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Abnormal Accumulation of Technetium-99m Hepatobiliary Agent in a Case of Idiopathic Bile Peritonitis

Since the publication by Spencer et al (*1*), iodine-131 rose bengal (*2*) and the recent Tc-99m IDA derivatives have been used to demonstrate bile leakages following cholecystectomy (*3*), biliary-intestinal tract anastomosis (*4*), ductal rupture in hepatic trauma (*5*), and gallbladder perforation (*6*). Abnormal accumulation of Tc-99m hepatobiliary agent with idiopathic bile leak has also been described recently (*7*). In that patient, however, there was no clinical or chemical evidence of jaundice, cholelithiasis, biliary ductal dilatation, cholecystitis, or pancreatitis. Although a small amount of bilious fluid was aspirated, the significance of the abnormal accumulation of hepatobiliary agent is difficult to determine since there was no surgical documentation of a site of leakage.

We recently encountered a patient who had intraperitoneal extravasation of Tc-99m hepatobiliary agent, with surgical confirmation of idiopathic bile peritonitis.

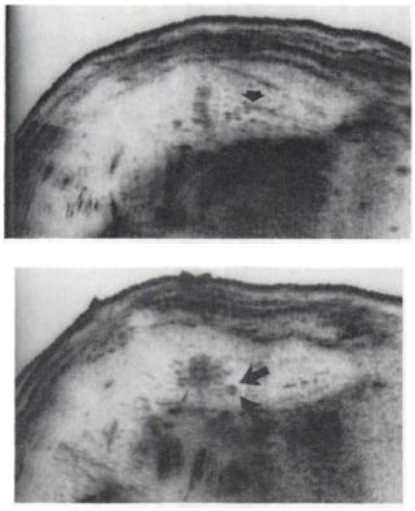

FIG. 1. Ultrasonogram of biliary tree showing dilated duct (top, arrow) with calculus (bottom two arrows) in left lobe of liver.

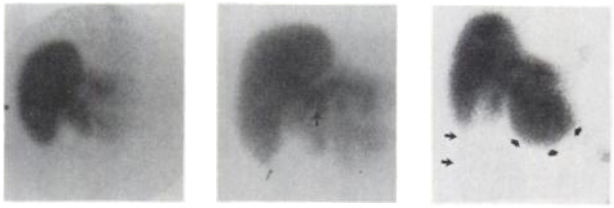

FIG. 2. Anterior hepatobiliary images. Left: 10 min after tracer injection, showing configuration of left lobe of liver. Center: 2 hr after injection; common duct is obstructed (arrow), gallbladder not visible. Right: 7 hr after injection. Increased activity in left lobe (3 arrows); tracer along right side of abdomen (2 arrows); gallbladder still invisible.

A 79-yr-old white male was admitted with upper abdominal pain and jaundice of a few days' duration. There was no fever, nausea, vomiting, constipation, or diarrhea. The patient denied alcohol abuse. There was no past history for similar pain.

Physical examination demonstrated marked tenderness in the epigastrium and in both upper quadrants of the abdomen, with guarding. The pertinent laboratory values were: total bilirubin 6.0 mg per dl, direct bilirubin 3.3 mg per dl, and serum amylase 902 units. With the initial impression of obstructive jaundice due to calculus in the common bile duct or acute pancreatitis, an ultrasonogram of the biliary tree was obtained, followed 24 hr later by radionuclide hepatobiliary imaging and TCT examination of the upper abdomen. The ultrasound examination (Fig. 1) demonstrated a moderately dilated, thick-walled gallbladder containing several calculi. There was dilatation of the biliary tract, with a focal density indicating calculus in a dilated duct in the left lobe of the liver. The pancreas was not visualized. There was no abnormal fluid collection in the upper abdomen. The hepatobiliary images (Fig. 2) were obtained with 5.4 mCi (200 MBq) of Tc-99m diisopropyl IDA derivative. Sequential anterior views of the upper abdomen were obtained until 7 hr after injection. The gallbladder was not visualized. The radioactivity was not seen in the gastrointestinal tract until 2 hr after the injection. There was increasing tracer concentration along the left lobe of the liver and a curvilinear collection of the radionuclide was noted along the right abdominal wall. These findings were thought to represent extravasation of bile. In addition, there were multiple small col-

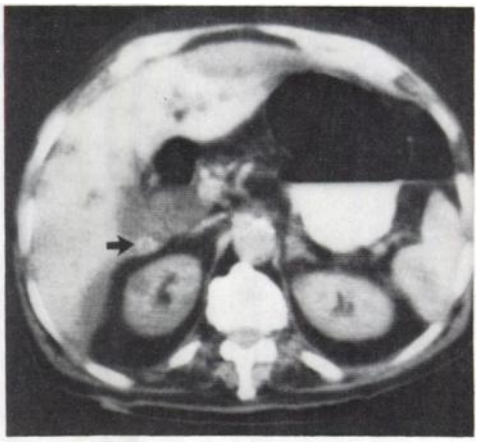

FIG. 3. TCT through liver (not enhanced) demonstrates fluid collection along anterior aspect of left lobe and in subhepatic space. Calculus (arrow) is also seen in gallbladder.

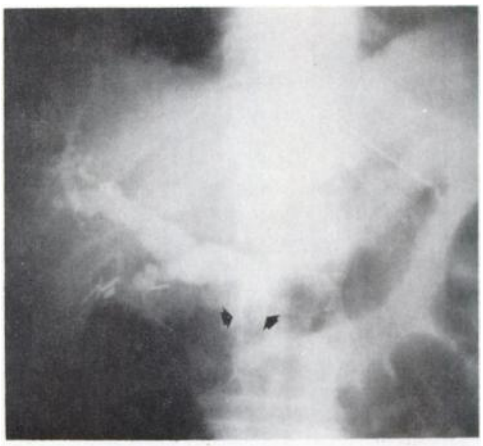

FIG. 4. Intra-operative cholangiogram. Large calculus lies in distal common bile duct (two arrows), with no discharged contrast material in duodenum. Bile ducts are markedly dilated. Left hepatic duct is not opacified.

lections of tracer in the abdomen in a view obtained at 7 hr; these were considered to lie within the small bowel. Retrospectively, they might have been due to intraperitoneal extravasation of the radionuclide. The common duct was mildly dilated. Subsequently, TCT examination of the upper right abdomen (Fig. 3) was performed with oral administration of dilute barium sulfate. There was fluid in the peritoneal cavity, with a large collection along the anterior aspect of the left lobe. There was no solid or cystic lesion in the pancreatic head.

Laparotomy revealed a large amount of bile-stained fluid in the peritoneal cavity. There was no tear or perforation in the stomach, duodenum, gallbladder, or the extrahepatic ducts. A small area of greenish discoloration was seen on the anterior aspect of the left lobe of the liver. This was thought to be the site of bile leakages or permeation. In the initial intra-operative cholangiogram there was no flow of contrast material into the duodenum from the common bile duct, and only the right hepatic duct was opacified (Fig. 4). Exploration of the biliary tree revealed several calculi in the dilated common duct and one in the left hepatic duct. These were removed, and a repeat operative cholangiogram demonstrated good opacification of the entire biliary tree with no retained calculi. A cholecystectomy was performed.

There are several possible causes for extravasation of bile. The increased intraductal pressure secondary to distal obstruction may cause perforation of a duct wall weakened due to adjacent cholangitic abscess or scar formation (*8*). It may be secondary to the effect of pancreatic trypsin on bile passages with mucosal ulceration and consequent perforation (*9*). A third theory suggests that bile leakage may be secondary to the perforation of mucous-gland diverticula present in the ductal wall. It is common not to visualize a definite tear or perforation in the biliary tree, which may seal off once the intraluminal pressure is released (*8*). On the other hand, there need not be a tear or perforation in the biliary tract for bile to extravasate. There is enough evidence to indicate that bile can filter or permeate through the ductal wall if there is pancreatic trypsin mixed with bile (*9*). In our patient, probably the latter mechanism caused the bile leakage from the occluded left hepatic duct.

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