

### Still More Applications of Hepatobiliary Scintigraphy

“When can HIDA help?”, asked a recent *Journal* editorial (1), referring, of course to the new technetium-labeled acetanilide-iminodiacetic derivatives that have become the agents of choice for hepatobiliary imaging (2). Several areas have been suggested where this group of agents might significantly assist in the clinical diagnostic process:

1. the differential diagnosis of obstructive jaundice;
2. determination of the presence of cystic-duct patency in suspected acute cholecystitis;
3. visualizing unusual pathways of bile drainage and intestinal bile flow;
4. distinguishing the left lobe of the liver from spleen when oblique views cannot separate the two organs.

One might add to these a fifth use, that of determining whether filling defects noted on the technetium sulfur colloid liver scan are related to normal (or abnormal) hepatobiliary function. Hepatic filling defects are, of course, nonspecific, but with ultrasound or I-131 rose bengal one can distinguish a prominent gallbladder or choledochal cyst from other causes of such defects (3,4). Tc-99m HIDA has proven extremely valuable in our laboratory in demonstrating that some nonfunctioning defects—either within the lower right hepatic lobe or appearing as possible areas of extrinsic compression at the right inferior margin of that lobe—are due to a normally functioning hepatobiliary system. We inject the patient with Tc-99m HIDA immediately after observing such abnormalities on the Tc-99m sulfur colloid (TcSC) liver scan and visualize the normal gall bladder within 15 to 30 min.

The filling in of such “cold areas” on TcSC scan in another disorder of the hepatobiliary system is the subject of a report by Yeh, Liu, and Huang of the Veterans General Hospital, Taiwan, in this issue of the *Journal* (5). These workers have reproduced the data of Wang et al. (6) in showing the value of another technetium hepatobiliary agent, Tc-99m pyridoxylidene glutamate (PG) in the diagnosis of intrahepatic lithiasis (Tc-99m HIDA was not available to Yeh et al. at the beginning of this study). This condition, which is uncommon in North America, can be classified into three forms:

1. primary intrahepatic lithiasis involving only the intrahepatic biliary tree;
2. mixed intra- and extrahepatic lithiasis including “migratory intrahepatic lithiasis” caused by improper operative technique;
3. secondary intrahepatic lithiasis related to anatomic conditions causing stricture and stasis or infection (7).

This latter type of hepatic-stone disorder is a complication of biliary tract tumors, of Caroli's disease (congenitally dilated bile duct) (8,9), and also of inflammatory conditions of the biliary tree, especially of bacterial and parasitic (round worms, liver fluke) origin. Intrahepatic calculi are pigment gallstones, consisting largely of bilirubin and its derivatives. As to pathogenesis, it is reasonable to hypothesize that, in part, these calculi are produced in the biliary passages by organisms that hydrolyze conjugated bilirubin (bilirubin glucuronide) with their beta-glucuronidase (10).

The incidence of intrahepatic lithiasis in Western countries has been reported recently as ranging from 1 to 2.4% of all biliary lithiasis (7), although Yeh et al. quote numbers of 5 to 8% from much older series. In the Orient up to 50% of patients with biliary stones may have pigment lithiasis with intrahepatic stones (5). The clinical presentation of intrahepatic lithiasis is that of Charcot's triad—upper abdominal pain, fever, and jaundice (11)—in 58% of a recent series of 36 patients with the condition, with 97% of these experiencing pain, 77% jaundice, and 60% fever (8).

The 19 completely studied cases of Yeh et al. all showed intrahepatic cold areas suggestive of biliary dilatation on TcSC scan (compared to two of 14 in the data of Wang et al. (6)). Technetium

tium-99m PG always showed complementary intrahepatic pooling, probably secondary to partial obstruction, although adsorption of Tc-99m PG to the calculi was not excluded. Similarly, in the data of Wang et al., "local residual liver activity" was seen in all 14 patients with intrahepatic stone, but in only one of 11 with a common duct stone. In the 12 cases of Yeh et al., where a complete contrast cholangiographic study was performed, only one had a "positive" result. In the data of Yeh et al. the sensitivity of intrahepatic pooling in the Tc-99m PG scan was 100% and specificity was 95%, although it is not clear how the 101 "apparent normal individuals acting as the control group" were chosen. Heretofore the diagnosis of intrahepatic calculi has been quite difficult preoperatively, since intravenous cholangiography does not permit a greatly detailed study of the intrahepatic biliary tree, although there are predictable roentgenographic signs (7). Percutaneous transhepatic cholangiography may be more demonstrative, with endoscopic retrograde cholangiography "that most important preoperative investigatory method." However, in only four of 36 was the diagnosis made preoperatively, in a series published in 1979, without benefit of HIDA scanning (7).

Hepatobiliary scanning, supplemented if necessary with intravenous cholecystokinin, is becoming an important part of the workup of patients whose symptoms suggest acute cholecystitis or cholangitis (12,13). The two sets of data from Taiwan highlight yet another important use of Tc-99m hepatobiliary scanning for intrahepatic calculi, a condition affecting millions of people, especially in the Orient.

EDWARD B. SILBERSTEIN  
University of Cincinnati Medical Center  
Cincinnati, Ohio

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