

The Role of Hepatobiliary Imaging in the Evaluation and Management of Patients with Common Bile Duct Gallstones

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CASE PRESENTATION

An 87-yr-old female presented with a chief complaint of severe epigastric pain. The pain had started 1.50 days earlier and lasted all day and part of the night. The pain was characterized as burning and radiating to her chest. She denied any radiation to her neck or arms and denied any dyspnea, although she did have some diaphoresis associated with the pain. The discomfort was sometimes related to eating and often was associated with belching. She had experienced two episodes of nausea and vomiting on the day prior to admission. She had been experiencing mild rectal bleeding for several months which had been attributed to hemorrhoids, and for which she had been taking iron supplements.

Her past medical history was significant for a myocardial infarction 13 yr earlier, as well as hypertension. Her past history was also significant for diverticulosis which was discovered by barium enema 11 yr earlier. Her surgical history was significant for a hysterectomy many years earlier. In addition, she had a 20-pack year smoking history, although she stopped smoking 13 years prior to admission. She also admitted to moderate alcohol consumption. The family history and review of systems were noncontributory.

The patient's medications at home included propranolol for hypertension, procainamide for occasional arrhythmias, dicyclomine for bowel spasticity, and ferrous sulfate for anemia.

Upon examination, the patient appeared thin but well developed and in no significant distress. Her temperature was 97.8 orally, pulse 60 bpm, blood pressure

146/40, and a respiratory rate of 18. The rest of her physical exam was essentially benign with the following findings: anicteric sclera; Grade II/VI nonradiating systolic murmur, which was loudest at the left sternal border; lungs were clear to auscultation and percussion; and a nearly normal abdominal exam with only minimal tenderness in the epigastric area and no evidence of organomegaly or abdominal masses. There were no signs of peripheral edema or other stigmata of congestive heart failure. On rectal exam, the patient's stool was brown and trace heme-positive.

Laboratory assessment was notable for a BUN of 30 mg/dl, a total bilirubin of 3.0 mg/dl, and a CBC revealed a hemoglobin level of 7.3 gm/dl with a white blood cell count of 11.6 thousand/ μ l. EKG demonstrated a normal rate and rhythm with left ventricular hypertrophy by voltage and nonspecific ST-T-wave changes but normal intervals and a normal axis. Chest x-ray was unremarkable except for mild cardiomegaly.

Due to her pain and cardiac history, she was admitted to rule out a myocardial infarction as well as to evaluate her anemia.

Over the following days, cardiac isoenzymes were obtained and her anemia was treated with blood transfusions, however, the exact source of the gastrointestinal bleeding remained unclear. Two days after admission, it was noted that she was becoming jaundiced. Liver function tests performed at that time demonstrated an alkaline phosphatase of 557 IU/l (normal <200) as well as moderately elevated liver transaminases (AST = 100 IU/l, ALT = 110 IU/l), and a markedly elevated total bilirubin of 11.7 mg/dl with a direct bilirubin of 7.6 mg/dl. Her white blood cell count also was rising and was measured at 14.3 thousand/ μ l with a left shift. She also developed a fever of 101.6 orally. These findings suggested obstructive jaundice and possibly cholangitis. Blood cultures were performed and the patient was started on ampicillin and gentamicin empirically (these cultures eventually demonstrated no growth). A hepatobiliary study was ordered and performed to evaluate

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biliary drainage. The scan, performed with ^{99m}Tc -diisopropylimino diacetic acid (DISIDA), demonstrated fairly good uptake of tracer into the liver, but there was no significant excretion into either the gallbladder, the common bile duct or the bowel. This did not change on delayed images obtained up to 4 hours after injection (Fig. 1). There was also a defect in the porta hepatis suggestive of a dilated common bile duct. The study was read as very suggestive of high-grade common bile duct obstruction. Next, an ultrasound was performed which demonstrated cholelithiasis and "sludge" in the gallbladder as well as a dilated common bile duct but normal intrahepatic ducts (Fig. 2). At this point, although it appeared that she would be ruled out for a myocardial infarction, her CPK-MB fractions were borderline and it was felt that she may have had some myocardial damage. Because the patient was thought to be at high risk for surgery, an endoscopic retrograde cholangiopancreatography (ERCP) was performed to evaluate the source of possible upper gastrointestinal bleeding as well for treatment of what appeared to be obstructive jaundice due to choledocholithiasis.

ERCP revealed no gastric lesions and showed that the duodenum was also normal except for some periampullary diverticuli. The pancreatic duct was normal, but the common bile duct was moderately dilated and a 1-cm stone was noted high in the common duct (Fig. 3). Attempts at retrieving the stone in a basket were unsuccessful, so a 1.2-cm sphincterotomy was performed to allow the stone to pass on its own.



FIGURE 1. A 4-hr delayed image of the hepatobiliary study performed with ^{99m}Tc -DISIDA. There is fairly good uptake of tracer by the liver with only a small amount of blood-pool activity remaining. Renal excretion is seen with much tracer in the urinary bladder. There is no evidence of excretion into either the bowel or gallbladder consistent with the "liver scan" appearance and strongly suggestive of common bile duct obstruction. There is also photopenia in the porta hepatis (arrows) probably due to dilated ducts. (Published with permission from *Clin Nucl Med* 1985;10:264.)

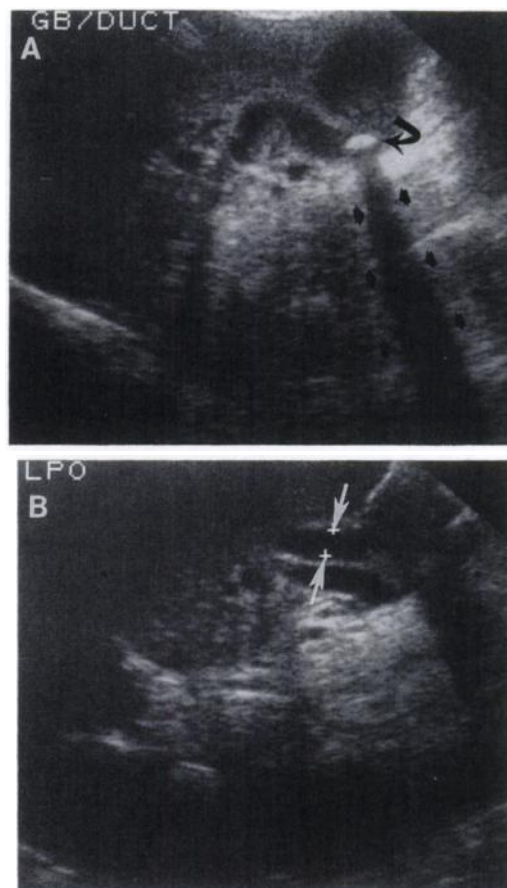
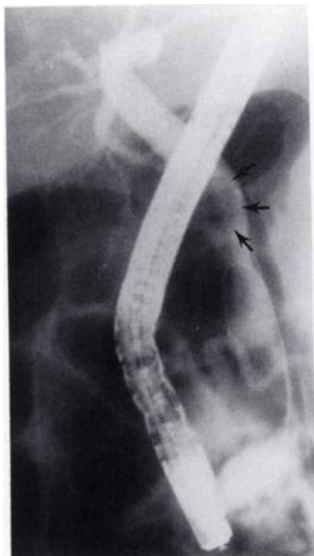


FIGURE 2. (A) Sonogram of the right upper quadrant. The gallbladder contains low-level echos indicating sludge. There is also an echogenic focus (curved arrow) with shadowing (small arrows), representative of a large gallstone. (B) Ultrasound view of the common bile duct (arrows) which is dilated and measures 9 mm. (Published with permission from *Clin Nucl Med* 1985; 10:264.)

Over the following days, the patient's bilirubin gradually dropped and her jaundice resolved. It was felt that her common bile duct stone had passed. However, the patient continued to have occult gastrointestinal bleeding similar to that before the ERCP, though the ERCP had not shown any evidence of an upper gastrointestinal bleeding site. The next step was to evaluate the lower gastrointestinal tract for bleeding and she was scheduled to have a barium enema. However, on the day that this was to be performed, the patient had a seizure and then a cardiopulmonary arrest. She was resuscitated, intubated, and transferred to the medical intensive care unit (MICU). The events that followed demonstrated that she had suffered a non-hemorrhagic stroke (noted on computed tomography) which probably caused the seizure. She also had suffered an acute anteroseptal myocardial infarction, although it was uncertain whether the myocardial infarction preceded the stroke. In the MICU, she was in cardiogenic shock and required pressors to maintain an adequate blood pressure. Following this event, she was unresponsive and believed to

FIGURE 3. This fluoroscopic image was obtained during ERCP. The endoscope is seen in the duodenum with a catheter inserted through the ampulla of Vater into the common bile duct. Contrast media infused into the common bile duct demonstrates dilation and a filling defect (arrows) in the mid-portion of the duct representative of a calculus. Contrast also fills the intrahepatic biliary ducts which appear normal. (Published with permission from *Clin Nucl Med* 1985;10:264.)



have sustained significant cerebral damage. She still had intermittent low-grade fevers and a repeat ultrasound was performed to visualize the biliary system. This demonstrated a single stone in the gallbladder but the ductal dilatation that had been seen on the previous ultrasound was now resolved with a common duct diameter of 4 mm, thus confirming that the common bile duct stone had passed (Fig. 4).

Despite initial improvement in the patient's cardiac status, approximately 1 wk after the first cardiac arrest, she arrested again. Attempts at resuscitation were unsuccessful and the patient died.

DISCUSSION

This case demonstrates the utility of hepatobiliary imaging in cases of obstructive jaundice due to common bile duct stones. In such cases, decisions regarding treatment can be simplified once common bile duct obstruction is known. With the advent of ERCP, choledocholithiasis can now be treated without surgery. This is especially important in patients such as the one presented here where the risks of surgery are great.

Most referring clinicians may not be aware of the use of hepatobiliary imaging for obstructive jaundice. Scanning of the biliary tree with an imino diacetic acid (IDA) compound such as DISIDA allows accurate detection of obstruction of the common bile duct. It also can distinguish obstruction of the cystic duct, as is seen in acute cholecystitis, from obstruction of the main bile duct. Studies such as ultrasound and CT scans may detect dilation of the biliary tree, however, they are often less sensitive in acute biliary obstruction before the bile duct dilates. Since both acute cholecystitis and biliary obstruction may cause similar acute abdominal pain, a biliary scan appears to be the diagnostic procedure of choice in a patient suspected of biliary colic. In such patients, it may be appropriate to proceed from a biliary scan suggesting acute biliary ob-

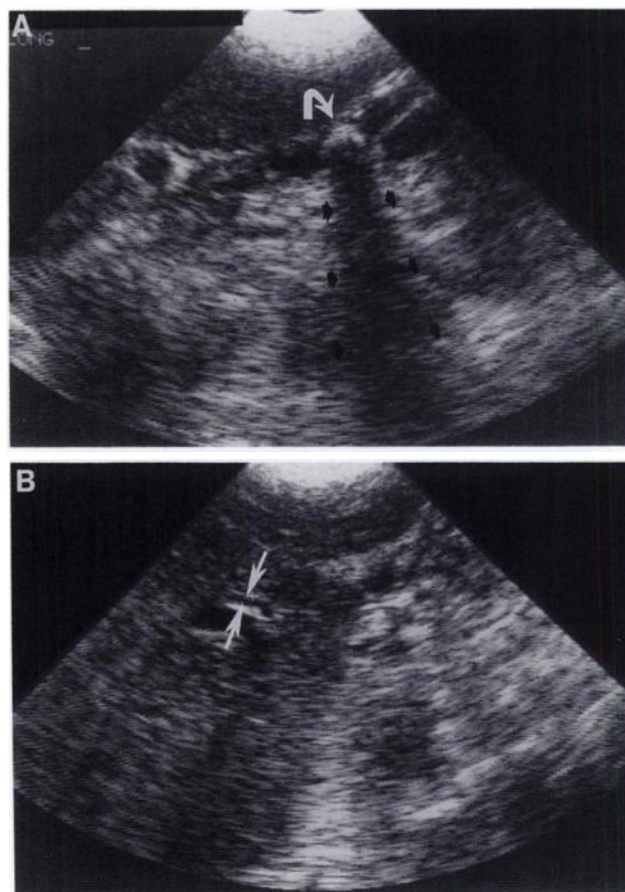


FIGURE 4. An ultrasound performed several days following ERCP. (A) An echogenic structure (curved arrow) in the gallbladder with shadowing is consistent with a gallstone. (B) The common bile duct (arrows) which measures 4 mm is a significant change from the image in Figure 2B performed before ERCP, and indicates that the common bile duct is no longer obstructed.

struction to diagnostic and therapeutic ERCP rather than continuing the noninvasive diagnostic evaluation.

Common bile duct obstruction causes the characteristic "liver scan appearance" with hepatobiliary imaging: There is prompt and avid uptake of the tracer by the liver but no excretion into the biliary system or bowel on either early or delayed images, presumably due to elevated biliary pressure that prevents the flow of bile from the hepatocytes into the biliary ducts (1,2). In cases with common hepatic duct distension, a photopenic defect may be seen in the porta hepatis. The "liver scan appearance" is common with most forms of CBD obstruction, such as choledocholithiasis, primary or secondary tumors of the common bile duct (pancreatic carcinoma, cholangio carcinoma, ampullary carcinoma, metastatic tumors, etc.) and pancreatitis, but occasionally can be seen in cases of sepsis, peritonitis, hepatocellular disease, portal vein thrombosis, biliary atresia, and Dubin-Johnson syndrome (3-6). The majority of cases are due to choledocholithiasis. It should be noted that the predictive value of the "liver scan appearance" has been shown to drop in scans performed with p-isopro-

pyl imino diacetic acid (PIPIDA) when the bilirubin levels rise above 10 mg/dl (3).

To understand how biliary scanning may be helpful in the management of gallstone obstruction of the common bile duct, we will review some important aspects of the pathophysiology of common duct stones and their non-operative management. Over half a million cholecystectomies are performed annually in the United States, and about 15% of these patients have a common duct stone (7). The first cholecystectomy was performed about 100 years ago when diagnosis of common duct stones was based upon clinical symptoms and findings. Now we have a great array of diagnostic techniques, including oral cholangiogram, ultrasound, CT scan, hepatobiliary imaging, transhepatic cholangiogram, and ERCP. The therapeutic options for management of common duct stones now include, not only surgery, but also percutaneous manipulations through the liver and retrograde endoscopic manipulations of the biliary tree. These may be supplemented with agents that can dissolve cholesterol gallstones or mechanical techniques such as extracorporeal shock wave lithotripsy (ESWL) or pulsed laser to fragment stones permitting easier extraction.

There are three types of gallstones and the composition of the stones affects their diagnosis and management. The most common gallstone in the gallbladder is the cholesterol stone. Eighty-five percent of cholesterol stones are radiolucent, but calcium salts make the stone radio-dense in the other 15% (8). The two other types of stones are composed primarily of calcium bilirubinate, which in its purest form does not contain enough calcium by weight to make it radio-dense on a plain abdominal film. Calcium bilirubinate stones which form in the gallbladder, especially in older patients, are hard crystal in type and contain an increased amount of calcium salts such as a calcium carbonate; in about 85% of patients these stones are, therefore, radio-dense. These pigment stones are referred to as "black stones."

In contrast to the black stones that develop within the gallbladder, softer "brown" pigment stones form within the bile duct. These stones contain fewer calcium salts and are rarely radio-dense. The brown stones contain fatty acids such as propionic acid not found in black stones. Most brown stones develop after removal of the gallbladder, but similar stones have been seen in patients living in the Orient and are associated with a dilated biliary tree. These patients may not have stones within the gallbladder. Patients living in more industrialized urban societies in Europe and North America rarely have brown stones in the bile duct if the gallbladder is intact (9).

Most bile duct stones found at the time of cholecystectomy in the United States are of the same composition as the stones found within the gallbladder (9). That is, they are either cholesterol stones or, less commonly, black stones. For up to a year following cholecystectomy, most stones found within the bile duct will be cholesterol or

black stones, suggesting that these residual stones passed from the gallbladder and were not detected at the time of surgery. In contrast, patients found to have common duct stones more than a year after cholecystectomy will usually have brown stones within the duct, consistent with a recurrent gallstone. Since most stones in the common duct will either be cholesterol or brown stones, they are usually radiolucent. Examination of bile may be helpful since cholesterol stones are associated with typical cholesterol crystals and amorphous calcium bilirubinate may be seen with pigment stones. During ERCP and attempted stone extraction, samples of bile or bits of crystals may be obtained and examined microscopically to help distinguish pigment from cholesterol stones. Distinction of cholesterol and pigment stones is important if solvents to dissolve nonextractable cholesterol stones endoscopically are used.

The frequency of common duct stones found at cholecystectomy varies with the age of the patient. About 5% of patients under 60 yr of age who undergo cholecystectomy have common duct stones, whereas a third of those over 60 may have common duct stones (10). In some surgical series in which the common duct was explored for suspected stones, duct stones were found in only 60% of patients. Patients in whom no stones were found may have passed them into the intestine. Alternatively, surgeons may miss 2%–15% of retained stones. Following cholecystectomy, patients with perampullary diverticuli have an increased risk of developing common duct stones (11). Sphincter of oddi pressure in patients with perampullary diverticular has been reported to be lower than normal and there is an increased risk of such patients having infected bile. It is possible that bacteria within the biliary tree contributes to the development of brown stones.

About 90% of common duct stones may be removed at the time of therapeutic retrograde cholangiography (12). Endoscopic sphincterotomy permits introduction of balloon-tip catheters and wire baskets into the biliary tree and stones that are small enough are extracted intact. Stones greater than 1.5 cm in diameter become more difficult to remove, and those over 2 cm frequently require fragmentation or dissolution before removal. Dissolution can be attempted with perfusion of the bile duct with mono-octan-ol or perhaps with methyl *tert*-butyl ether, but these solvents do not dissolve brown stones and the side-effects associated with them have limited their use (12).

Fragmentation of large common duct stones can be performed with strong wire baskets. Some centers are experimenting with pulsed-laser fragmentation or electrohydraulic fragmentation of retained stones, two very promising techniques (13). The use of extracorporeal shock wave lithotripsy (ESWL) to fragment large common duct stones has been reported. A study by Sauerbruch et al. included 113 patients who had endoscopic sphincterotomy and placement of a biliary catheter either percutaneously or transnasally (a nasobiliary catheter) for introduction of radio-contrast. Shock waves were then directed fluoroscop-

ically toward the bile duct gallstones. Ninety percent of stones were fragmented, and in 86% of patients, complete clearance of the bile duct was possible. Only patients who had failed stone removal at ERCP were subjected to ESWL. Since 90% of stones may be removed at the time of endoscopic sphincterotomy, approximately 98% of all patients with common duct stones may have nonoperative clearance of their stones.

It is now widely accepted that post-cholecystectomy patients with common duct stones should have these removed endoscopically (15). In Europe and England about 50% of patients with intact gallbladders are having common duct stones removed endoscopically. Generally, these patients tend to be older and poorer operative risks than the patients subjected to surgery, as was the case with the patient presented here. Hospital stay following endoscopic sphincterotomy and common duct stone removal averages about 2 days in contrast to about 7 days for the operative common duct exploration. Mortality for endoscopic common duct stone removal is less than 1%, even in reports involving mainly older and poor operative risk patients (12). If there are stones remaining in the gallbladder following endoscopic sphincterotomy, about 15% of such patients will develop cholecystitis over the next few years and require operative cholecystectomy. Some of the gallstones may pass without causing significant symptoms, but most of the patients continue to have asymptomatic gallstones. The presence of cholangitis or acute gallstone pancreatitis is believed by many to be an indication for endoscopic sphincterotomy and nonoperative management. We generally treat the infection or pancreatitis to stabilize the patient prior to endoscopic management.

In summary it is accepted widely that poor operative risk patients with common duct stones should be treated with endoscopic sphincterotomy and stone extraction. If a patient has an intact gallbladder and is a good operative risk, there is controversy as to whether endoscopic techniques should be used. Some studies have suggested that endoscopic removal of common duct stones be performed and then operative removal of the gallbladder. The introduction of laparoscopic cholecystectomy has further complicated this decision. We have treated several patients with common duct stones immediately prior to elective laparoscopic cholecystectomy. This combination has allowed rapid recovery of the patients and may prove appropriate for the younger good operative risk patients.

If a patient has acute cholecystitis, surgical therapy will probably be most appropriate. These are the very patients who can be diagnosed most accurately with hepatobiliary imaging, emphasizing the interaction between the newer diagnostic and therapeutic techniques.

Hepatobiliary imaging may play a significant role in evaluation of biliary colic. Further work needs to be done to determine the sensitivity and specificity of this technique in a patient with an acutely obstructed duct. However, the case presented here and data gathered from the literature appears to provide evidence for its usefulness in the appropriate setting.

REFERENCES

1. Weissmann HS, Rosenblatt RR, Sugarman LA, Badia JD, Freeman LM. Early diagnosis of acute common bile duct obstruction by Tc-99m-IDA (iminodiacetic acid) cholescintigraphy [Abstract] *J Nucl Med* 1980;21:P41.
2. Noel AW, Velchik MG, Alavi A. The "liver scan" appearance in cholescintigraphy, a sign of complete bile duct obstruction. *Clin Nucl Med* 1985;10:264-269.
3. Egbert RN, Braunstein P, Lyon KP, Miller DR. Total bile duct obstruction. Prompt diagnosis by hepatobiliary imaging. *Arch Surg* 1983;118:709-712.
4. Hughes KS, Marrangoni AG, Turbner E. Etiology of the obstructive pattern in hepatobiliary imaging. *Clin Nucl Med* 1984;9:222-226.
5. Bar-Meir S, Baron J, Seligson U, Gottesfeld F, Levy R, Gilat T. ^{99m}Tc-HIDA cholescintigraphy in Dubin-Johnson and Rotor syndromes. *Radiology* 1982;142:743-746.
6. Klingensmith WC III, Kuni CC, Fritzberg AR. Cholescintigraphy in extrahepatic biliary obstruction. *AJR* 1982;139:65-70.
7. Mullen JL, Rosato EF, Ipsen J, Rosato FE. Gallstone characteristics in the diagnosis of choledocholithiasis. *Ann Surg* 1972;176:718-720.
8. Trotman BW, Petrella EJ, Soloway RD, Sanchez HM, Morris TA III, Miller WT. Evaluation of radiography lucency or opaqueness of gallstones as a means of identifying cholesterol or pigment stones. *Gastroenterology* 1975;68:1563-1566.
9. Malet PF, Dabiezies MA, Huang G, Long WB, Gadacz TR, Soloway RD. Quantitative infrared spectroscopy of common duct gallstones. *Gastroenterology* 1988;94:1217-1221.
10. Barie PS, Jacobson IM. *Gallbladder disease*. In: Zakim D, Boyer T, eds. *Hepatology, a textbook of liver disease*, second edition. Philadelphia: WB Saunders Co.; 1990:1516.
11. Løtveit T, Foss OP, Osnes M. Biliary pigment and cholesterol calculi in patients with and without juxtaapillary duodenal diverticuli. *Scand J Gastro* 1981;16:241-244.
12. Long WB. Endoscopically assisted diagnosis, removal and dissolution of choledocholithiasis. In: Cohen S, Soloway RD, eds. *Contemporary issues in gastroenterology—gallstones*. New York: Churchill Livingstone; 1985:4:259-265.
13. Cotton PB, Kozarek RA, Schapiro RH, et al. Endoscopic laser lithotripsy of large bile duct stones. *Gastroenterology* 1990;99:1128-1133.
14. Sauerbruch T, Stern M. Fragmentation of bile duct stones by extracorporeal shock waves, a new approach to biliary calculi after failure of routine endoscopic measures. *Gastroenterology* 1989;96:146-152.
15. Summerfield JA. Biliary obstruction is best managed by endoscopists. *Gut* 1988;29:741-745.