

Technetium-99m-Medronate Uptake in Hepatic Necrosis Associated with Budd-Chiari Syndrome

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A ^{99m}Tc -MDP bone scan performed on a 52-yr-old female for possible bone metastasis revealed prominent hepatic uptake. Subsequently, a ^{99m}Tc -SC scan revealed tracer uptake in the caudate lobe with diminished uptake in the remainder of the liver. Further imaging with Doppler ultrasound and hepatic venography confirmed a diagnosis of Budd-Chiari syndrome. Hepatic necrosis, demonstrated on CT imaging, was secondary to Budd-Chiari syndrome and was felt to be the cause of ^{99m}Tc -MDP hepatic uptake in this patient.

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The radiologic manifestations of hepatic vein occlusion (Budd-Chiari syndrome) have been well described in the literature (1,2). The scintigraphic findings in radionuclide imaging of Budd-Chiari syndrome using ^{99m}Tc -sulfur colloid (SC) are normal or increased activity involving the caudate lobe, with diminished uptake in the remainder of the liver (3-5). This report presents a patient who had imaging findings of Budd-Chiari syndrome and whose bone imaging study revealed hepatic uptake of ^{99m}Tc -medronate (MDP), secondary to hepatic necrosis with microcalcifications. Hepatic uptake of ^{99m}Tc -labeled phosphates related to hepatic necrosis has been described, but apparently not in relation to Budd-Chiari syndrome (6-9).

CASE REPORT

A 52-yr-old female presented with a palpable lump in her breast. Mammography and subsequent breast biopsy revealed probable infiltrating ductal carcinoma. Physical exam revealed hepatosplenomegaly but no other abnormal findings. An abdominal CT scan at an outside hospital revealed low density throughout the right and left lobes of the liver which was thought to represent metastatic disease. Ultrasound was also interpreted as probable metastatic disease to the liver with ascites. The patient developed nausea, vomiting, increasing abdominal pain and renal failure and was transferred to this hospital for care. Her physical

exam now revealed a protuberant abdomen, icterus and a fluid wave indicative of ascites.

Upon admission, the patient was in acute renal failure with a blood urea nitrogen of 73 mg/dl (normal: 8-20) and a blood creatinine of 5.1 mg/dl (normal: 0.6-1.1). Direct bilirubin measured 3.3 mg/dl (normal: 0-0.4), total bilirubin measured 3.8 mg/dl (normal: 0-1.3), and alkaline phosphatase measured 351 U/liter (normal: 0-115). She also demonstrated a coagulopathy with prothrombin time (PT) of 17 sec (control: 11 sec) and partial thromboplastin time (PTT) of 39 sec (control: 26 sec). A complete blood count revealed a white blood count of 24,300 cells/ μl (normal: 4,800-10,800) and a red blood cell count of 6.1×10^6 cells/ μl (normal: $4.2-5.4 \times 10^6$). Bone marrow aspirate and radionuclide red cell volume determination were consistent with polycythemia vera.

The patient underwent a CT scan which revealed an area of diffuse low density throughout the right and left lobes of the liver with sparing of the caudate lobe. Diffuse microcalcifications were also present throughout the right and left lobes which were not demonstrable on plain film examination. Marked ascites was present as well as multiple splenic infarcts (Fig. 1). These findings were considered most compatible with Budd-Chiari syndrome secondary to polycythemia vera (10). Doppler ultrasound of the liver documented absent flow in the hepatic veins further supporting the diagnosis of Budd-Chiari syndrome (11).

In light of all imaging studies strongly supporting a diagnosis of Budd-Chiari syndrome, review of the outside breast biopsy was recommended. In the interval, radionuclide bone imaging was performed with ^{99m}Tc -MDP to exclude any evidence of bony metastasis. This study showed prominent hepatic uptake including peripheral involvement of the right and left lobes. There was no evidence of bony metastasis (Fig. 2). Other ^{99m}Tc -MDP studies from the same MDP preparation revealed no hepatic uptake. Subsequent liver-spleen imaging with ^{99m}Tc -SC revealed tracer uptake in the area of the caudate with diminished uptake in the remainder of the liver. Also demonstrated was evidence of ascites and shift of tracer activity to an enlarged spleen, to the bone marrow and to the lungs (Fig. 3). These findings are characteristic of Budd-Chiari syndrome.

Importantly, extensive review of the pathology specimen from the patient's breast biopsy revealed benign papillomatosis. A liver transplant was felt to be the treatment of choice in this patient with hepatic necrosis from Budd-Chiari syndrome and a pre-transplant work-up, including angiography, was performed. Angiography, one of the classic imaging methods for the diagnosis of Budd-Chiari syndrome, revealed the characteristic "spider web" pattern of intrahepatic collateral veins on hepatic wedge

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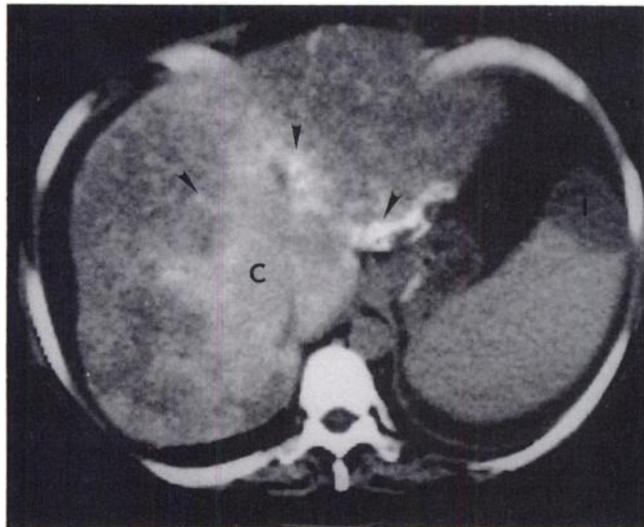


FIGURE 1. Abdominal CT reveals diffuse hypodensity throughout much of the right and left lobes of the liver with associated microcalcifications (arrowheads). There is virtual sparing of the caudate lobe (C). Also note splenic infarct (I) and ascites.

venography with an irregular filling defect in one of the hepatic veins (12) (not shown).

DISCUSSION

The Budd-Chiari syndrome is a relatively rare syndrome of hepatic vein or inferior vena cava occlusion. It has no known etiology in two-thirds of cases. It is recognized to be associated with polycythemia vera, as in this case, as well as neoplasms, trauma, congenital abnormalities, pregnancy, and oral contraceptives. Radiographic imaging with CT, sonography, angiography, scintigraphy, and MR has been well described. Technetium-99m-SC imaging in patients with Budd-Chiari syndrome characteristically reveals increased activity in the caudate lobe with diminished uptake in the remainder of the liver. The caudate lobe, having a separate venous drainage to the inferior vena cava, is maintained with resultant hypertrophy. In addition, there is usually evidence of tracer shift to the spleen and bone marrow (3,4).

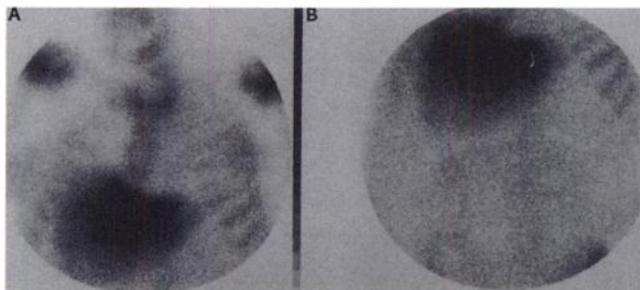


FIGURE 2. Technetium-99m-MDP bone imaging study. Anterior chest (A) and anterior abdominal (B) images show prominent hepatic uptake.

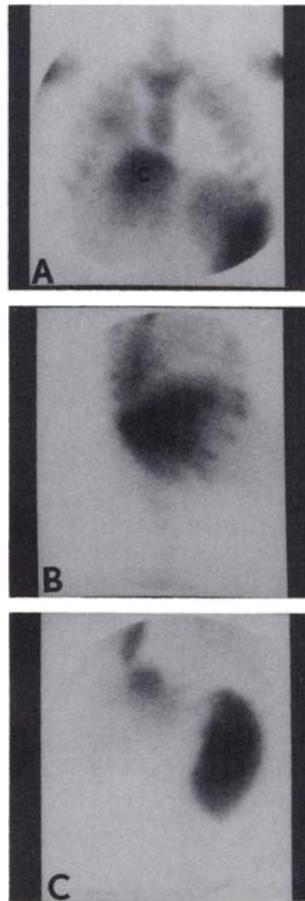


FIGURE 3. Technetium-99m-SC imaging study. Anterior (A), right lateral (B) and LAO (C) images reveal the characteristic scintigraphic findings in Budd-Chiari syndrome of increased caudate (C) activity with diminished uptake in the remainder of the liver. Also note tracer redistribution to the spleen, bone marrow, and lungs along with splenomegaly and probable ascites.

In the case presented, ^{99m}Tc -MDP imaging reveals prominent hepatic uptake secondary to hepatic necrosis (with microcalcifications) from Budd-Chiari syndrome. Uptake of ^{99m}Tc -pyrophosphate and ^{99m}Tc -HMDP has been reported in cases of hepatic necrosis, but these cases were not associated with Budd-Chiari syndrome (6-9). Extrasosseous uptake on skeletal scintigraphy has been described in association with many conditions. The mechanism by which radiolabeled phosphates such as ^{99m}Tc -MDP accumulate in tissues other than bone is unclear. However, the association of uptake of radiolabeled phosphates with hepatic necrosis is well-established. The uptake of bone-seeking radiopharmaceuticals in extrasosseous locations may be due to calcium accumulation in mitochondria after disruption of the cell membrane or calcium deposition in other areas of necrotic tissue (13,14). While the uptake of ^{99m}Tc -labeled phosphate compounds has been shown in hepatic necrosis, this phenomenon apparently has not been reported in hepatic necrosis related to Budd-Chiari syndrome. The pathophysiology of Budd-Chiari syndrome results in a ^{99m}Tc -SC uptake pattern which is characterized by its prominent uptake within the caudate lobe. The physiologic information provided by the uptake of radiolabeled phosphates as a marker of hepatic necrosis may be useful in certain clinical situations as exemplified in this case.

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