Large Focal Defect on Liver/Spleen Scan Caused by Fatty Liver and Masquerading as Neoplasm

Nanda Khedkar, Benjamin Pestika, Howard Rosenblate, and Charles Martinez

Departments of Nuclear Medicine, Internal Medicine and Hepatology, Lutheran General Hospital, Park Ridge, Illinois

Focal fatty infiltration of the liver may be mistaken for metastatic disease, primary tumor or other space-occupying lesions on CT or ultrasound. Usually, a ^{99m}Tc-sulfur colloid scan is sensitive in documenting the presence of Kupffers cell in such a process. We present a case that was suggestive of focal fatty infiltrate on a CT scan, nondiagnostic on ultrasound, and seen as a large focal defect on the ^{99m}Tc-sulfur colloid liver/spleen scan. A ^{133}Xe inhalation study, however, did show uptake in the area of fatty infiltration. A needle biopsy confirmed the diagnosis.

J Nucl Med 1992; 33:258–259

Hepatic fatty infiltrate is a frequently identified pathologic process. Alcoholism, drugs such as steroids, obesity and malnutrition are some etiologic factors. Many times, these lesions are rapidly progressive and may be misdiagnosed on the computed tomography (CT) scan as metastatic lesions. They may decrease in size or disappear after the offending agent is removed.

A diagnostic dilemma occurs because the fatty infiltration appears on the CT examination as an irregular area of decreased density. An ultrasound examination will typically show discrete areas of hyperechogenicity. Frequently on ultrasound, however, findings are not typical and at times abnormalities can be misdiagnosed as metastic or primary neoplasms (*1*).

Technetium-99m-sulfur colloid, on the other hand, usually shows uniform hepatic uptake reflecting normal distribution of reticuloendothelial cells in the liver (*1,2*). Xenon-133, a fat-soluble inert gas (*5*), will show increased (or positive) activity in liver lesions secondary to focal fatty infiltration (*2,3,5*).

The following case is of interest because it shows a focal defect on a ^{99m}Tc-sulfur colloid scan with positive focal ^{133}Xe uptake. This finding is contrasted with another case report demonstrating focal fatty infiltration of the liver which failed to sequester either ^{99m}Tc-sulfur colloid or ^{133}Xe (*4*).

Received May 14, 1991; revision accepted Sept. 4, 1991.

For reprints contact: Nanda Khedkar, MD, Associate Director, Nuclear Medicine Depart., Lutheran General Hospital, Park Ridge, IL 60068.

CASE REPORT

A 60-yr-old white female with a history of noninsulin-dependent diabetes mellitus, hypercholesterolemia, hypertension and coronary artery disease presented to her physician for evaluation of nausea. The patient had been previously placed on Niacin which was stopped with subsequent resolution of nausea.

Her physical examination showed normal vital signs. Mild thyroid enlargement was noted. Examination of the heart, lungs and abdomen were unremarkable. The work-up revealed elevated SGOT, SGPT, and alkaline phosphatase which did not completely normalize after Niacin was stopped. Because of the persistent abnormalities in the results of liver function tests, an abdominal CT was obtained that showed a large defect in the left hepatic lobe (Fig. 1A). The CT was suggestive of geographic fatty liver, but ultrasound findings were inconclusive.

A ^{99m}Tc-sulfur colloid scan showed a large focal defect (Fig. 1B) corresponding to the lesion seen on the CT scan. A ^{67}Ga-citrate scan (Fig. 2B) was also obtained, which showed only moderate activity in the lesion when compared to normal adjacent liver tissue as seen on the hepatobiliary scan (^{99m}Tc-DISIDA).

Because the CT scan suggested focal fatty infiltration and both the ^{67}Ga and hepatobiliary scans showed intrahepatic activity within the area in question, a ^{133}Xe-hepatogram image was obtained. This study showed prominent activity within the left hepatic lobe lesion (Fig. 2A). A fine-needle biopsy of the liver lesion under CT guidance revealed marked fatty changes and mild portal fibrosis. The number of RE cells seen appeared adequate (Fig. 3).

DISCUSSION

There is increasing recognition of focal fatty infiltration in the liver as an ancillary finding (*7*). The CT scan

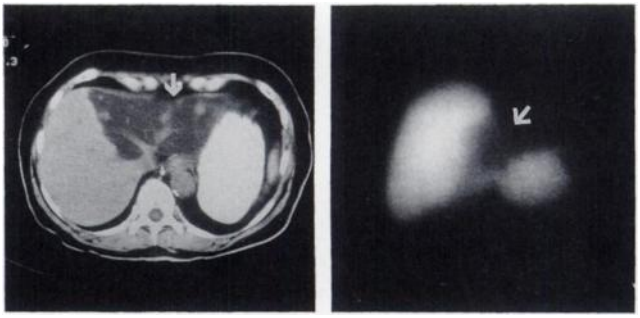

FIGURE 1. (A) CT scan of the liver shows the corresponding large low-density lesion (arrow). (B) Anterior view from ^{99m}Tc-sulfur colloid reveals a large defect in the left and adjacent right hepatic lobes (arrow).

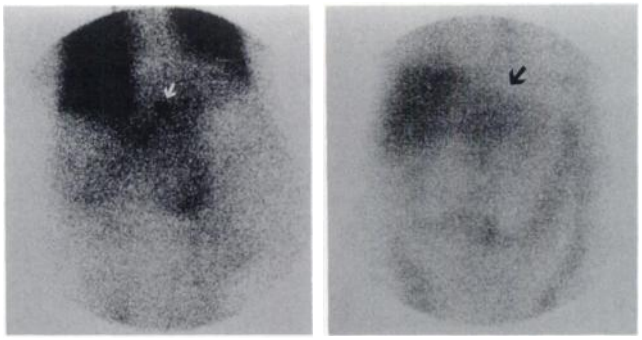

FIGURE 2. (A) Xenon hepatogram image shows uptake in the left lobe lesion. Dark focus near left hepatic lobe represents radioactive marker at the xiphisternum (arrow). (B) Anterior view of abdomen from the 72-hr ^{67}Ga citrate scan showing uptake in the liver lesion (arrow).

typically shows a region of radiolucency with a corresponding hyperechoic region on ultrasound. Before the advent of the CT scan and ultrasound, focal fatty infiltrates may not have come to the attention of imaging physicians, since in most cases, the ^{99m}Tc-sulfur colloid liver scan showed uniform and normal uptake (*4*).

False-negative studies with ^{99m}Tc-sulfur colloid are rare. There is only one other report describing a focal defect on liver/spleen scan and that case was associated with a negative defect on the ^{133}Xe liver image (*4*).

Unlike ultrasound and CT, ^{99m}Tc-sulfur colloid imaging is a functional examination and the presence of intact reticuloendotheial cells will be associated with uniform uptake in the hepatic lesion of focal fatty infiltration on a technetium colloid scan. When the degree of fatty infiltrate is minimal, the radionuclide scan may be completely normal. However, as the amount of fatty infiltration increases, hepatomegaly with varying degrees of altered radiocolloid uptake may occur (*6*). This may explain the finding of irregular uptake described in the literature (*8*).

In our case, however, in spite of normal reticuloendothelial cell distribution on biopsy, a focal defect was noted. Gallium-67 and ^{99m}Tc-DISIDA both showed uptake in the lesion. These radiopharmaceuticals are normally taken up in the hepatocytes. It is possible that there may be anatomic or physiologic shunting of blood flow away from the hepatic area of fatty infiltrations. This function, when combined with the short plasma half-life of ^{99m}Tc-sulfur colloid, may explain the difference between radiocolloid uptake in normal tissue and areas involved with focal fatty infiltration.

The longer plasma half-life of ^{67}Ga and ^{99m}Tc-DISIDA may result in the positive uptake of these tracers in areas of fatty infiltration. Since ^{133}Xe is fat-soluble, its uptake by fatty tissue will depend not only on blood flow but on the concentration of this gas in the mixture, duration of exposure of ^{133}Xe to the lesion and, finally, fat content in the lesion (*5*). These factors all combine to explain ^{133}Xe uptake in the liver lesion in our case.

A false-positive study with ^{99m}Tc-sulfur colloid in a focal fatty infiltrate has not been described in the literature (*6*). Therefore, normal uptake of ^{99m}Tc-sulfur colloid in the lesion rules out a malignant process and helps establish the diagnosis of focal fatty infiltrate. However, as seen in this patient, a focal defect in the liver scan, although rare, does not rule out the presence of focal fatty infiltrate.

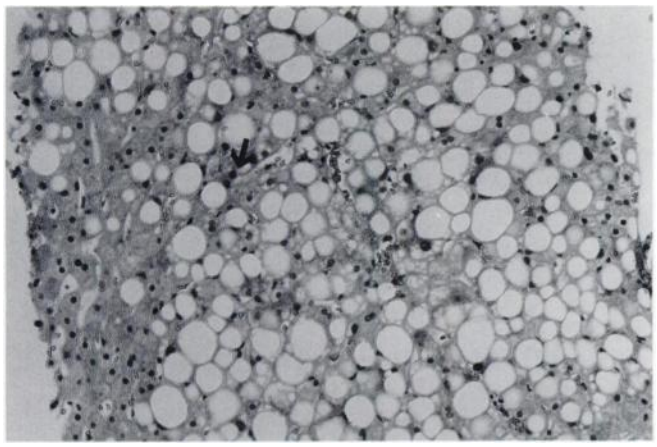

FIGURE 3. Liver parenchyma with marked fatty change and hyperplastic Kupffer cells (arrow).

ACKNOWLEDGMENTS

The authors would like to thank Yolanda P. Trujillo, MD for reviewing the biopsy slides and Diana Wittman for her assistance.

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