PSEUDOTUMORS IN ACUTE HEPATITIS

The November issue of the *Journal* contains a case report in which the authors note that their case is only the second one reporting discrete nonfunctional pseudotumorous regions in this disease (*1*). It seems relevant to mention a patient we had in 1972.

An afebrile 34-year-old man presented after 1 month of weakness and loss of appetite. He had scleral icterus but no other pertinent physical findings. Laboratory data included an alkaline phosphatase, 37 U (normal less than 25 U) SGOT, 378; SGPT, 93; total bilirubin, 1.8 with a direct of 0.9. The Australian antigen was negative. A barium meal was normal, but the gallbladder was not visualized.

The clinical impression was viral hepatitis, but doubt was raised when we noted a large filling defect in the liver about the portal region in a scan using ^{99m}Tc-sulfur colloid (Fig. 1). The spleen was normal size. Biopsy was performed 4 days later in the region of the filling defect. The specimen (Fig. 2) yielded a basically preserved liver architecture with multiple clusters of necrotic and degenerating cells. No evidence of cholangitis or malignancy was noted and the diagnosis was subacute hepatitis with a random pattern suggesting viral etiology. The patient recovered quickly with all symptoms, physical findings, and laboratory results returning to normal.

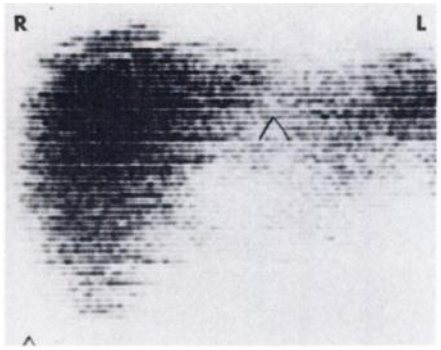

FIG. 1. Anteroposterior liver scan.

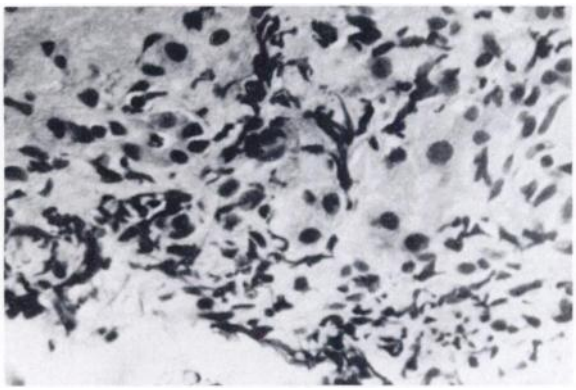

FIG. 2. Liver biopsy.

We had done very few scans on patients with acute hepatitis but apparently such scans as ours and that done by Winston, et al and Shapiro are occasionally seen at large medical centers but seldom reported (*2*). The distinct filling defects in acute hepatitis should be accepted as part of the expected findings on nuclear imaging although this was not apparent in earlier reviews. Realization of this can prevent grasping for another diagnosis in a patient with a filling defect on scan, and who, by all other evidence, appears to have acute hepatitis. This phenomenon should not be surprising considering the pathologic evidence. Regions of severe involvement have been seen in livers that otherwise have spotty involvement (*3*). The lack of phagocytic function is not only probably due to actual reticuloendothelial cellular damage which does occur in hepatitis but also may be due to geometric factors as pointed out by Koenigsberg, et al (*4*). They noted that hepatocyte swelling would greatly diminish the number of reticuloendothelial cells per unit area.

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THE AUTHOR'S REPLY

We thank Dr. Myerson for his additional case report. Interpreting abnormalities in tracer accumulation at the porta hepatis region is always difficult but this could certainly be considered a pseudotumor. A followup study would have been of great interest.

We could not, however, concur that such focal defects "should be accepted as part of the expected findings on nuclear imaging" in acute hepatitis without some qualification. It is still, in our experience, sufficiently unusual that some sort of verification that one is dealing with only acute hepatitis is warranted. Although one might wish to defer a biopsy, at least a followup scan should be planned.

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