

## INTRAHEPATIC FOCAL LESION IN ACUTE VIRAL HEPATITIS

Mordecai Koenigsberg and Leonard M. Freeman

*Albert Einstein College of Medicine of Yeshiva University  
and The Bronx Municipal Hospital Center, Bronx, New York*

***A case of viral hepatitis exhibiting a large focal defect on  $^{99m}\text{Tc}$ -colloid scintigraphy is presented. Uptake of  $^{75}\text{Se}$ -selenomethionine was shown in this region. The appearance returned to normal over a 6-month period. Familiarity with this appearance should help avoid misinterpretation of space-occupying disease.***

The many possible causes of focal lesions on colloid liver scans have been reviewed recently (1). Viral hepatitis is generally not considered when one encounters such localized defects. This report shows that massive focal inflammation in viral hepatitis also may produce a localized diminution of colloid uptake in liver scintigraphy.

### CASE REPORT

A 15-year-old black female was admitted with jaundice as well as bizarre behavior. She had been well until 4 weeks earlier when she first noticed that her eyes were yellow. Evaluation at another institution revealed abnormal liver chemistries. She was followed as an outpatient but became anorectic and weak. Bizarre behavior developed 2 days before admission, progressing to unresponsiveness on the day of admission. No history of hepatitis contact or drug addiction was elicited.

On physical examination the patient was very lethargic and deeply jaundiced. A smooth liver edge was palpable 1–2 fingerbreadths below the right costal margin. The spleen was not palpable. Minimal response to painful stimuli was elicited, and her reflexes were hyperactive but symmetrical. No asterix was shown.

Laboratory results revealed an hematocrit of 46.6%, WBC 9,800/mm<sup>3</sup> with a normal differential. Total bilirubin was 35 mg/100 ml (24 mg/100 ml direct), SGOT 300 units/ml, SGPT 490 units/ml, LDH 670 units/ml, and the alkaline phosphatase 40.0 K-A units/ml. The total serum protein was 6.4

gm/100 ml (albumin 2.0 gm/100 ml), and the prothrombin time was prolonged to 26 sec (control of 12 sec). No Australian antigen was detected. Blood urea nitrogen, glucose, and electrolytes were within normal limits. Electroencephalography showed a diffusely abnormal record.

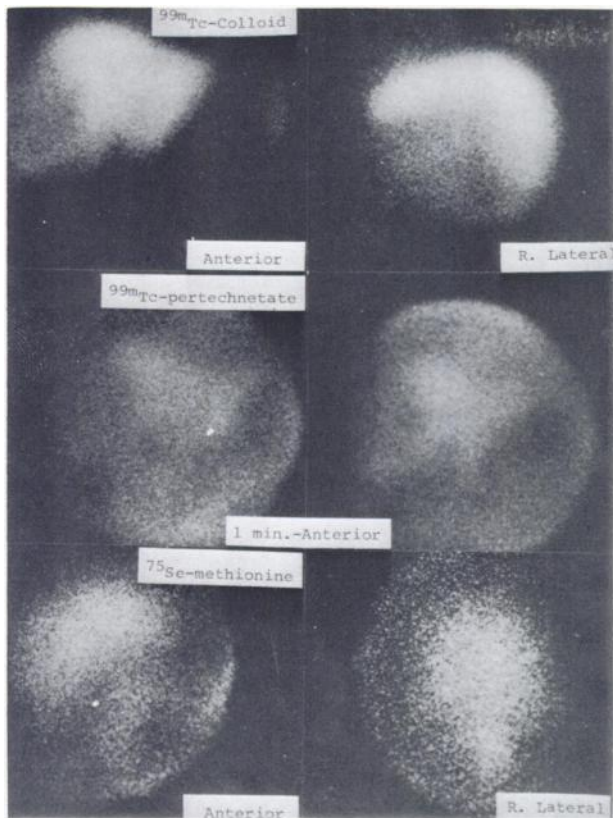
The patient was placed on supportive therapy with fresh frozen plasma, low protein diet, neomycin enemas, and aquamephyton. Very slow improvement was noted, and prebiopsy  $^{99m}\text{Tc}$ -sulfur colloid scintigraphy 1 month after admission showed a large rounded defect in the posterolateral aspect of the right lobe of the liver (Fig. 1, top). The lesion was relatively avascular on a dynamic  $^{99m}\text{Tc}$ -pertechnetate study (Fig. 1, middle), but  $^{75}\text{Se}$ -selenomethionine examination showed incorporation (Fig. 1, bottom). Celiac angiography confirmed the presence of a large avascular mass in the lateral portion of the right lobe of the liver. The vessels near the mass were irregular, but no true hypervascularity was seen. Liver biopsy (Fig. 2) of the avascular region showed marked swelling of hepatocytes (4–5 times normal), multinucleated giant cells, and bile stasis. Kupffer cells were markedly diminished in number in the examination of numerous fields. The appearance was consistent with viral hepatitis.

At this time total bilirubin was 11.2 mg/100 ml (10.8 mg/100 ml direct), SGOT 134 units/ml, SGPT 52 units/ml, LDH 440 units/ml, and the alkaline phosphatase 47.5 K-A units/ml. The prothrombin time was 17.2 sec (control of 11.7 sec).

The patient continued to improve and after discharge was followed as an outpatient. Six weeks after the first study, repeat  $^{99m}\text{Tc}$ -sulfur colloid scintigraphy showed some diminution in the size of the right lobe defect (Fig. 3, top). A  $^{75}\text{Se}$ -selenomethionine study

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For reprints contact: Mordecai Koenigsberg, Albert Einstein College of Medicine, Dept. of Radiology, 1300 Morris Park Ave., Bronx, N.Y. 10461.



**FIG. 1.** Radionuclide imaging studies on 6/22/72 during acute hepatitis. Technetium-99m-colloid images (top row) show large focal defect posterolaterally in right lobe. Cumulative exposure of  $^{99m}\text{Tc}$ -pertechnetate flow study (middle row) shows same region to be avascular. Selenium-75-selenomethionine examination (bottom row) reveals good uptake in same area. Pancreas is also visualized beneath liver.

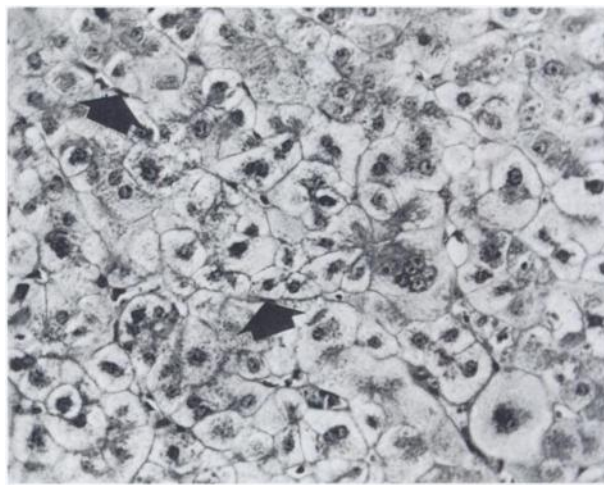
once again showed active protein synthesis in this region. She felt well at this time. Total bilirubin was 1.5 mg/100 ml, SGOT 72 units/ml, SGPT 60 units/ml, LDH 370 units/ml, and the alkaline phosphatase 37.5 K-A units/ml. The total serum protein was 8.0 gms/100 ml (albumin 3.4 gm/100 ml).

She remained well clinically, and 4 months later the liver was normal in appearance on re-examination with  $^{99m}\text{Tc}$ -sulfur colloid (Fig. 3, bottom). Blood chemistries were normal at this time.

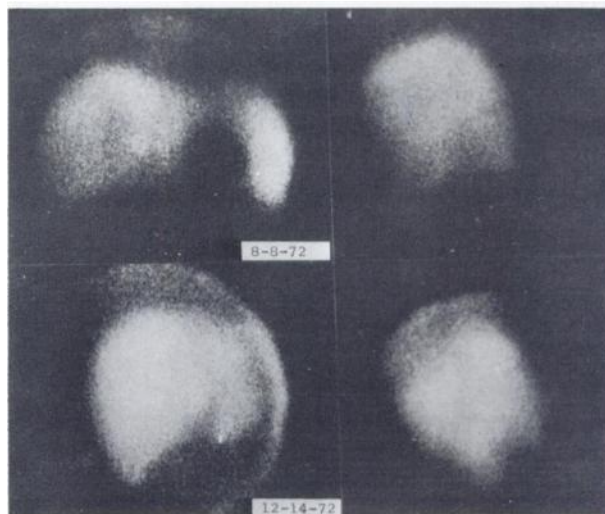
#### DISCUSSION

The detection of space-occupying disease within the liver using labeled colloid is predicated upon the concomitant destruction or loss of function of parenchymal and reticuloendothelial cells. The presence of avid  $^{75}\text{Se}$ -selenomethionine uptake in an area of poor  $^{99m}\text{Tc}$ -colloid activity is seen more frequently in neoplastic processes (2). A disease entity producing marked reticuloendothelial destruction or dysfunction while exhibiting avid radiomethionine uptake in non-neoplastic hepatocytes is an unusual combination of events.

Though viral hepatitis characteristically shows simultaneous involvement of the whole liver, regions of more severe involvement in a liver otherwise showing spotty necrosis have been described (3). The marked swelling of parenchyma in these segmental regions may partly explain the dissociation of RES function and hepatocyte protein metabolic activity noted in this case. In the biopsy fields reviewed, the hepatocytes appear to be increased 4–5 times normal in size. This would represent an 8–12-fold increase in overall parenchymal cell volume, which would be reflected as a comparable decrease in the relative distribution of Kupffer cells, assuming that the latter underwent no change



**FIG. 2.** Photomicrograph of liver biopsy specimen showing changes of viral hepatitis. Swelling of hepatocytes, multinucleate giant cells and bile stasis are evident. Kupffer cells (arrows) are markedly diminished and identified with difficulty (H and E, X 340).



**FIG. 3.** Serial  $^{99m}\text{Tc}$ -colloid scintigrams during and following recovery from hepatitis. Anterior and right lateral images 6 weeks after initial study (top row) show area of diminished uptake to be less well defined. Similar views at 6 months (bottom row) show return to normal-appearing liver.

of their own. Using this assumption, even if the Kupffer cells functioned normally, one would expect the radioactivity due to phagocytized colloid in this region to be only one-eighth to one-twelfth of that noted in adjacent areas with little or no parenchymal swelling.

The swollen hepatocytes may further act to reduce focally clearance of colloid by constriction of the vascular channels. This is reflected by the avascular appearance noted in the radiopertechetate flow study. Selenomethionine would be expected to be less flow dependent since it is cleared much more slowly than colloid.

However, Kupffer cell destruction or dysfunction may be superimposed on the hepatocyte changes. In fact, injury to the Kupffer cell may be the primary event in acute viral hepatitis (4). This would further reduce the expected amount of radioactive colloid trapped in this region. Other possibilities include blockage of phagocytosis, perhaps due to exhaustion of the receptor sites in the few remaining Kupffer cells, following intensive phagocytosis of debris from degenerating hepatocytes (5). The capacity for phagocytic work also may be diminished as a result of physical encroachment by the surrounding ballooning parenchyma.

Fleischer, et al (6) also noted <sup>75</sup>Se-selenomethionine uptake in an area of poor <sup>99m</sup>Tc-colloid activity in one patient with massive hepatic necrosis. The patient did not survive. Pathological examination of the liver was not reported. The patient in our report showed complete recovery over a period of a few months. The improvement in the colloid liver scan

paralleled the clinical progress and normalization of the blood chemistries. Undoubtedly this reflects resolution of parenchymal swelling and Kupffer cell repopulation in the area of massive inflammatory involvement. The recognition of the potential existence of this scintigraphic appearance in patients with acute hepatitis should be of great benefit to the physician interpreting radionuclide liver images.

#### ACKNOWLEDGMENTS

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