GALLIUM SCANNING IN ACUTE HEPATIC AMEBIC ABSCESS

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In two cases of acute hepatic amebic abscess ^{67}Ga-citrate was seen to accumulate in the periphery of the abscess cavity. The area of gallium localization appears to be in the region of hyperemia seen histologically and arteriographically. Liver scanning with ^{67}Ga-citrate may be more accurate than radiocolloid liver scanning for assessing the size and resolution of acute hepatic amebic abscess.

Liver scans with colloidal agents are now routinely obtained in the clinical evaluation of patients with suspected hepatic disease including hepatic abscess. Gallium-67-citrate liver scanning performed after a radiocolloid study is a second modality which can increase the specificity and accuracy of diagnosis (*1–3*).

Previous investigators have concluded that hepatic amebic abscesses, unlike pyogenic abscesses, do not accumulate ^{67}Ga-citrate and have included such cases among their correct "negatives" (*1–3*). In contrast to this experience, we have recently encountered two cases which demonstrated identical patterns of ^{67}Ga accumulation. Analysis of the ^{67}Ga distribution in comparison with the ^{99m}Tc-sulfur-colloid scans in the same patients has indicated the probable site of localization and shed additional light on the pathophysiology of acute hepatic amebic abscesses.

CASE REPORTS

Case 1. BW, a 22-year-old white man, was admitted to Walter Reed General Hospital in January 1972 with a 4-month history of intermittent fever and diarrhea. The patient's symptoms originally began in October 1971 while he was stationed in Vietnam. He required hospitalization there for control of severe diarrhea and fever to 104°F. An etiology was not established. However, a 10-day trial course of tetracycline was given and all symptoms subsided allowing return to duty. Approximately 1 month later the patient was readmitted in Vietnam with recurrence of the fever and milder diarrhea. He was transferred to Walter Reed General Hospital for evaluation and treatment.

The pertinent physical findings on admission were a low-grade temperature (99°F) and a mild painless hepatomegaly.

The leukocyte count was 9,800 with a normal differential. Hemoglobin was 11.4 gm% and hematocrit was 34%. The sedimentation rate was elevated; 54 mm in 1 hr. There was elevation of the prothrombin time (25.4 sec) and reversal of the albumin/globulin ratio (4.42/2.58). The remaining liver function tests including the SGOT, SGPT, LDH, alkaline phosphatase, and bilirubin were normal.

The chest film demonstrated an elevated right hemidiaphragm with a right pleural effusion.

A ^{99m}Tc-sulfur colloid liver scan on January 26, 1972 revealed a large focal defect in the anterior superior aspect of the right lobe (Fig. 1A). The scan finding and the clinical situation was thought to be caused by an amebic abscess but necrotic tumor could not be excluded. A ^{67}Ga liver scan was then performed on January 28, 1972, which demonstrated a smaller focal defect bordered by a rim of increased gallium accumulation in the region of the radiocolloid scan defect (Fig. 1B).

Amebic complement fixation titers and hemagglutination tests were markedly positive and the patient was treated with Flagyl. His symptoms and scan defects disappeared with this treatment.

Case 2. A 34-year-old white man was admitted to Walter Reed General Hospital in August 1973

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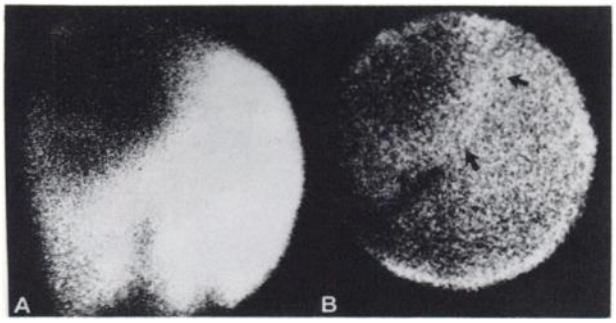

FIG. 1. Hepatic amebic abscess. (A) Anterior view of ^{99m}Tc sulfur colloid scan demonstrating large defect in anterior, superior aspect of right lobe. (B) Anterior view of ^{67}Ga-citrate scan obtained with patient in identical position. Apparent size of abscess defect is smaller and is surrounded by rim of increased activity (arrows). Total area of central defect plus rim of increased gallium uptake in (B) equals area of radiocolloid defect in (A).

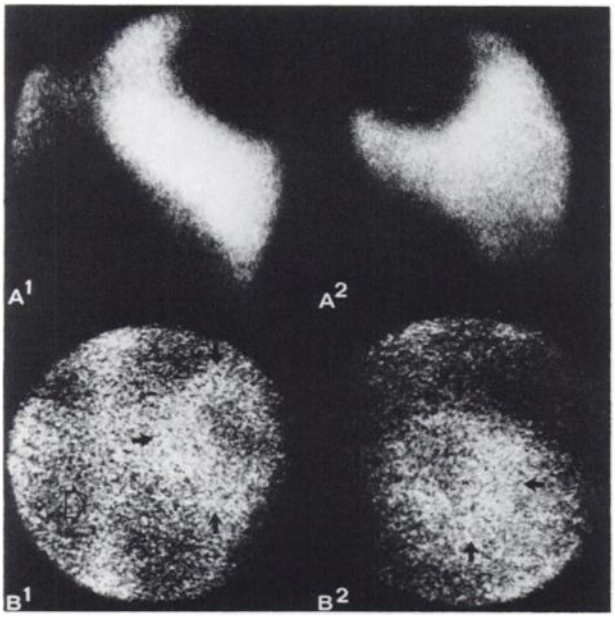

FIG. 2. Hepatic amebic abscess. (A) Posterior (A^1) and right lateral (A^2) views of ^{99m}Tc-sulfur colloid scan reveal large filling defect in posterior, superior aspect of right lobe. No liver activity is seen around superior portion of lesion. (B) Posterior (B^1) and right lateral (B^2) views of ^{67}Ga-citrate scan obtained with patient in same respective positions. Again note that central defect is smaller than in radiocolloid study and is surrounded by rim of increased activity (arrows), which completely encircles lesion in both views. Open arrow indicates ^{67}Ga-citrate uptake by vertebrae.

with an 8-day history of fever, myalgia, weakness, and right pleuritic chest pain.

Pertinent physical findings were mild hepatomegaly without tenderness and decreased breath sounds in the right lower lung.

A chest film revealed elevation of the right hemidiaphragm with a small pleural effusion.

The leukocyte count was 11,500 with a normal differential. The hemoglobin was 12.5 gm% and the hematocrit was 30%. There was mild elevation of alkaline phosphatase 120 I.U. and the Alpha-2 globulin fraction was 1.67 gm%.

A ^{99m}Tc-sulfur colloid liver scan on August 28, 1973 showed a large space-occupying lesion in the posterior superior aspect of the right lobe (Fig. 2 A1,A2). A ^{67}Ga liver scan was done 48 hr later which showed a smaller space-occupying lesion with an increased accumulation of gallium in the periphery. This defect corresponded in location to the defect seen on the radiocolloid scan (Fig. 2 B1,B2).

Amebic complement fixation titers and hemagglutination inhibition tests were markedly positive. The patient was started on therapy with improvement of his symptoms.

DISCUSSION

Radiocolloid scans reflect the distribution of reticuloendothelial activity in the liver. Cauron, et al (*4,5*) and Aquirre-Garcia (*6*) have shown that normal phagocytic activity of Kupffer cells is interfered with in the area immediately surrounding acute amebic abscesses. This is due to an intense inflammatory response in the boundary area which also corresponds to a zone of increased vascularity demonstrable angiographically (*7*).

In a series of patients studied with both a radiocolloid (^{113m}In) and a blood-pool agent (^{113m}In-transferrin), Cauron found a marked discrepancy in the size of the individual lesions as seen with the two radiopharmaceuticals. Defects in the blood-pool scans represented only the central necrotic abscess cavities themselves. Defects in the radiocolloid studies represented both the necrotic center as well as the surrounding inflammatory zone of inhibited reticuloendothelial cells. On the average the abscess itself accounted for only 24% of the total area of the defect seen on the radiocolloid scan (*4,5*).

As noted in Figs. 1 and 2, both of our cases demonstrate a discrepancy in the size of the defects seen in the ^{99m}Tc-sulfur colloid scan and the ^{67}Ga-citrate scans. Moreover, the rim of increased activity on the ^{67}Ga liver scan is essentially contained within the area of the ^{99m}Tc-sulfur colloid scan defects. This combination of findings is explained by an increased accumulation of ^{67}Ga-citrate in the inflamed hyperemic zone about the actual abscess and a decreased accumulation of ^{99m}Tc-sulfur colloid in both the abscess and in the zone of hyperemia. These assumptions are supported by the research of Blair, et al (*8*) and Harvey, et al (*9*).

Histologically, the transition from acute to chronic amebic abscess takes place slowly over a period of 2 to 3 months. During this time the central necrotic area may cavitate and a delicate wall of granulomatous tissue forms but the most marked changes occur

in the inflammatory rim which regresses (*6,7*). The histological changes present in the transformation of an acute amebic abscess to a chronic amebic abscess are demonstrated arteriographically by resolution of the intense blush in the boundary zone of the abscess (*7*). This fact may account for the discrepancy in the literature regarding the healing rate of amebic abscesses demonstrated by serial radiocolloid scans (*10,11*). The apparent rapid resolution seen on radiocolloid studies is largely due to return of phagocytic function of the reticuloendothelial cells secondary to the regression of the inflamed, hyperemic region about the rim of the abscess. The prolonged liver scan defects reported by some authors (*11,12*) may represent scar tissue replacing liver parenchyma in the central area of necrosis.

From the two cases presented, ^{67}Ga liver scanning is a more accurate way to assess the size of acute hepatic amebic abscesses than radiocolloid scanning and may prove to be superior for following their resolution.

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