Renal Uptake of Tc-99m Methylene Diphosphonate after Radiation Therapy

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For most renal lesions, the abnormalities demonstrated with Tc-99m skeletal agents resemble those with Tc-99m renal agents. In contrast, an instance of obviously increased renal uptake of Tc-99m MDP due to radiation therapy is reported. There were only minimal imaging abnormalities with Tc-99m glucoheptonate. The radiotherapy was given to a child after surgical removal of a left adrenal ganglioneuroblastoma. The increased accumulation occurred, 5 mo after radiotherapy, in an unshielded portion of the renal parenchyma, which received 3000 rads in 28 days. The abnormality had disappeared by 11 mo. All other imaging studies were normal, including i.v. urography, ultrasonography, and computed tomography.


Renal abnormalities are often discovered incidentally in skeletal imaging studies performed with Tc-99m complexes of pyrophosphate or diphosphonates. Usually they cause a decreased concentration or a defect in the kidney area similar to the changes seen with Tc-99m renal agents. In contrast, this report describes the time sequence of increased renal uptake of Tc-99m MDP following radiation therapy.

At a routine physical examination, a firm, left upper-quadrant mass was discovered in an asymptomatic 7-year-old white girl. Specific questioning uncovered no history of palpitation, sweats, diarrhea, tachycardia, weight loss, fever, abdominal pain, shortness of breath, or urinary symptoms. Routine blood values, urinalysis, and 24-hr excretion of epinephrine, norepinephrine, 5-hydroxyindole acetic acid, and vanilmandelic acid were normal. Chest x-ray film, barium enema, and liver-spleen scan were normal. A partially calcified mass in the left upper retroperitoneum was seen on an i.v. urogram, and a corresponding six- by eight-cm solid tumor was shown by ultrasonography. On aortography, the mass was mildly vascular, supplied by the middle and inferior adrenal vessels.

At surgery a seven- by twelve-cm, whitish-tan, lobulated, firm mass was identified anterior to the left kidney and inferior to the spleen and pancreas. The left renal vein was adherent to the tumor anteriorly and was sprayed around the mass. The tumor was adherent to the capsule of the kidney, but was stripped free with relative ease. Enlarged nodes in the para-aortic chain and left renal hilum, from one to six cm in diameter, were removed without compromising the blood supply of the left kidney even though they surrounded the renal vessels. "Malignant ganglioneuroma (ganglioneuroblastoma) of the left adrenal involving lymph nodes" was reported.

Postoperative irradiation was given through directly opposed 11- by 15-cm, anterior and posterior fields with a four-MV linear accelerator. Therapy began on the 18th postoperative day and both fields were treated at each session, 5 days per week. The entire left kidney received 1,650 rads
in 150-rad increments during 15 elapsed days. A lead block of 5 half-value thicknesses was then interposed to protect the lateral half of the kidney, and the dose to the involved lymphatic region was raised to a total of 3,000 rads in 28 days (Fig. 1).

FIG. 2. (A) Tc-99m MDP bone scintiphoto 2 mo after radiotherapy; normal kidneys. All bone images taken 2 hr after injection. (B) Bone image after 5 mo, demonstrating increased Tc-99m MDP uptake in supero-medial aspect of left kidney. (C) Bone image after 6 mo (posterior view) showing intense uptake along entire medial aspect of left kidney. (D) LPO view from same procedure as in C: increased activity definitely in a renal parenchymal distribution. (E) Normal distribution of Tc-99m MDP in kidneys after 11 mo. (F) One-hour image from a Tc-99m glucoheptonate renal study after 5 mo (corresponding in time to the positive MDP images).
chloromerodrin; the lateral portion of the kidney had been excluded from the radiation portal. A radiation dose of 2300 R, delivered in 5 wk, has been considered hazardous to the renal parenchyma (2). However, temporary reduction in glomerular filtration rate and renal plasma flow has been observed in man with doses as low as 400 rads (5). Further investigation will be required to determine the radiation-dose threshold for the increased renal uptake of Tc-99m skeletal agents in radiation nephritis.

REFERENCES


Reversible Functional Asplenia
In Chronic Aggressive Hepatitis

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A 61-year-old man presented with aggressive hepatitis. Howell-Jolly bodies were present in circulating erythrocytes and the spleen failed to accumulate intravenously administered Tc-99m sulfur colloid. The patient thus demonstrated functional asplenia. He was treated with high doses of steroids. Four years later, Howell-Jolly bodies were no longer present in circulating erythrocytes. In addition, the spleen had regained the ability to accumulate intravenously injected radiocolloid. Hence, the patient had reversed his functional asplenia. The reported cases of this disorder (reversible functional asplenia) were reviewed and a preliminary classification was proposed.


The concept of functional asplenia (anatomic presence of the organ but without the ability to accumulate intravenously administered radiocolloid) was first described in association with sickle cell disease in 1969 (7). Since then, functional asplenia has been identified in several disorders (2,3). It has also been recognized that some instances of functional asplenia may be transient or at least temporarily reversible (4,5). For example, functional asplenia associated with sickle-cell anemia could be transiently reversed by transfusion of normal red blood cells (4). Reversible functional asplenia has been demonstrated in some cases of cyanotic congenital heart disease (5), and episodically in hemoglobin SC disease (6). Recently a child with combined immunodeficiency disease has been identified as having reversible functional asplenia (7). In the present report we describe a case of reversible functional asplenia associated with chronic aggressive hepatitis. A classification of the disorders associated with this reversible splenic dysfunction is proposed.

CASE REPORT

A 61-year-old white man had been admitted to the hospital 4 years previously because of fever, anorexia, and jaundice. Liver-function tests at that time were grossly abnormal with total serum bilirubin of 4.2 mg/dl, alkaline phosphate 106 units, SGOT 460, and SGPT of 502. Hemoglobin was 14 g/dl with a hematocrit of 40%. There were abundant Howell-Jolly bodies in the peripheral erythrocytes. A radiocolloid liver-spleen scan, performed with 3 mCi of Tc-99m sulfur colloid i.v. showed hepatomegaly with a pattern consistent with diffuse hepatocellular disease. The spleen was not visualized and was presumed to be functionally absent (Fig. 1, top). A liver biopsy showed hepatic necrosis with swelling of hepatocytes with granular cytoplasm. There were areas of inflammatory bridging of portal tracts, as well as early peripheral fibrosis. A diagnosis of chronic aggressive hepatitis with functional asplenia was made. The patient was started on 40 mg of prednisone daily. This was reduced to

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