
SPECT Imaging in the Diagnosis of Budd-Chiari Syndrome

Chun K. Kim, Christopher J. Palestro, and Stanley J. Goldsmith

*The Andre Meyer Department of Physics-Nuclear Medicine, The Mount Sinai Medical Center
Mount Sinai School of Medicine, New York, New York*

We recently performed planar and single photon emission computed tomographic (SPECT) liver/spleen scintigraphy in a patient with Budd-Chiari syndrome. While planar imaging revealed only nonspecific hepatocellular dysfunction, tomographic images demonstrated increased radiotracer accumulation in the caudate lobe of the liver, the classical scintigraphic finding in this entity. This case suggests that SPECT may enhance the sensitivity of the scintigraphic diagnosis of this uncommon condition, especially when the occlusion of the hepatic veins is incomplete.

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Focally increased hepatic activity on technetium-99m sulfur colloid (^{99m}Tc]SC) images has been described in a variety of conditions (1,2). Most often the area of increased tracer accumulation is in the quadrate lobe of the liver, and is secondary to superior vena caval obstruction. Occasionally this increased activity is within the caudate lobe of the liver, and when present, is highly suggestive of the Budd-Chiari syndrome (BCS) (3-5).

We report a case of BCS in which the planar ^{99m}Tc]SC images were nondiagnostic, while SPECT clearly identified increased tracer accumulation in the caudate lobe of the liver, the classic scintigraphic finding in this entity.

CASE REPORT

A 42-yr-old female was admitted to our institution with a 2-day history of chills, diaphoresis, generalized abdominal pain, and abdominal distension. Past history was remarkable for primary thrombocytosis diagnosed by bone marrow biopsy 1 yr prior to admission. The patient had had multiple bleeding and clotting events as a result of the disease, and had been treated with hydroxyurea. Physical examination revealed a moderately distended abdomen; the liver was moderately enlarged and tender to palpation. Laboratory values were

remarkable for a platelet count of 545,000/ μl (normal 130,000-400,000).

In light of the patient's history and physical findings, the two principal diagnoses entertained were BCS and intrahepatic bleeding. Abdominal ultrasonography revealed ascites and moderate hepatomegaly; the hepatic veins were not clearly seen.

Technetium-99m SC scintigraphy was performed on the 3rd day after admission. Planar imaging revealed nonspecific hepatocellular dysfunction (Fig. 1). The SPECT study performed immediately after planar imaging unequivocally demonstrated increased tracer uptake in the caudate lobe of the liver, with relatively decreased uptake in the remainder of the organ: the classical scintigraphic presentation of BCS (Fig. 2). Magnetic resonance imaging (MRI), performed after completion of the radionuclide study revealed flattening of the inferior vena cava (IVC) as well as intraluminal thrombus (Fig. 3).

The patient was placed on intravenous heparin, and a peritoneovenous shunt was placed to reduce ascites. She suffered multiple bleeding and clotting complications including cerebral hemorrhage, sepsis, disseminated intravascular coagulopathy, thrombosed peritoneovenous shunt and pulmonary embolism during her hospital stay and died due to cardiopulmonary arrest on the 36th day after admission.

DISCUSSION

Budd-Chiari syndrome is a rare condition that may appear with catastrophic onset or develop insidiously over many months (6). It is produced by hepatic venous outflow obstruction at the level of the inferior vena cava, hepatic veins, or hepatic venules. This syndrome is associated with thrombus, certain medications, hypercoagulable states, and congenital webs or membranes of the IVC (7,8). Diagnosis of this condition is difficult, and mortality is high; prompt diagnosis is essential if proper therapy is to be instituted. While history and physical examination may suggest BCS, the diagnosis is frequently confirmed only by invasive procedures such as liver biopsy and hepatic venography.

The ^{99m}Tc]SC pattern of increased tracer activity in the caudate lobe of the liver is considered characteristic of this entity, and is related to the anatomy at the hepatic venous drainage.

The liver is drained by three main veins: the right and the left hepatic veins, which drain the right and the

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For reprints contact: Chun K. Kim, MD, Box 1141, The Andre Meyer Department of Physics-Nuclear Medicine, The Mount Sinai Medical Center, Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, NY 10029.

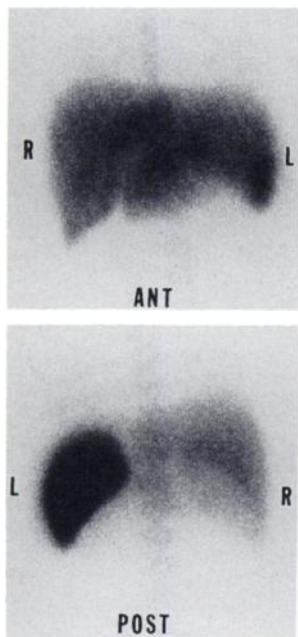


FIGURE 1
Anterior and posterior planar [^{99m}Tc]SC images demonstrate hepatomegaly with heterogeneous uptake, and colloid shift to the spleen and bone marrow. Although there may be minimally increased activity in the region of the caudate and left lobe, the overall findings are relatively nonspecific and inconclusive for the diagnosis of BCS.

left lobes respectively, and the middle hepatic vein, which drains the central portions of the liver including the caudate lobe. The caudate lobe is also drained by several small veins that empty directly into the IVC. In BCS, even when the major hepatic veins are occluded, the small veins of the caudate lobe often remain patent, and the caudate lobe becomes the preferential route of passage of blood through the liver, explaining the phenomenon of increased tracer uptake in this lobe on [^{99m}Tc]SC imaging.

Increased caudate lobe activity is not always present on planar imaging, and in a significant number of cases of BCS the [^{99m}Tc]SC pattern is nonspecific (3,4,9,10). Picard et al. reviewed 131 cases of BCS reported in the literature and observed that the classical finding (increased caudate lobe activity) was seen in 83 cases (63%), diffuse hepatomegaly with either homogeneous or, more commonly, heterogeneous uptake in 26 cases (20%), and various other findings in 14 cases (17%) (4). The variation in scintigraphic patterns is dependent on the particular venous drainage pathways that remain patent (4), degree of occlusion (5), and its chronicity (9). The planar image findings in this case, indeed, belong to the nonspecific category, whereas SPECT performed at the same time demonstrates the classic findings. Therefore, it is conceivable that in some instances, (i.e., incomplete hepatic venous occlusion) while increased caudate lobe uptake may in fact be present, it may not be evident on planar images because the contrast between the caudate lobe and the remainder of the liver may be low and the deep location of this lobe (just anterior to the IVC) hinders its evaluation. By eliminating the contribution of the surrounding hepatic parenchyma to the image, SPECT affords improved visualization of the caudate lobe.

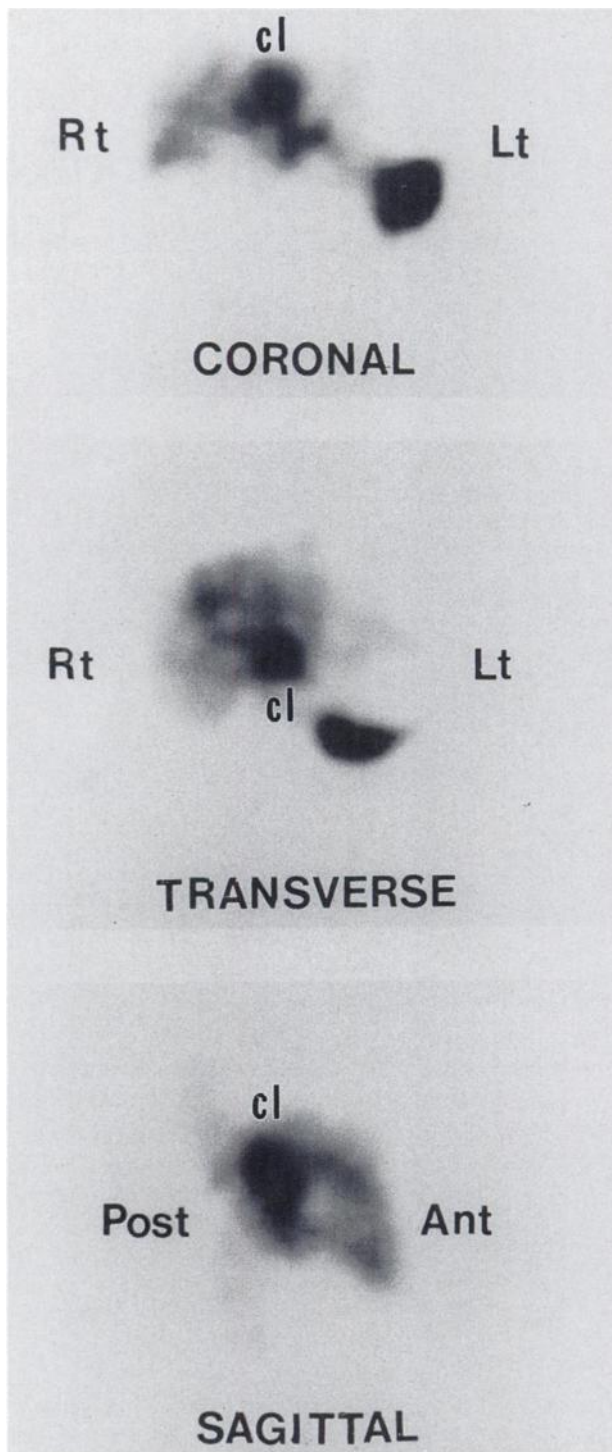


FIGURE 2
SPECT more clearly demonstrates increased uptake in the caudate lobe (cl), with relatively decreased uptake in the remainder of the liver.

We suggest that in patients with suspected BCS, if planar imaging is inconclusive, SPECT may provide additional confirmatory data, thereby improving the utility of the radionuclide study. The combination of the anatomic abnormalities on MRI and physiologic abnormalities (indicating altered blood flow) on [^{99m}Tc]

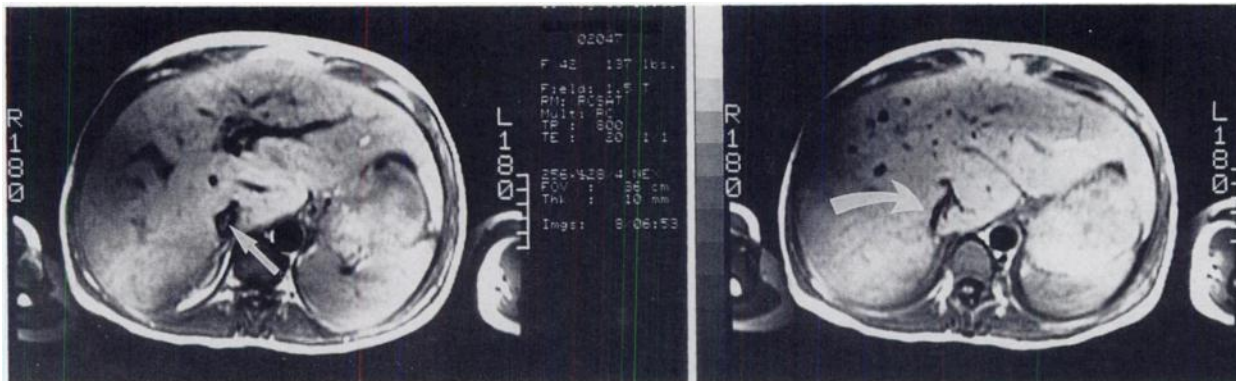


FIGURE 3

T1-weighted MRI at the level of the ninth and tenth thoracic vertebrae reveals flattening of the IVC (curved arrow) as well as intraluminal thrombus (straight arrow).

SC SPECT imaging, in the appropriate clinical setting, could conceivably spare the patient more invasive procedures such as liver biopsy and hepatic venography.

REFERENCES

1. Datz FL. *Gamuts in nuclear medicine*. Norwalk: Appleton-Century-Crofts; 1983;154-155.
2. Silberstein EB, Ruppert D. Liver-colloid imaging. In: Silberstein EB, McAfee JG, eds. *Differential diagnosis in nuclear medicine*. New York: McGraw-Hill, Inc., 1984;161-179.
3. Tavill AS, Wood EJ, Kreel L, Jones EA, Gregory M, Sherlock S. The Budd-Chiari syndrome: correlation between hepatic scintigraphy and the clinical, radiological, and pathological findings in 19 cases of hepatic venous outflow obstruction. *Gastroenterology* 1975;68:509-518.
4. Picard M, Carrier L, Chartrand R, Franchebois P, Picard D, Guimond J. Budd-Chiari syndrome: typical and atypical scintigraphic aspects. *J Nucl Med* 1987;28:803-809.
5. Meindok H, Langer B. Liver scan in Budd-Chiari syndrome. *J Nucl Med* 1976;17:365-368.
6. Parker RGF. Occlusion of the hepatic veins in man. *Medicine* 1959;38:369-402.
7. Boyer TD. Major Sequelae of cirrhosis. In: Wyngaarden JB, Smith LH, eds. *Cecil textbook of medicine*. 17th ed. Philadelphia: WB Saunders Company, 1985:840-845.
8. Robbins SL, Cotran RS, Kumar V. The liver and biliary tract. In: Robbins SL, Cotran RS, Kumar V, eds. *Pathologic basis of disease*. 3rd ed. Philadelphia: WB Saunders, 1984;884-959.
9. Carulli N, Boraldi F, Roncaia R, Piaggi W. Liver scans in the Budd-Chiari syndrome. *JAMA* 1973;223:1161.
10. Chaudhuri TK, Chaudhuri TK, Suzuki Y, Christie JH. Liver scan in the Budd-Chiari syndrome. *JAMA* 1972;221:506-507.