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Pitfalls in Technetium-99m HIDA Biliary Imaging: Duodenal Diverticulum Simulating the Gallbladder

Imaging of the gallbladder with Tc-99m iminodiacetic acid compounds is being used increasingly to diagnose acute cholecystitis. Here we report an interesting pitfall that we encountered recently. The radiotracer accumulated in a duodenal diverticulum that simulated the gallbladder. Oblique and right lateral views clarified the anatomical relationships and led to a correct interpretation of the study.

A 68-year-old black female presented with anorexia, vomiting, and diarrhea associated with sharp upper abdominal pain for 2 days. Her temperature was 37°C, pulse-100, BP 140/70 with orthostatic changes. She was moderately dehydrated. The rest of her examination was unremarkable. Her white blood cell count was 9500, with 45% bands and 47% polymorphonuclear leukocytes. The stool was brown and negative for polymorphonuclear cells. A Tc-99m HIDA* scan was performed because of the clinical suspicion of acute cholecystitis (Fig. 1, left). Following the intravenous injection of 5.0 mCi of technetium-99m HIDA, anterior images of the right upper quadrant were taken at 1 and 5 min, and every 5 min thereafter for 1 hr. The image at 25 min (Fig. 1, left, A) revealed a curvilinear structure identified as the distal common bile duct and descending duodenum. The hepatic ducts were poorly seen. At 50 min (1B) a dense accumulation of tracer was superimposed on the common bile duct and duodenum. Several loops of jejunum were also visualized. A left anterior oblique view at 65 min showed tracer activity close to the common bile duct and duodenum (1C). On the right lateral view (1D) the tracer appeared well behind the expected location of a normal gallbladder. Our interpretation was nonvisualization of the gallbladder due to acute or chronic cholecystitis. Ultrasound (Fig. 1, center) revealed the anterior position of the gallbladder just under the anterior abdominal wall (arrow). An upper G.I. series, performed a year before this study, had revealed a duodenal diverticulum in the second portion of the duodenum (Fig. 1, right), so we concluded that the Tc-99m HIDA had accumulated in the duodenal diverticulum. A cholecystectomy revealed acute and chronic cholecystitis with cystic duct obstruction.

Technetium-99m HIDA biliary imaging is helpful in the early assessment of patients with suspected cholecystitis (*1*). Hepatic uptake of tracer peaks within 5-10 min, with visualization of the common bile duct, gallbladder, and duodenum occurring 15-30 min after injection. Persistent nonvisualization of the gallbladder indicates obstruction of the cystic duct due to acute or chronic cholecystitis and/or cholelithiasis. Views 4-24 hr after injection may be required, as in some cases gallbladder visualization is delayed. Oblique and right lateral views are necessary for reliable location of the gallbladder or other areas of unusual persistent activity, which include abnormally located gallbladders or duodenal diverticula.

Abnormal positions of the gallbladder have been reported in the left upper quadrant, midline, abdominal wall, suprahepatic and intrahepatic regions, in the falciform ligament, and in retroperitoneal areas (*2*). In our study, the ultrasound examination allowed us to exclude the possibility of an abnormally placed gallbladder. The upper G.I. series had revealed a duodenal diverticulum in the area where the radioactivity was detected. Duodenal diverticula are common. They have been seen in 1-5% of upper G.I. series and noted in 22% of autopsy reviews. They are most common in the descending portion of the duodenum, but also occur in the third and fourth portions. Stasis of gastrointestinal contents is often seen because of absence of the muscular layer of the bowel wall. They are of little clinical importance unless extremely large and cause obstruction symptoms (*2*).

Oblique and right lateral views are necessary to avoid pitfalls in the interpretation of hepatobiliary tracer studies. Other imaging modalities, such as ultrasound and barium studies, may be helpful in selected patients. This case illustrates that a duodenal diver-

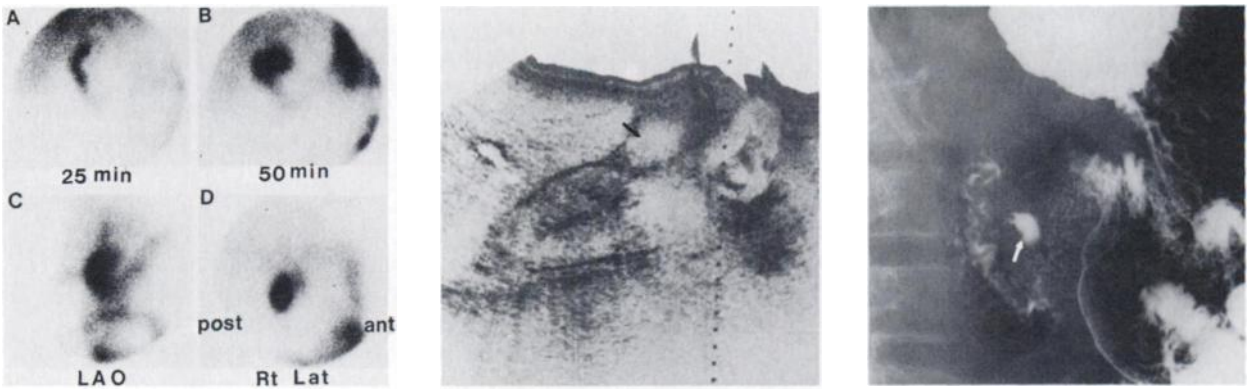

FIG. 1. (*Left*) Tc-99m HIDA study. (A) at 25 min, curvilinear streak of activity is due to common bile duct and duodenum. (B) at 50 min, a focus of tracer activity is superimposed upon the curvilinear structure. (C) LAO at 65 min shows focus of tracer retaining proximity to curvilinear common bile duct and duodenum. (D) Right lateral view: focus of tracer lies deep to anterior abdominal wall. (*Center*) Ultrasound in a longitudinal section, 8 cm to right of midline. Gallbladder (arrow) shown just below anterior abdominal wall. Incidental finding was a renal cyst at right lower pole. (*Right*) Upper G.I. series shows barium-filled diverticulum of descending duodenum (arrow).

ticulum could be mistaken for the gallbladder unless all structures are properly identified in three dimensions.

FOOTNOTE

* n-(2,6-dimethylphenylcarbamoylmethyl) iminodiacetic acid, Union Carbide Corp., Tuxedo, NY.

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Transient Functional Hyposplenia and Fever

The concept of hyposplenism was established by Dameshek in 1955 (*1*) when he reported a case of nontropical sprue and hyposplenism, first suspected because of the appearance of Howell-Jolly bodies and target cells in the peripheral smear. In 1969 Pearson et al. (*2*) described the inability of an anatomically present spleen to accumulate radioactive colloid in children with sickle cell disease. It became evident subsequently that hyposplenia occurred in other diseases as well (*3-6*).

We describe a case of acquired transient functional hyposplenia associated with a fever of unknown origin.

A 45-year-old black male presented with a 1-wk history of fever and chills. His fever reached 104°F daily and was accompanied by headache and weight loss.

On physical examination, he was in moderately acute distress, with a rectal temperature of 104°F and a heart rate of 105. There was a soft, grade I/VI midsystolic murmur at the left sternal border. Liver was palpable 2 cm below the right costal margin, with a 12-cm span. The spleen was not palpable. The remainder of his examination was unremarkable.

Hemoglobin was 8.8 g/dl, hematocrit 26.6%, and white blood cell count 4,300, with 43% polymorphonuclears, 7% bands, 48% lymphocytes, and 2% monocytes. The platelet count was 198,000. The reticulocyte count was 0.6%. Peripheral smear revealed Burr cells, schistocytes, occasional spherocytes, but no Howell-Jolly bodies. Prothrombin time, partial thromboplastin time, fibrinogen, and thrombin time were normal. Fibrin degradation products were present. Serum haptoglobulin was 160 (normal 60-170 meq%). Direct Coombs test was negative. G6PD was decreased to 69 (normal 140-280 units per billion cells). Hemoglobin electrophoresis revealed 94% of Hb A and 3.3 of Hb A2. The bone marrow aspirate showed decreased cellularity. Chest radiograph and the biochemical profile were normal. LE preparation, RA latex, and ANA were negative. Serum assays for immune complexes were not done.

His temperature continued to spike to 104°F daily and he remained acutely ill. Bacteriologic and serologic studies were unrevealing. On the second hospital day, a Tc-99m sulfur colloid liver-spleen scan revealed minimal hepatomegaly with poor visualization of the spleen. On the fifth hospital day, a splenic flow study was performed. The subsequent static images yielded a spleen that was functional but not of normal intensity (Fig. 1, left).

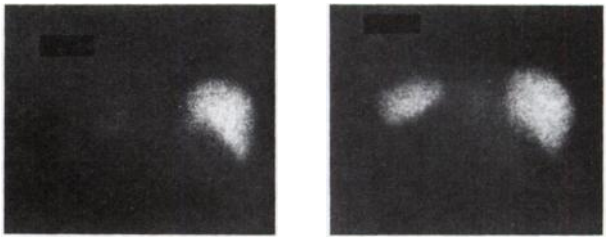

FIG. 1. (Left) Liver-spleen scan, posterior view on Day 5, revealing functional but weak spleen. (Right): Normal liver-spleen scan, posterior view, 6 wk after discharge.

(Although splenic uptake appeared to have increased since the first study, a valid comparison could not be made because of differences in technique.) By the seventh day, the patient was afebrile. Haptoglobulin at this time was decreased to 12. A few Howell-Jolly bodies were noted on the peripheral smear for the first time. By the 12th hospital day, his reticulocyte count increased to 7.3%. His hematocrit rose to 28.1%, and his white blood cell count to 5,200.

Following discharge, he continued to improve symptomatically and his hematocrit rose to 44% three months later. A repeat spleen scan done 6 weeks after discharge was normal (Fig. 1, right).

This patient presented with an acute febrile illness, the cause of which was not determined. His hematological status was noteworthy for the development of anemia and leukopenia. He developed transient functional hyposplenia, manifested by a decreased uptake of technetium sulfur colloid on splenic scan, and by the presence of Howell-Jolly bodies in the peripheral smear.

In 1978, Spencer et al. (*7*) reviewed the world literature and found an incidence of six cases of functional asplenia in over 4,476 consecutive liver scans at three hospitals, from January 1975 to July 1976. He proposed a classification of causes of functional asplenia and divided them into two categories: circulatory disturbances and effects on reticuloendothelial cell function (Table 1).

The functional hyposplenia that occurred in our case does not appear to fit into either of these categories. A transient splenic

TABLE 1. CAUSES OF FUNCTIONAL ASPLENIA

I. Circulatory disturbances
  (a) Gross
    1. Splenic artery blockade
    2. Splenic vein occlusion
    3. Combined vascular occlusion
  (b) Microscopic
    1. Hemoglobin SS
    2. Hemoglobin SC
    3. Thalassemia
    4. Other, such as polycythemia vera
II. Effects on splenic reticuloendothelial (RE) cells
  (a) RE blockade and irradiation (thorotrast loading)
  (b) Combined irradiation and chemotherapy
  (c) Replacement of RE cells by tumor (such as lymphoma, myeloma, or metastases) or by infiltrate such as amyloid
  (d) Cellular damage, as in nutritional deficiency or immunocompetent problems (celiac sprue)
  (e) Possible effects of splenic anoxia