

Choledocholithiasis Mimicking Sphincter of Oddi Dysfunction

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Case Report

A 36-year-old woman without any significant medical or surgical history developed biliary-type right upper quadrant, epigastric abdominal pain, and liver function tests (LFTs) revealed alanine aminotransferase and aspartate aminotransferase elevations of three times the normal levels, for which she was admitted to our hospital for further care and management. The patient had had five similar episodes over the previous 2 years and had been treated in various emergency departments, never having been admitted prior to this episode.

The patient presented to our emergency department in January 2006, with complaints of nausea and abdominal pain. Abdominal computed tomography (CT) scan was unremarkable (Figure 1). Ultrasound of the abdominal right upper quadrant revealed a dilated common bile duct (CBD) measuring approximately 10 mm, but without visible stones or intrahepatic biliary duct dilation (Figure 2). In addition, no filling defect was seen within the ductal system. Gallbladder lumen appeared normal in size, and there was no gallbladder wall thickening, pericholecystic fluid, sludge, or cholelithiasis evident.

Because biliary-type abdominal pain is commonly associated with dilated CBD on ultrasound and abnormal LFTs, the patient was clinically diagnosed with sphincter of Oddi dysfunction (SOD) and scheduled to undergo endoscopic retrograde cholangiopancreatography (ERCP) with sphincter of Oddi manometry (SOM). ERCP revealed extensive amounts of biliary sludge and numerous stones stacked up in the CBD, although the gallbladder itself did not contain stones (Figures 3 and 4).

Discussion

First described less than three decades ago, SOD is a clinical syndrome best categorized by the Milwaukee



Figure 1. Normal computed tomography scan of the patient's abdomen at presentation. Arrows point to the common bile duct.

Classification system based on laboratory, clinical, and radiologic features in patients with recurrent abdominal pain, biliary pain, elevated aminotransferase levels, and dilated CBD on imaging studies, all of which are criteria that have been evaluated in multiple studies.¹⁻⁵

SOD is a benign noncalculous obstruction of bile or pancreatic drainage at the level of the sphincter of Oddi. This condition is clinically associated with either biliary pain or idiopathic pancreatitis, depending on the portion of the affected sphincter. Patients with suspected SOD can be subdivided into type I, II, or III SOD, depending on associated clinical evidence. Multiple noninvasive tests have been utilized to aid in diagnosis but have been complicated by poor sensitivity and specificity. Until several years ago, the gold standard for studying and treating this pathologic condition was ERCP. Currently, SOM is the gold standard for confirming diagnosis, although questions remain regarding its sensitivity and specificity. Sphincterotomy of the affected portion of the sphincter is

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Figure 2. Ultrasound showing dilated common bile duct without any obvious stones.

the treatment of choice and has been shown to be effective for the palliation of symptoms in type I, and occasionally in type II, SOD.

The differential diagnosis in our patient included SOD, papillary stenosis, ampullary lesions, and choledocholithiasis. What makes this case particularly interesting is that conventional imaging studies such as ultrasound and CT scan of the abdomen were negative, except for a mildly dilated CBD. This case reinforces that choledocholithiasis and cholelithiasis are the most important differential diagnoses for right upper quadrant pain with elevated liver enzyme levels, and emphasizes that choledocholithiasis should always be taken into consideration, even if the usual imaging studies (ie, sonography and CT scanning) are negative.

ERCP is highly accurate in diagnosing choledocholithiasis, but it is the most invasive of the available methods. Complications of ERCP include a mortality rate of 0.1–1.3% and a morbidity rate of 5–19%,⁶ with a 3–30% incidence of post-ERCP pancreatitis. Less invasive methods for the diagnosis of choledocholithiasis include ultrasonography (US), magnetic resonance cholangiopancreatography (MRCP), CT, and endoscopic ultrasonography (EUS). In a study comparing the results of US, MRCP, and ERCP in 83 patients who had a suspicion of biliary disease, the sensitivity and specificity for ERCP in detecting stones were 98% and 100%, respectively, which were better than any other imaging modality. The sensitivity and specificity for US were 71% and 97.2%,⁷ respectively, whereas the sensitivity and specificity for MRCP were 97.4% and 97.2%, respectively, in the diag-

nosis of choledocholithiasis (n=38) for both procedures. In contrast, CT has a high specificity (94%), but low sensitivity (35–85%), due to its inability to detect noncalcified stones.⁸ EUS is another extremely accurate test in the diagnosis of choledocholithiasis, with a risk of complications similar to that of upper gastrointestinal endoscopy. In a retrospective chart review of 30 patients who underwent EUS, with or without ERCP, for suspected choledocholithiasis, ERCP did not identify any new cases of choledocholithiasis following a negative EUS in 21 patients.⁶ In a separate prospective study examining 32 patients with suspected biliary pancreatitis, the sensitivity of US, CT, MRCP, and ERCP for identifying choledocholithiasis was 20%, 40%, 80%, and 90%, respectively.⁹

Nevertheless, choledocholithiasis still occurs in 10% of patients presenting with cholecystectomy.¹⁰ Effective treatment includes both repeat cholecystectomy and clearance of the entire biliary tract. There is evidence that elective and emergent clearance of the bile duct by ERCP has advantages over open bile duct exploration, which is usually combined with open cholecystectomy. In the elective setting, ERCP therapy and laparoscopic bile duct clearance are equivalent for the majority of stones. In a research study conducted from September 21, 1989, to December 31, 2001, 3,580 patients presented with symptomatic biliary tract disease and laparoscopic cholecystectomy was performed in 3,527 patients (99.5%).¹⁰ Of the 3,417 laparoscopic cholangiograms, 344 (9.7%) were abnormal. Laparoscopic common bile duct exploration successfully cleared the duct in 317 of the 344 patients with abnormal cholangiograms (92.2%).

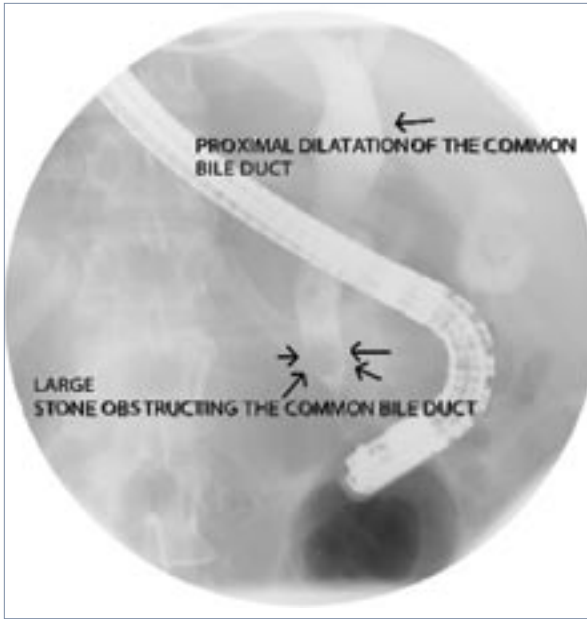


Figure 3. Endoscopic retrograde cholangiopancreatography showing stones in the common bile duct.



Figure 4. Endoscopic retrograde cholangiopancreatography showing dilated common bile duct filled with stones.

Conclusion

When possible, elective management of choledocholithiasis via the endoscopic approach (ie, ERCP) should be offered. The role for ERCP in the management of acute calculous bile duct disease remains central and optimal. Following successful endoscopic bile duct clearance, most patients should undergo laparoscopic cholecystectomy as early as possible to reduce the risk of a further biliary event or to treat any cholecystitis that may be present. However, for the small group of elderly or frail patients who are considered a high surgical risk for laparoscopic cholecystectomy, judicious conservative management of the in situ gallbladder is justified and acceptable.

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Review

Sphincter of Oddi Dysfunction: Stones, Spasm, or Stenosis?

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The case study by Hadique and colleagues describes a 36-year-old woman who presented with biliary colic, aminotransferase elevations at three times the normal levels, and a 10-mm bile duct on ultrasound and computed tomography (CT) examination.¹ There was no evidence of either gallstones or common bile duct stones, and a tentative diagnosis of sphincter of Oddi dysfunction (SOD) was made. Endoscopic retrograde cholangiopancreatography (ERCP) revealed choledocholithiasis. Whether the patient underwent sphincterotomy, balloon dilation of the sphincter with stone extraction, perampullary botulinum toxin injection, or laparoscopic or open common bile duct exploration at the time of cholecystectomy was not reported.

There are clinical clues in a patient such as this that point to stones, spasm, or papillary stenosis as the etiology of the symptoms. A single episode of biliary colic, in conjunction with the elevation of liver function tests with or without hyperamylasemia or dilated biliary tree on imaging, signifies stone disease until proven otherwise. The authors do not specify the timing of ERCP relative to clinical presentation or whether the patient maintained symptoms and laboratory abnormalities prior to manipulation. This information is crucial, even before considering a diagnosis of SOD. Just as continuous, unrelenting epigastric or right upper quadrant pain is highly unlikely to be related to sphincter spasm or even stenosis, a single pain episode in this setting simply demonstrates that the diagnostic technology employed is imperfect. In fact, common bile duct stones are seen on ultrasound in only one half to two thirds of patients with choledocholithiasis and approximately one third of patients studied with CT.²⁻⁴ Both endoscopic ultrasound (EUS) and magnetic resonance cholangiopancreatography (MRCP) would have confirmed the diagnosis in this patient, although their application is contingent upon the relative skill sets of surgeons and therapeutic endoscopists in an institution.²⁻⁵ In hospitals in which surgeons routinely perform

transcystic laparoscopic common bile duct exploration, an EUS or MRCP would have resulted in a laparoscopic approach in this 36-year-old patient in whom unnecessary sphincter manipulation could potentially result in acute or possible long-term complications. In institutions without this expertise, ERCP can be both diagnostic and therapeutic for common bile duct stones, often reserving laparoscopic cholecystectomy for patients with concomitant gallstones or imaging evidence of concomitant gallbladder disease.⁶⁻¹⁰

SOD is a multifactorial disease with complex symptoms. Purported to cause recurrent biliary colic, as well as acute and relapsing pancreatitis, SOD is diagnosed depending upon both definition of the disorder and, to some extent, geography.¹¹⁻¹³ For instance, many of our colleagues in Europe and Asia are skeptics of SOD, defining type I SOD (ie, dilated ducts, delayed drainage, 1.5–2-fold elevation in aminotransferase levels on at least 2 occasions in a patient with recurrent biliary colic) as simply a consequence of chronic gravel or sludge passage with subsequent papillary stenosis. Type II SOD (ie, intermittent pain plus either ductal dilation or liver function abnormalities, since delayed ductal drainage has recently been dropped as a diagnostic criterion) is purported to result from gravel passage without structural stenosis. Type III SOD (ie, intense pain) is a functional disorder that covers a spectrum of problems including gastroparesis, irritable bowel syndrome, nonulcer dyspepsia, and sphincter spasm.¹⁴ Moreover, previous studies have documented that these patients demonstrate visceral hyperalgesia and pain that reproduces the symptoms simply by balloon inflation in the duodenum.

Papers written by Sherman and Lehman,¹⁵ Baillie,¹³ and Petersen^{16,17} have provided vital reviews of SOD. This commentary will synopsise only a few salient points.

1. Most patients do not fit within standard SOD categories.¹⁴ For example, physicians may not be certain whether a patient with a 9-mm bile duct, pain, and aminotransferase elevations of 1.4 times the normal level should be classified as type I SOD, type II SOD, or neither. Likewise, it is unclear how a patient with a 15-mm common bile duct, periodic severe pain superimposed upon a background ache, but normal aminotransferase levels should be classified. Similarly, in a patient with an intact gallbladder and no imaging evidence of stones, recurrent biliary colic and 1.5-fold transaminase elevation could be a function of gravel passage or sphincter dysfunction. The diagnosis changes if the patient has a 10-mm or 3-mm common bile duct.

2. Although a variety of noninvasive studies have been advocated in the diagnosis of SOD, including morphine-

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prostigmin test, biliary scintigraphy transit studies, cholecystokinin ultrasound, secretin EUS or MRCP, and diagnostic trials of ampulla of Vater botulinum toxin injections, SOD manometry remains the gold standard of diagnosis.^{13,15,17-22}

3. Although moderated by the placement of small-caliber stents in the pancreatic duct (whether or not sphincter pressure is abnormal and whether or not a biliary, pancreatic, or dual sphincterotomy is performed), post-ERCP pancreatitis occurs at least 2–5 times more often in patients with SOD, as opposed to patients who undergo ERCP for choledocholithiasis.^{15,22} Moreover, the sphincter is a dynamic structure, and our group, as well as others, has studied patients with vastly discordant sphincter pressures when undertaking SOD manometry on different days.

4. The main conundrum does not lie with type I SOD or even true type II patients, almost all of whom have their biliary sphincters ablated without undertaking manometry prior to their referral to a tertiary pancreaticobiliary referral center—it lies with the type III SOD patients with only pain, or pain plus aminotransferases or amylase elevated 10% on one or more occasion. These patients have a normal pancreaticobiliary tree on CT and MRCP, have often undergone a negative EUS or one that is interpreted as having several diagnostic criteria for chronic pancreatitis, and may or may not have an intact gallbladder. This latter patient group, previously defined by Cotton as the group that needs ERCP the least and the group most likely to be injured by it, has, at best, a 50% chance of pain improvement whether one ablates one sphincter or two.^{16,17,23-25} This finding suggests that our gold standard is, at best, a gilded one. Most surgeons do not remove gallbladders for chronic right upper quadrant pain unless there is imaging or scintigraphic evidence of stones or dyskinesia and pain reproduction with scintigraphy.^{26,27} We, as endoscopists, have not developed that same level of discipline, and many patients are subjected to ERCP with little to recommend it other than the laments of the patients. This is particularly ironic as the risk of ERCP in this subgroup may be a log factor higher than an empiric cholecystectomy. Hopefully, an upcoming multicenter controlled trial investigating sphincterotomy versus sham-sphincterotomy for type III SOD patients will answer questions about the incidence of SOD in these patients, their psychological profile, the efficacy of single or dual sphincterotomy in the short term and long term, procedural complications, and their quality-of-life scores pretreatment and posttreatment.¹³ Until that time, diagnostic ERCP in these patients is best limited to pancreaticobiliary referral centers.

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