jnm/CASE REPORT

PSEUDOTUMORS IN ACUTE HEPATITIS

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A second case in which the finding of focal defects (pseudotumors) on hepatic scintiphotos was apparently caused by acute hepatitis is described. Resolution of these focal defects occurred following institution of corticosteroid therapy.

This is the second report of a patient with acute hepatitis whose liver scan revealed focal nonfunctional areas ("pseudotumors") which disappeared following corticosteroid therapy.

CASE REPORT

A 38-year-old woman presented with right upper-quadrant pain, nausea, and vomiting of 6 days duration. On the day of admission, scleral icterus and dark urine were noted. There was no history of previous liver or gallbladder disease, injections, or heavy ethanol ingestion. The only medications recently used were an anovulatory agent, Norlestrin, and hydrochlorothiazide, 50 mg daily, for premenstrual headaches.

On physical examination the pulse rate was 120 and the temperature was 99.5°F. There was obvious icterus and moderate tenderness on deep palpation in the right upper quadrant. The liver and spleen were not enlarged by palpation or percussion.

Hemoglobin was 14 gm/100 ml, leukocyte count 5500/mm^3, total bilirubin 10.2 mg/100 ml with 6.8 mg/100 ml direct, alkaline phosphatase 70 U (normal, less than 35 U), SGOT 3,620 U (normal, less than 40 U), albumin 3.8 gm/100 ml, globulin 2.7 gm, and prothrombin time 100%.

Although biliary obstruction was suspected clinically, laboratory data were contradictory. The patient was therefore followed at bed rest. After 4 days, pain was less but total bilirubin had increased to 15.6 mg/100 ml, alkaline phosphatase had fallen to 36 U, prothrombin time to 42%, and the SGOT was still markedly elevated at 2,640 U. A liver scan revealed moderate hepatic enlargement with three regions of focally reduced function consistent with space-occupying lesions (Fig. 1A and B). A test

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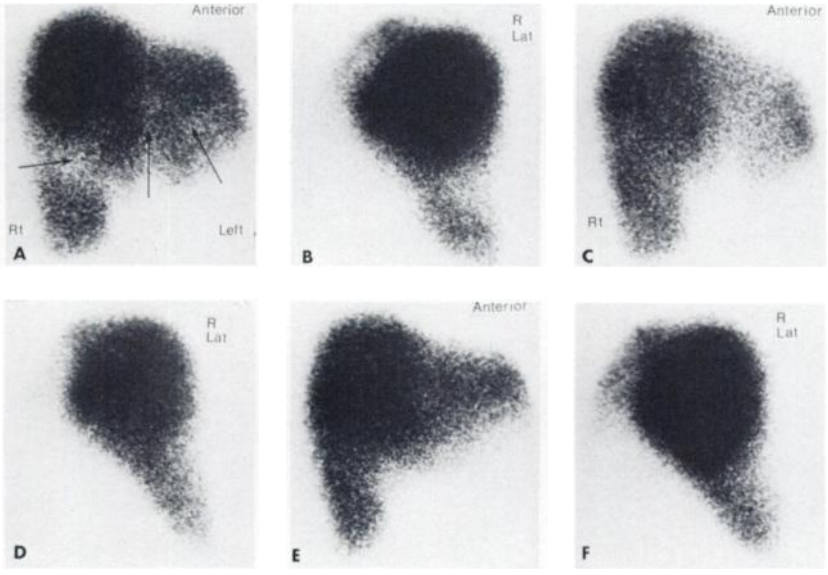

FIG. 1. (A, B) Hepatic scintiphotos at initial admission. Arrows indicate focal defects. (C, D) Hepatic scintiphotos 17 days later shortly after starting steroids. (E, F) Hepatic scintiphotos after another 18 days.

for hepatitis-associated antigen by the countercurrent electrophoresis method was negative.

After administration of vitamin K with a rise in prothrombin time to 50% a liver biopsy was performed that revealed changes of acute hepatitis with a marked inflammatory cell infiltrate (Fig. 2).

One week later, total bilirubin had risen to 26 mg% and prothrombin time had fallen to 25%. Although the patient felt relatively well, it was elected to begin prednisone therapy at 60 mg/day. There was a prompt return of all laboratory and liver scan findings toward normal (Fig. 1C–F). Steroids were tapered over a 4-week period, then discontinued. The patient has remained well and a followup oral cholecystogram and test for hepatitis-associated antigen antibody were negative.

DISCUSSION

Localized cold areas have long been noted on hepatic scans in cirrhosis although normally accompanied by other clues such as splenomegaly, increased marrow trapping of colloidal particles, or occasionally hypertrophy of the left hepatic lobe. Such local changes may be more common in postnecrotic than in Laennec's cirrhosis though certainly occurring in both types (*1–5*).

The patient's initial complaint of severe abdominal pain along with discrete cold areas on the liver scan prompted consideration of hepatic infarction. However, she was in sinus rhythm without history or findings suggestive of cardiac disease as a source of emboli. Furthermore, the scan findings differed from a previously reported case of hepatic infarction in which a wedge-shaped defect was noted with its base at the hepatic border (*6*). Pre-existing liver disease, either chronic hepatitis with cirrhosis, abscesses, or tumor, is unlikely in view of the history, clinical course, biopsy findings, and subsequent resolution as noted on repeat scintiphotos.

Liver scan appearance in acute hepatitis has been considered to be rather nonspecific and has received relatively little attention in the literature. Some degree of hepatomegaly with mild patchiness of tracer distribution is generally described (*4,6–8*). The occurrence of discrete nonfunctional "pseudotumorous" regions has been noted in only one previous report (*9*).

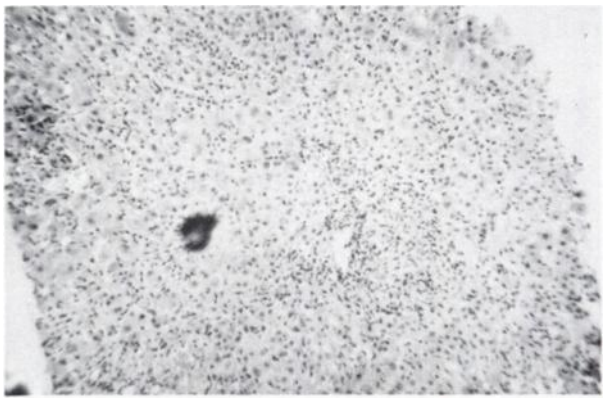

FIG. 2. Liver biopsy shortly after initial admission.

The reason for such focal defects in a presumably diffuse inflammatory process remains obscure. In both instances, the disease was quite severe so that these may represent foci of subacute hepatic necrosis —widespread areas of collapse and necrosis bridging several hepatic lobules—rather than the usual patchy necrosis confined to individual lobules. The rapid resolution whether spontaneous or in response to corticosteroids suggests an inflammatory or edematous basis perhaps related to the ballooning of hepatocytes noted by Koenigsberg, et al (*9*).

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