Bile Leak From Gallbladder Perforation Mimicking Bowel Activity and a False-Negative Result in a Morphine-Augmented Cholescintigraphy

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Cholescintigraphy of a patient with bile leak demonstrated intra-abdominal activity that mimicked normal bowel activity. Because the gallbladder was not visualized, morphine was injected intravenously. Gallbladder activity after morphine injection was misleading in the finding of chronic cholecystitis. Concurrent abdominal sonography and computerized tomography revealed a thickened gallbladder wall with a gallstone and pericholecystic fluid collection. Exploratory laparotomy confirmed acute and chronic cholecystitis, cholelithiasis, cholecdocholithiasis, and a pericholecystic abscess. The false-negative conclusion for acute cholecystitis in the patient’s morphine-augmented cholescintigraphy resulted from an acceleration of bile leakage due to pre-existing gallbladder perforation.

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CASE REPORT

A 70-yr-old male with a 3-day history of nausea, vomiting and abdominal pain was admitted with an impression of a small-bowel obstruction secondary to acute cholecystitis and questionable common bile duct obstruction. Two months previously, the patient had undergone total gastrectomy and Roux-En-Y esophagojejunostomy because massive hemorrhage of multiple gastric ulcers that extended to the gastroesophageal junction. His past medical history included a laryngectomy for squamous cell carcinoma and an above-the-knee amputation of the left leg 13 and 48 yr ago, respectively. Physical exam on admission revealed a midline scar on the abdomen, and left-lower-quadrant tenderness with mildly distended abdomen. Intravenous antibiotics were instituted on the day of admission.

The first hospital day the patient underwent hepatobiliary scintigraphy. Scintigraphy showed a prominent porta hepatitis and dilated common bile duct, with no obvious visualization of the gallbladder until after a 2-mg morphine intravenous injection. Mild and diffuse increase in uptake in the right abdomen was more prominent after morphine administration (Fig. 1). The same day, computerized tomography (CT) and ultrasound (US) of the abdomen were performed; the CT showed a thickened edematous gallbladder wall and was suggestive of pericholecystic fluid collection (Fig. 2). The US showed a thickened gallbladder wall and a single acoustic shadow in the gallbladder suggestive of a calculus (Fig. 3). Laboratory findings at the time of admission included alkaline phosphatase 1299 (n = 38–126 U/liter), AST 60 (n = 5–42 U/liter), ALT 35 (n = 7–60 U/liter) and amylase 84 (n = 30–110 U/liter).

On the second hospital day, the patient underwent an exploratory laparotomy. Operative findings included multiple gallbladder stones, perforated gallbladder, pericholecystic abscess, adhesion of the small bowel to the liver and gallbladder, numerous (50) common bile duct stones, and extensive bowel adhesion. Enterolysis of adhesions, cholecystectomy and common-bile-duct exploration were performed. Morphological diagnoses of the gallbladder confirmed acute and chronic cholecystitis and cholelithiasis.

DISCUSSION

Two linear areas of faint activity in the abdomen suggested bowel activity in our patient’s cholescintigraphy. During the imaging procedure, because no gallbladder activity was visualized, although some abdominal activity was apparent, morphine sulfate was administered intravenously to aid in the diagnosis of acute versus chronic cholecystitis. Any indication of gallbladder activity on the 60 min image was quite faint and difficult to detect. The right and left areas of abdominal activity (as indicated by solid arrows on the 45-min image) could represent activity in the bowel or not in the bowel. In either case, the persistent configuration throughout the imaging may be explained by intraperitoneal bowel adhesions, later found in exploratory laparotomy, which were consequent to the previous total gastrectomy.

Stones found in the common bile duct during surgery can cause nearly complete obstruction of the common bile duct. The small amount of drained bile in the bowel might not be enough to be visible on scintigraphic imaging. Once morphine is given intravenously, it causes contraction of the sphincter of Oddi resulting in increased intraluminal bile duct pressure and would then serve to hinder appear-

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ance of radioactivity in the bowel. Delay in appearance of bowel activity for more than 3.5 hr has been documented (1). The fact that our scintigraphic findings demonstrated more prominent activity in one of these two linear areas, concurrently with radioactivity of the gallbladder area, confirmed a bile leak from the perforated gallbladder. These findings reflected acceleration of bile leakage from the pre-existing gallbladder perforation.

Radioactivity in the gallbladder area after morphine administration may represent radioactivity in the gallbladder, radioactive in the pericholecystic region (pericholecystic activity), or both. In a relatively low-pressure condition, the gallbladder would accumulate radiotracer in the bile before the bile leaked out; therefore, radioactivity in the gallbladder region presumably represented both pericholecystic activity and intracholecystic activity. Whether morphine contributes to the perforation of the gallbladder is debatable (2). In our case, morphine might have aggravated bile leakage (evidenced by more activity in the regions of the intraperitoneal cavity). Without CT and/or US detection of pericholecystic fluid collection, leakage to the pericholecystic region might be missed.

Though morphine-augmented cholecsintigraphy can reduce imaging time from 4–24 hr to 60–90 min and serves as an alternative to delayed imaging in the differentiation of acute cholecystitis from chronic cholecystitis (3), false-positive results have been reported, especially in severely ill patients (4–6). False-negative results have also been reported (7). The explanation suggested for a false-positive was the dislodging of a cystic duct stone from increased pressure, allowing bile flow to the gallbladder. Our patient’s gallbladder visualization and histopathological confirmation of acute cholecystitis may constitute a false negative. Presumably his cystic duct was patent. Increased intraluminal pressure of the bile duct alone, secondary to sphincter of Oddi contraction in response to the morphine and the pressure of stones impacted in the common bile duct, allowed bile flow through the cystic duct and leakage from pre-existing gallbladder perforation.

Gallbladder perforation is an unusual condition, with a mortality rate of approximately 5% (8), that is usually secondary to acute cholecystitis (9). Cholescintigraphic patterns of gallbladder perforation include free-spill pericholecystic activity and chronic cholecystoenteric fistula (10). US findings of the gallbladder include pericholecystic fluid (10) or pneumobilia with gallstones, and CT findings include pericholecystic fluid collection (8,11). Cholescintigraphic detection of gallbladder perforation is reported to be 50% (10); US detection, 18% (10). Although cholecsintigraphy appears superior to US, both imagings are relatively insensitive in the detection of gallbladder perforation (10). Cholescintigraphy combined with 67Ga-citrate imaging (8), CT, and US (8,11) has been described; the combined radiologic imaging methods complement one another, leading to highly accurate preoperative diagnosis of gallbladder perforation (8,11). Our case concurred with previous reports in that fluid collection was demonstrated by CT and US, and cholelithiasis and thickened gallbladder wall were shown by US. In turn, these changes helped clarify the findings on cholecsintigraphy.
In summary, our case illustrated a false-negative result in morphine-augmented cholescintigraphy. Combined diagnostic imagings—cholescintigraphy, CT, and US—of the abdomen complement one another, enabling accurate preoperative diagnosis.

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REFERENCES