FOCAL PORTA HEPATIS SCINTISCAN DEFECTS: WHAT IS THEIR SIGNIFICANCE?

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A total of 537 consecutive liver scintiscans were retrospectively reviewed and 80 of them revealed suspicious focal decreased activity in the region of the porta hepatis. Postmortem, surgical, or biopsy correlation was obtained in 40 of these cases: 14 were pathologically negative; 9, cirrhosis or fibrosis; 10, metastases; 3, dilated bile ducts; 1, viral hepatitis; 1, hepatic laceration; 1, falciform ligament cyst; and 1, ruptured gallbladder with abscessed head of the pancreas. Thus, only 42% represented significant disease. Sixty-eight percent of the defects were seen only on the anterior scintiscan. Appearance of the majority of defects was nonspecific. Subjective grading of defects according to size and comparative decrease in density was not beneficial. Elevations of serum alkaline phosphatase, total serum bilirubin, and serum glutamic-oxalacetic transaminase were nonspecific.

Of the several types of nuclear medicine scintiscanning procedures performed in a modern nuclear medicine department the liver scintiscan is certainly among the most difficult to interpret. This difficulty relates to limitations of equipment resolution; difficulty in portraying an area of decreased activity in a large organ with prominent activity; numerous developmental variations in configuration; superimposed or inherent anatomic structures, such as the porta hepatis, gallbladder, hepatic veins, costal margin, vertebrae; and nonspecificity of scintiscan defects (*1,2*). Numerous authors have recommended caution in interpretating marginal irregularities and single focal defects, especially if these are located in the region of normal anatomic structures or are seen on only one scintiscan projection (*1–3*). This author has not infrequently experienced the frustration of having called a scintiscan defect in the porta hepatis anatomic or insignificant, only to find at surgery a significant lesion, and vice versa. For these reasons, a retrospective review of this hospital's experience regarding single, focal porta hepatic defects, and their correlation with clinically significant findings, was undertaken.

MATERIALS AND METHODS

All liver scintiscans performed at St. Paul-Ramsey Hospital during the period March 1, 1971, to March 1, 1974, were evaluated for the presence of single focal decreased activity in the region of the porta hepatis. Scintiscans were evaluated retrospectively by an experienced nuclear medicine physician without the knowledge of clinical history or previous interpretation. Marginal defects in the porta hepatis region were not considered. The clinical charts of all patients with porta hepatis defects were then reviewed, and surgical and pathologic correlation was made when possible. Correlation was also made with serum alkaline phosphatase, total serum bilirubin, and serum glutamic-oxalacetic transaminase (SGOT). The data were analyzed and grading according to subjective interpretation of defect prominence (size and comparative decrease in intensity) was attempted. Single focal hepatic defects were considered significant when they represented neoplasm or surgical lesion. Single porta hepatis defects caused by normal anatomic structures or cirrhotic or fibrotic changes were not considered significant. All liver scintiscans were performed with ^{99m}Tc-sulfur colloid using either the Picker 5-in. rectilinear scanner with a 3-in. focal length, ½-in. resolution collimator, and 14 × 17-in. film, or with the Searle Radiographics Pho/Gamma HP scintillation camera with a high-sensitivity, parallel-hole collimator and 70-mm film. All scintiscans included at least an anterior and right lateral projection, and more recent

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TABLE 1. PATHOLOGIC CORRELATION OF 40 PORTA HEPATIS SCINTISCAN DEFECTS

Subjective grading	Pathologically negative	Cirrhosis or fibrosis	Metastases	Obstructed dilated common bile ducts	Viral hepatitis	Laceration	Falciform cyst	Ruptured gallbladder	Total	Percent significant pathology
Grade I	8	3	2	2					15	27
Grade II	2	2	7	1	1			1	14	71
Grade III	3	3				1	1		8	25
Grade IV	1	1	1						3	33
Total	14	9	10	3	1	1	1	1	40	42

scintiscans performed with the camera included posterior projections.

RESULTS

Of the 537 consecutive hepatic scintiscans reviewed, 80 were considered to have significant or questionably significant decreased activity in the region of the porta hepatis. Of these 80, 40 had postmortem, surgical, or biopsy data. Only the 40 scintiscans that were correlated will be discussed in this paper.

Table 1 lists the pertinent correlative findings of the 40 scintiscans discussed in this paper.

Two of the three cases with dilated common bile ducts listed in Table 1 were caused by stones obstructing the common bile duct at the ampulla (Fig. 1), and one was caused by metastatic carcinoma of the head of the pancreas, which invaded the region of the ampulla and obstructed the common bile duct.

Metastases in this study included blood-borne or lymphatic spread of neoplasm to the porta hepatis, as well as direct extension of tumor from adjacent structures. Figure 2 shows the scintiscan of a 67-year-old male patient with carcinoma of the head of the pancreas, with extension of the tumor along lymphatics and invasion of the porta hepatis region.

The one case of viral hepatitis (Fig. 3) occurred in a 20-year-old jaundiced male. Pathologic correlation was not obtained but the patient did have a positive Australian antigen test. This patient recovered clinically, but a repeat liver scintiscan 3 months later revealed the same porta hepatis defect.

Of particular interest was the case of an asymptomatic 49-year-old female admitted with a nontender, crepitant, right-upper-quadrant mass (Fig. 4). At surgery a large developmental cyst of the falciform ligament was found and excised without complication.

The most striking scintiscan in this series occurred in a 70-year-old female with a palpable epigastric mass and slightly elevated liver function studies (Fig. 5). Exploratory laparotomy and liver biopsy re-

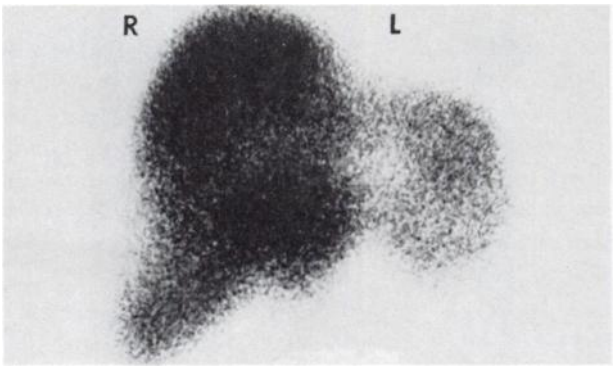

FIG. 1. Stones obstructing common bile duct causing dilatation of ducts. Focal decreased activity in region of porta hepatis noted only on this anterior scintiscan projection. Note ill-defined bands of decreased activity extending from porta hepatis defect into midportion of right lobe and inferior portion of left lobe of liver.

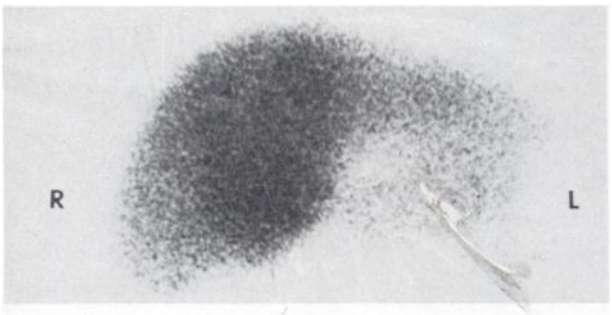

FIG. 2. Carcinoma of head of pancreas invading porta hepatis. Focal decreased activity in region of porta hepatis noted only on this anterior scintiscan projection.

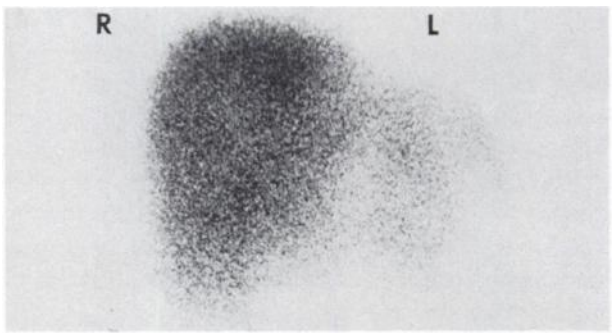

FIG. 3. Viral hepatitis. Focal decreased activity in the region of porta hepatis noted only on this anterior scintiscan.

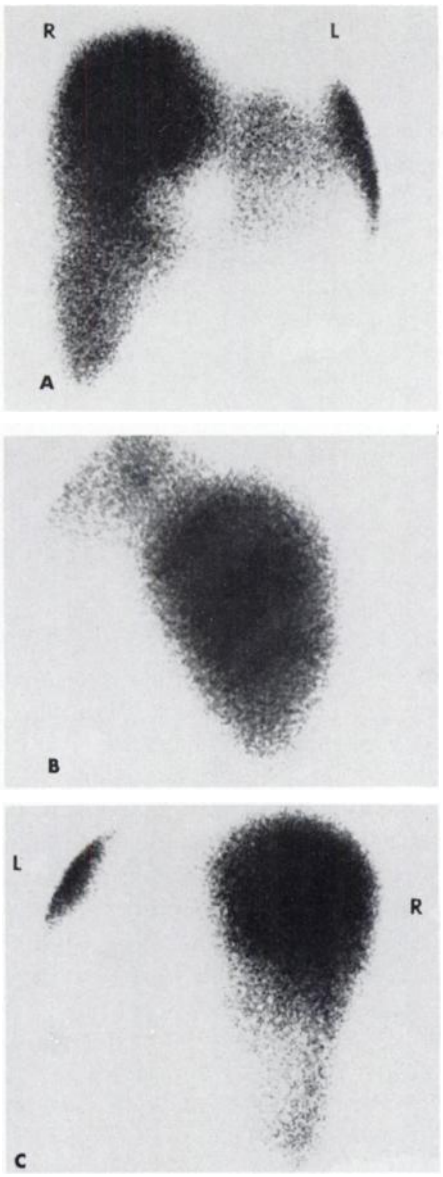

FIG. 4. Anterior (A), right lateral (B), and posterior (C) scintiscans showing falciform ligament cyst. Focal decreased activity in region of porta hepatis is noted only on anterior scintiscan despite prominence of defect.

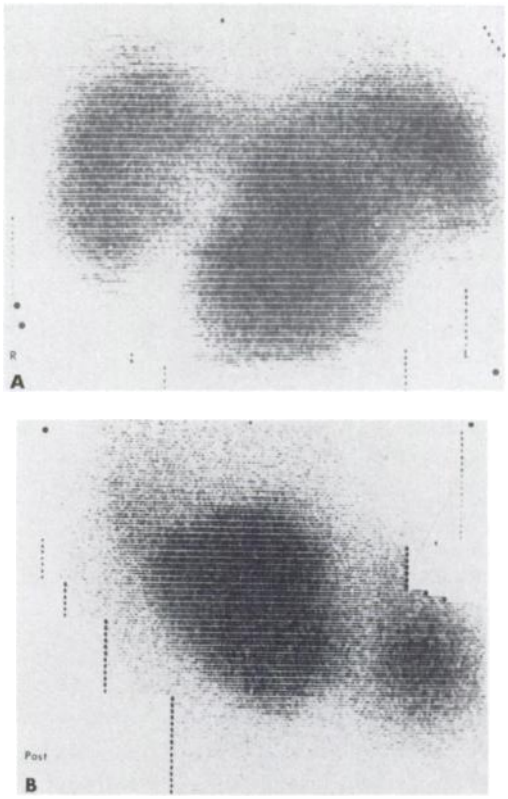

FIG. 5. Cirrhosis and focal scarring. (A) Anterior scan shows large area of decreased activity in region of porta hepatis and hypertrophy of left lobe of liver. (B) Right lateral scan shows vertical band of decreased activity.

TABLE 2. PATHOLOGIC CORRELATION OF 13 PORTA HEPATIS SCINTISCAN DEFECTS SEEN ON TWO PROJECTIONS

Defect	Number seen on two projections
Pathologically negative	3
Cirrhosis and/or fibrosis	3
Laceration	1
Metastases	6
Total	13

vealed hypertrophied left lobe of the liver and extensive scarring in the region of the porta hepatis. Biopsy in this region revealed active portal cirrhosis with fatty metamorphosis and alcoholic hyaline.

Of the 40 cases of porta hepatis scintiscan defects in this series only 13 defects were seen on two projections of the scintiscan and these were all on the anterior and right lateral scintiscans. No porta hepatis defects were detected on the posterior scintiscans. Pathologic correlation of the 13 cases seen on two projections is shown in Table 2.

Table 3 shows correlation of pathologic findings with serum alkaline phosphatase, total serum bilirubin, and SGOT.

DISCUSSION

Anatomically, the porta hepatis is located at the inferior surface of the liver between the quadrate lobe anteriorly and the caudate lobe posteriorly. From the posterior aspect, the porta hepatis lies between the right and left lobes of the liver and, from the anterior aspect, it lies under the medial segment of the left lobe of the liver. In the anterior scintiscan, the porta hepatis should lie to the left of a line drawn vertically through the middle of the liver. In the anterior projection, the porta hepatis can cause a marginal-type defect, but more frequently it lies several centimeters up from the inferior margin of the liver and appears as a vague area of decreased

TABLE 3. CORRELATION OF PORTA HEPATIS FINDINGS WITH SERUM ALKALINE PHOSPHATASE, TOTAL SERUM BILIRUBIN, AND SGOT

Type	Number of cases	Elevated serum alkaline phos-phatase	Elevated total serum bilirubin	Elevated SGOT
Pathologically negative	14	2	0	5
Cirrhosis or fibrosis	9	2	3	4
Metastases	10	6	3	5
Obstructed bile ducts	3	3	3	3
Viral hepatitis	1	1	1	1
Hepatic laceration	1	0	0	0
Falciform ligament cyst	1	0	0	0
Ruptured gallbladder	1	1	1	1
Total	40	15	11	19

activity. In the right lateral scintiscan the porta hepatis is rarely seen, but should lie somewhere near the central portion of the lateral projection depending on the position of the patient and the angulation necessary to separate the liver from the spleen (*4*). On the posterior projection of the scintiscan, the porta hepatis lies partially behind and partially just to the right of the vertebral column and is not usually identified (*5,6*).

The large number of pathologically negative cases in this series confirms the difficulty of distinguishing, by scanning, between normal and diseased structures in the region of the porta hepatis. Nonparenchymal structures cause decreased activity in the region of the porta hepatis because they do not concentrate colloid particles as does normal hepatic tissue; they, therefore, present as an area of relative decreased activity.

The difficulty in distinguishing between cirrhotic or focal fibrotic scarring and metastatic defects on liver scintiscans is certainly not peculiar to the porta hepatis region. Cirrhotic changes and fibrosis are among the most frequent causes of misdiagnosis of liver scintiscan abnormality (*7*). In this series, when porta hepatis scintiscan defects from cirrhosis or fibrosis (9/40) were added to the undiseased cases (14/40), a total of 57.5% (23/40) of the porta hepatis scintiscan defects were insignificant clinically.

Metastases to the porta hepatis frequently occur and are enhanced by the generous blood supply (hepatic artery, portal vein) and lymphatic drainage to the porta hepatis area, as well as the proximity of the porta hepatis to the pancreas, ampulla, and gallbladder from which neoplasms can extend directly.

The three cases of dilated bile ducts in this series substantiate the comments made by DeLand and Wagner, who stated, "Since the bile ducts may simulate metastatic lesions, even a clear-cut focal area of decreased activity in the region of the porta hepatis should be interpreted with caution in patients with obstructive jaundice" (*6*). Two papers (*8,9*) have described a typical liver scan pattern for obstructed biliary ducts, consisting of band-like areas of decreased activity radiating from an area of decreased activity in the region of the porta hepatis similar to Fig. 1. Both references recognized that false-positive and false-negative results can occur. Of the three cases of obstructed, dilated biliary ducts in this series, all three showed decreased activity in the region of the porta hepatis but only one (Fig. 1) showed the radiating bands of decreased activity.

In addition to causes of portal defects seen in this study, the differential diagnosis must also include other causes of focal liver scintiscan defects, such as hepatoma (*10,11*), abscess, infarction (*12*), choledochal cyst (*13*), and dilated splenic vein (*14*).

Several cases of intrahepatic focal scintiscan defects associated with acute viral hepatitis have been reported in the recent literature (*15,16*). Most of them shrank in size with clinical improvement. The single case of viral hepatitis in this series with decreased porta hepatis activity was not verified by liver biopsy but the patient did have a positive Australian antigen test. Since the scintiscan defect was unchanged after 3 months, it is possible the defect was not related to the hepatitis.

The importance of visualizing abnormality in two projections on roentgenograms and scintiscans has become axiomatic in radiology and nuclear medicine. In this series 27 of 40 (68%) of the correlated porta hepatis scintiscan defects were seen only on the anterior projection (Fig. 4) and 10 of these (37%) represented significant disease. Thus, one cannot depend on seeing significant porta hepatis defects in projections other than the anterior. Of the 13 cases seen in 2 projections, 7 (54%) were significant. Although the incidence of significant findings was higher when porta hepatis scintiscan defects were seen in two projections, the incidence was certainly significant when the defects were seen only on one projection.

Porta hepatis scintiscan defects are poorly seen on projections other than the anterior projection primarily due to location of the porta hepatis. In the lateral projection, the porta hepatis lies a considerable distance from the right body margin, approxi-

mately 14 cm in an adult, depending on his size. Collimator resolution at this distance is very poor. The porta hepatis region is poorly seen on the posterior scintiscan because it lies a considerable distance from the back of the patient, approximately 13 cm in the average adult, and it lies partially behind the vertebrae (*17*) which attenuate some of the radiation.

Subjective grading of scintiscan defects was not beneficial since even the least prominent defects, graded I in this series, had a 27% incidence of significance. Interestingly, Grade II defects had a higher percentage of significance than Grade III or Grade IV defects, which were larger and more prominent. This further substantiates the difficulty of attempting to determine pathologic significance on the basis of the size and intensity of a scintiscan defect.

The majority of porta hepatis scintiscan defects in this series did not have characteristic appearances that would be pathognomonic. One case of biliary duct obstruction (Fig. 1) did show characteristic radiating bands of decreased activity but the other two cases did not. The appearance of the hepatic lobes in Fig. 5 is consistent with cirrhosis, but the focal defect in the region of the porta hepatis is nonspecific. This lack of specificity might be partially overcome by utilizing complementary investigative techniques (*18–23*).

Serum alkaline phosphatase, total serum bilirubin, and SGOT did not correlate well with porta hepatis disease. Patients with liver metastases did have a higher incidence of elevated serum alkaline phosphatase than did patients in the pathologically negative and cirrhosis or fibrosis groups, but four of ten patients with metastases had normal serum alkaline phosphatase determinations and several patients in other categories had elevated alkaline phosphatase. Total serum bilirubin and SGOT determinations were equally nonspecific except that none of the pathologically negative cases had elevated total serum bilirubin.

CONCLUSION

Focal scintiscan defects in the region of the porta hepatis must be interpreted with caution because: (A) Only 42% of scintiscan defects represented significant abnormality—35% of defects were anatomic and 23% represented cirrhosis and/or fibrosis. (B) Defects seen in only one projection often represented significant pathology. (C) Defect appearance was usually nonspecific. (D) Size, prominence, and relative amount of decreased intensity as compared to the rest of the liver did not correlate well with significant pathology. Elevations of serum alkaline phosphatase, total serum bilirubin, and SGOT were nonspecific.

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