptor index via hepatic asialoglycoprotein receptor imaging: correlation with chronic hepatocellular damage. *Am J Gastroenterol* 1992;87:865–870.
4. O'Grady JG, Gimson AES, O'Brien CJ, Pucknell A, Hughes RD, Williams R. Controlled trials of charcoal hemoperfusion and prognostic factors in fulminant hepatic failure. *Gastroenterology* 1988;94:1186–1192.
5. Christensen E, Bremmelgaard A, Bahnsen M, Andreasen PB, Tygstrup N. Prediction of fatality in fulminant hepatic failure. *Scand J Gastroenterol* 1984;19:90–96.
6. Harrison PM, O'Grady JG, Keays RT, Alexander GJM, Williams R. Serial prothrombin time as prognostic indicator in paracetamol induced fulminant hepatic failure. *Br Med J* 1990;301:964–966.
7. Waxman AD. Scintigraphic evaluation of diffuse hepatic disease. *Semin Nucl Med* 1982;12:75–88.

8. Shiomi S, Ikeoka N, Minowa T, et al. Diagnostic value of liver scintigraphy in fulminant hepatitis and severe acute hepatitis. *Acta Hepatol Jpn* 1985;26:592–597.
9. Fleischer MR, Sharpstone P, Osborn SB, Williams R. Liver scintiscanning in acute hepatic necrosis. *Br J Radiol* 1971;44:401–402.
10. Kumahara T, Muto Y, Moriwaki H, Yoshida T, Tomita E. Determination of the integrated CT number of the whole liver in patients with severe hepatitis: as an indicator of the functional reserve of the liver. *Gastroenterol Jpn* 1989;24:290–297.
11. Kurtz AB, Rubin CS, Cooper HS, et al. Ultrasound findings in hepatitis. *Radiology* 1980;136:717–723.
12. Antar MA, Sziklas JJ, Spencer RP. Liver imaging during reticuloendothelial failure. *Clin Nucl Med* 1977;2:293–295.
13. Rao BK, Weir GJ Jr, Lieberman LM. Dissociation of reticuloendothelial cell and hepatocyte functions in alcoholic liver disease: a clinical study with a new ^{99m}Tc-labeled hepatobiliary agent. *Clin Nucl Med* 1981;6:289–294.

Positive Technetium-99m-Red Blood Cell Gastrointestinal Bleeding Scan after Barium Small-Bowel Study

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A 53-yr-old man with hepatic insufficiency and portal hypertension was hospitalized and underwent a work-up for gastrointestinal bleeding requiring multiple transfusions. The initial evaluation included a negative upper and lower endoscopy and a barium exam of the small bowel. Both studies failed to demonstrate any pathology to explain the bleeding. Immediately following the barium study, the patient had active bleeding. Because of the significant amount of intestinal barium, angiography was deferred. Technetium-99m-red blood cell (RBC) scintigraphy was undertaken to identify the site of bleeding. Despite intestinal barium, the ^{99m}Tc-RBC scan demonstrated an active bleeding site in the small bowel in the left abdomen. Therefore, ^{99m}Tc-RBC scintigraphy can be of clinical utility for identification of gastrointestinal bleeding, despite the presence of intestinal barium.

Key Words: technetium-99m; gastrointestinal tract hemorrhage; barium; attenuation

J Nucl Med 1996; 37:643–645

Technetium-99m-red blood cell (RBC) scintigraphy is an established technique for identification and localization of gastrointestinal bleeding. We report a case in which scintigraphy performed 2 hr after a barium small bowel examination demonstrated active bleeding. Concern about the effects of retained barium led us to explore the factors affecting photon attenuation. We describe a phantom experiment performed to investigate the attenuation of different barium solutions and discuss the underlying physics principles.

CASE REPORT

A 53-yr-old man with a history of cirrhosis and portal hypertension secondary to ethanol abuse presented for weakness. Because of a hematocrit of 25, which was significantly lower than his baseline, and guiac-positive stools, the patient was hospitalized and a work-up for gastrointestinal bleeding was undertaken. He was transfused 7 units of packed RBCs and 2 units of fresh-frozen plasma during the 48 hr after admission, and no further bleeding was noted for 4 days. Upper and lower endoscopy was negative. Prior to anticipated discharge, the patient underwent a small bowel follow-through radiographic exam, which involved the ingestion of 900 cc barium sulfate solution [45% weight-to-weight (w/w)]. Immediately following the normal small bowel exam, there was evidence of rebleeding when the patient passed a maroon stool mixed with bright red blood and barium. The radiology department was consulted about obtaining an angiogram. After discussion, however, it was concluded that angiography was inappropriate at that time and was deferred to await clearance of the barium (Fig. 1A). Scintigraphy instead was suggested.

Within 2 hr, a tagged red blood cell study was initiated to identify the site of bleeding. The patient's blood was labeled with 30.0 mCi (1110 MBq) ^{99m}Tc using the in vitro kit technique; analog images containing 500,000 counts were obtained at 5-min intervals with simultaneous computer acquisition using 1-min frames for 90 min. A changing pattern of abnormal activity was noted in the lower abdomen. Review of the cine and analog images allowed identification of intestinal bleeding and localization to the mid to distal small bowel (Fig. 1B-D). In addition, there was scintigraphic evidence of ascites with a pattern of decreased peripheral activity and relatively increased central abdominal activity (*1*).

During the 24 hr after the RBC scan, the patient intermittently passed stools variably maroon or mixed with bright red blood. The patient received 2 units of packed red cells during that period, and an additional 2 units during the subsequent 24 hr, during which time he remained clinically stable. The patient's total transfusion requirement during the hospitalization was 11 units of packed red cells and 2 units of fresh-frozen plasma.

Three days after the small bowel exam and bleeding episode, the patient underwent angiography of the superior mesenteric, inferior mesenteric and gastroduodenal arteries. Angiography demonstrated hepatofugal blood flow with porto-systemic collaterals (*2,3*) compatible with portal hypertension secondary to cirrhosis, but did not demonstrate any active bleeding or pathology to explain prior bleeding episodes.

In the absence of further bleeding and a stable hematocrit, the patient was discharged without further intervention, leaving the pathological diagnosis unresolved.

Phantom Study

A simple phantom experiment was performed to investigate the relative attenuation of different barium solutions compared to water (as an approximation to soft tissue). [Typically, barium preparations are described in terms of percent w/w, which indicates the number of grams of barium sulfate per gram of final prepared

Received Mar. 16, 1995; revision accepted Jul. 14, 1995.
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