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Sphincter of Oddi: ERCP Plus Sphincterotomy – Yes or No



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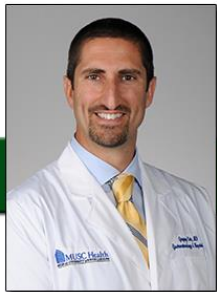
Note: For debate purposes, the pro and con positions for patient management will be taken by the invited authors. However, actual decisions regarding patient care must involve discussion of the risks and benefits of each treatment considered.

Case Presentation – Case developed by Ihab I. El Hajj, MD, MPH, Indiana University, Indianapolis, IN. A 57-year-old Caucasian female with history of smoking and COPD, was in her usual state of health until two years ago, when she experienced recurrent “attacks” of right upper quadrant pain, nausea and occasional vomiting, suggestive of biliary colic. The patient was evaluated by her primary care physician and initial work-up, which included basic blood work, liver chemistries and transabdominal ultrasound, were negative. The patient responded partially to prn Zofran and omeprazole 40 mg once then twice daily. With the persistence of her symptoms, the patient was referred to a gastroenterologist. Esophagogastroduodenoscopy (EGD) with gastric biopsies revealed chronic inactive gastritis without *Helicobacter pylori*. HIDA scan suggested biliary dyskinesia with an ejection fraction of 22%. An elective laparoscopic cholecystectomy was performed. An intra-operative cholangiogram showed no filling defect in the common bile duct (CBD) and pathology demonstrated chronic cholecystitis with no gallstones.

The patient was symptom-free for six months after surgery. She subsequently developed vague upper abdominal pain, intermittent nausea and irregular bowel movements. Labs, colonoscopy and repeat EGD were

normal. The patient was treated for suspected irritable bowel syndrome. She failed several medications including hyoscyamine, dicyclomine, amitriptyline, sucralfate, and GI cocktail. Over the following months, the patient noted recurrent “attacks” similar to the ones she had before surgery. She was evaluated in the emergency department on several occasions. Liver chemistries and pancreas enzymes were normal during the “attacks.” The patient never received narcotics. Computed tomography and magnetic resonance imaging with cholangiopancreatography were normal including the pancreas and bile duct that measured a maximum of 4.4 mm in diameter without a filling defect. Upper endoscopic ultrasound (EUS) showed a 3.9 mm CBD with no strictures, no microlithiasis and no endosonographic evidence of chronic pancreatitis. Type III sphincter of Oddi dysfunction-biliary type was suspected. The patient was evaluated in the pancreaticobiliary clinic and the potential need for endoscopic retrograde cholangiopancreatography (ERCP) with sphincter of Oddi manometry (SOM) and possible sphincterotomy was discussed. Procedural risks were also thoroughly discussed.

What would you advise her to do?



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PRO

ERCP plus sphincterotomy should be performed next in this patient.

The EPISOD study refuted decades of practice and a generally pro-sphincterotomy literature, albeit consisting of small trials without concealed allocation or retrospective cohorts of variable quality.¹ The EPISOD study showed no incremental benefit of sphincterotomy over diagnostic ERCP for patients with post-cholecystectomy abdominal pain meeting the Rome III definition of sphincter of Oddi dysfunction (SOD) type III. SOD skeptics claim they could have predicted this outcome, referencing studies documenting the high prevalence of duodenal visceral hyperalgesia,^{2,3} psychosomatic disorders,³⁻⁷ and central sensitization⁸ in this population. Why should sphincterotomy alleviate suffering when there are so many other potential contributing factors? The fundamental reason is that there is an underrepresented group whose sole etiology for their pain resides in the sphincter of Oddi; screening tools cannot reliably define this population. EPISOD enrollees included patients with the aforementioned confounding factors, so it isn't surprising that sphincterotomy failed to show a benefit.

Given the difficulty diagnosing chronic pancreatitis in the absence of end-organ damage, some patients with suspected SOD III might have early chronic pancreatitis and stand little chance to benefit from sphincterotomy. Patients with moderate to severe depression, as defined by the Beck Depression Inventory, were excluded. The accuracy of this screening instrument is fair (AUC < 0.80) among individuals with other chronic pain disorders, so some patients with un- or undertreated depressive disorders may have been randomized.⁹ Similarly, the majority of individuals undergoing SOM meet criteria for somatization and hypochondriasis.¹⁰ Finally, a lot of these patients suffer for many years prior to referral for ERCP; a substantial component of central sensitization has probably evolved in many of these subjects. This may have been particularly applicable in the EPISOD trial, since baseline symptoms had to reach a minimum level of severity to warrant inclusion. In these patients, sphincterotomy may be futile or performed too late in the disease course. These confounding factors may not have been adequately ruled out prior to randomization. Given the staggering statistics, patients with post-cholecystectomy pain must undergo formal psychological assessment before proceeding with ERCP. If psychosomatic disorders are absent, we may be one step closer to identifying a population who may benefit from sphincterotomy.

Both the clinical history and SOM poorly predict which patients with pain will benefit from sphincterotomy. Perhaps this most impacts the investigators who participated in the EPISOD trial, since we continue to receive

referrals to consider ERCP for patients with refractory pain. EPISOD has incriminated those of us who have performed ERCP for post-cholecystectomy biliary colic, confirming that our threshold for performing such an invasive intervention was not high enough. While many patients should now be spared potentially harmful and ineffective therapy, we are no closer to understanding the pathophysiology of and ideal treatment for these patients. We must aggressively identify the small subgroup of patients with post-cholecystectomy pain that may benefit from sphincterotomy, as it should be a rare condition. With such a personalized approach to “suspected SOD,” we will finally define patients who will benefit from endoscopic therapy. Until then, sphincterotomy remains a reasonable last resort for these desperate individuals after addressing the numerous confounders often present in this population.

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CON

ERCP plus sphincterotomy should not be performed next in this patient.

This patient is typical for one referred for consideration of ERCP with or without manometry and raises a number of important issues. Firstly, while cholecystectomy for biliary dyskinesia is now commonplace and almost a standard of practice, this too is a very controversial practice that has many similarities to the issue under discussion. The response rates for cholecystectomy are highly variable and do not correlate with the gallbladder ejection fraction.¹ Secondly, there is a high placebo response rate for most interventions for such patients and a six month improvement followed by recurrent pain is commonplace. Such placebo responses are typical in patients following sphincterotomy for SOD. Thirdly, the physician is to be congratulated for trying a number of medicines for functional gastrointestinal disease but a main question is whether the medication

trial, especially amitriptyline, was optimized. Lastly, the patient's vague upper abdominal pain associated with nausea and lower gastrointestinal symptoms (irregular bowel movements) strongly supports the referring physician's initial consideration of a functional etiology to account for her symptoms. Furthermore, her liver tests and bile duct caliber were normal even during an attack. She complains of "attacks" similar to ones prior to her surgery which underscore the fact that the cholecystectomy was neither effective nor warranted. Her current symptoms do not represent "post-cholecystectomy" pain, but rather the same nonbiliary pain she had prior to cholecystectomy. Must we now invoke the biliary sphincter as the cause given the similarity to her prior symptoms?

SOD is classified into three types.² Type I is characteristically diagnosed by a dilated biliary tree and abnormal liver tests. In this situation, a sphincter abnormality such as papillary stenosis is present and biliary sphincterotomy is highly effective. The liver tests should be at least 2x the upper limit of normal given the prevalence of fatty liver disease. Type II SOD is diagnosed when the patient has biliary pain accompanied by either bile duct dilation or elevation in liver chemistry tests. Biliary sphincterotomy in these patients is much less effective and if manometry is performed, will demonstrate elevation in sphincter pressure in 50% or fewer of patients.^{2,3} Type III SOD presents with normal liver tests and no biliary dilatation, as was found in our patient even during her attacks. One must ask why we should implicate the biliary system as a cause of her symptoms given the normal liver tests and no biliary dilatation? Is it just because her pain is in the right upper quadrant and she did not respond to cholecystectomy?

Are there any data to guide us in the management of such patients? There have been a number of studies, all unblinded and nonrandomized, which have suggested efficacy. However, a recently published randomized controlled trial of 214 patients, the EPISOD trial, clarified this issue.⁴ In this study, patients with biliary Type III SOD were randomized either to sham biliary sphincterotomy, biliary sphincterotomy or dual sphincterotomy (biliary and pancreatic) depending on the results of manometry. At one year follow-up, patients who did not undergo sphincterotomy had a statistically significantly better outcome (37%) than those who received either biliary or dual (biliary, pancreatic) sphincterotomy (23%). While this trial had limitations, it generated the best evidence to date that sphincterotomy in this situation is not effective and in fact was less effective than sham.

Why not just "cut" everyone and see if they improve, which is the practice adopted by many? The primary reason is the lack of efficacy and notable risk of adverse events.⁵ While there has been a significant reduction of post-ERCP pancreatitis with the use of rectal indomethacin and pancreatic ductal stenting, the risk of pancreatitis, even severe, is still notable, especially for a therapy that is ineffective. I instead favor conservative management, not only within academic centers, but also within the community where experience is variable and adverse events more likely. My esteemed colleague suggests the use of sphincterotomy as a last resort for desperate patients. I instead advocate that we not offer ineffective and risky interventions regardless of the patients or physicians level of desperation. Desperation does not justify intervention.

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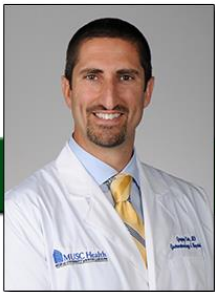


MODERATOR

Paul R. Tarnasky, MD
Methodist Dallas Medical Center

After reviewing the pro and con expert arguments, Moderator Paul Tarnasky, MD, poses the following questions to Dr. Cote:

1. What specific clinical criteria (e.g. pain characteristics, laboratory testing, and/or imaging) do you consider to be most consistent with SOD and a prerequisite before pursuing endoscopic evaluation and therapy?
2. Does sphincter of Oddi motility testing need to be done in patients before considering endoscopic therapy for SOD?
3. What endoscopic therapies (e.g. biliary and/or pancreatic sphincterotomy, Botulinum toxin injection) do you recommend and what factors go into deciding which are indicated in patients with suspected SOD?
4. Should an average volume ERCP endoscopist consider taking on the challenges associated with endoscopic evaluation and treatment of SOD?



PRO

Gregory A. Cote, MD, MS
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Dr. Cote responds:

Question 1

In my experience, intermittent and post-prandial pain that is short-lived (1-2 hours/episode) and not confounded by bowel or dyspeptic complaints is the single best predictor of response. Of course, reliable documentation of transient liver or pancreas chemistry elevation $> 2x$ upper limit of normal, with or without progressive duct dilation since cholecystectomy, increase my level of suspicion for sphincter of Oddi-related symptoms. Many of these patients have true sphincter of Oddi stenosis (e.g., elevation in basal sphincter pressure). Patients without laboratory or imaging abnormalities are more difficult to diagnose and the subgroup with an “Oddi problem” presumably have dyssynergic motility as opposed to true stenosis.

Since our patient has no objective abnormalities, I would favor additional medication trials such as calcium channel blockers, nitrates and serotonin-norepinephrine reuptake inhibitors, along with diet modifications (FODMAP). I always emphasize smoking cessation, given the known impact of smoking on pain modulation. In my experience, each trial is likely to fail and the patient will become increasingly anxious and therefore more

symptomatic. The lack of medication response typically leads to a very challenging patient interaction and I must question my prior conclusions. Am I sure the patient does not have an undiagnosed psychosomatic disorder? Is this pain truly disabling? Have I really tried every other alternative? If the symptoms are plausible, and the patient understands that the intervention is truly experimental and no longer supported by scientific evidence, then I would somewhat reluctantly proceed with ERCP.

Question 2

SOM is unreliable and the definition of dysfunction is oversimplified. Motility of the stomach, duodenum and sphincter of Oddi are highly intertwined.¹ However, sphincter of Oddi inhibition is independent of its surroundings, so dyssynergic motility of the gastro-duodenum and sphincter of Oddi is a much more plausible explanation for pain than elevation in basal sphincter pressure, as SOD is traditionally defined. The history of how SOD evolved to this simplistic definition is beyond the scope of this discussion, but elevation in basal pressure is more logical when considering a patient with true papillary stenosis (duct dilation) or recurrent acute/chronic pancreatitis; these entities have a distinct fibroinflammatory process involving the sphincter of Oddi, periampullary duodenum or both. On the other hand, patients with post-cholecystectomy pain and normal anatomy are more likely to have a true motility disorder. SOM is too crude a measure with the use further complicated by the uncertain intra- and inter-observer agreement. This is why currently available, noninvasive tests such as hepatobiliary scintigraphy perform so well for papillary stenosis and so poorly for patients classified as SOD Type III.² We need nociceptive biomarkers and better upper GI motility techniques—including the ability to monitor sphincter of Oddi function over 24 hours or longer—to more accurately select patients who respond to sphincterotomy.

Question 3

Current data do not support pancreatic sphincterotomy for recurrent acute pancreatitis and SOD III. The risk of post-ERCP pancreatitis, sphincter restenosis and perforation increase significantly when performing pancreatic sphincterotomy on a normal diameter pancreatic duct. If the physician and patient have already made the challenging decision to proceed with ERCP despite its inherent risk, a biliary sphincterotomy is reasonable since the incremental risk of this maneuver is small. I do not believe there are sufficient data to support botulinum toxin injection of the duodenal wall or major papilla at this time, although this is an intriguing concept since the risk would be minimal if cannulation can be avoided.

Question 4

No, particularly given that the adverse event and failure rates of ERCP are highest among individuals performing fewer than 115 ERCPs a year (roughly two per week). Physicians will have no sympathy from lawyers should a patient suffer an adverse event of ERCP when performed for unexplained abdominal pain, given its complex and multifactorial pathophysiology.

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MODERATOR

Paul R. Tarnasky, MD
Methodist Dallas Medical Center

Dr. Tarnasky poses the following questions to Dr. Wilcox:

1. Is there evidence to support the notion that SOD is not a specific disorder and instead more related to a generalized motility disorder?
2. What are the likely explanations and/or diagnoses for the symptoms in patients who are suspected to have but lack objective evidence for SOD?
3. Are non-endoscopic options for treatment of pain effective in patients who are considered as possibly having SOD?



CON

C. Mel Wilcox, MD, FASGE
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Dr. Wilcox responds:

Question 1

SOD is a symptom complex that has been associated with an abnormality identified by manometry. Given the fact that there is no correlation between an abnormal manometry and symptomatic response combined with the fact that response rates are generally low, this would suggest some other mechanism. A motility disorder or visceral hypersensitivity should be suspected. In an important study, Desautels et al¹ performed duodenal and rectal barostat studies in patients considered to have biliary SOD Type III as well as controls. They found that patients with Type III SOD had duodenal but not rectal hyperalgesia compared to the controls. Remarkably, duodenal balloon distention reproduced the patient's symptoms in all but one patient. Psychological testing showed high levels of somatization, obsessive compulsive behavior and anxiety. These data strongly support the notion that visceral hypersensitivity in the right psychological setting is etiologic.

Question 2

As noted above, many of us consider these patients to have functional abdominal pain, and the study by Desautels et al¹ supports this hypothesis. In addition, our patient under discussion had both upper and lower GI symptoms including nausea and diarrhea which would further suggest a motility disturbance such as irritable bowel syndrome. The fact that tricyclic antidepressants and antispasmodics can be effective in such patients lends additional support. In addition, many patients report symptom "improvement" yet on further close questioning remain symptomatic suggesting a chronic pain syndrome.²

Question 3

In patients presenting with a constellation of symptoms as in this patient, the diagnosis of a functional abdominal pain syndrome such as irritable bowel syndrome should be entertained and treated accordingly. Antispasmodic agents may be effective particularly when there is a spastic component to the pain. There is a wealth of data on the use of tricyclic antidepressants for irritable bowel syndrome and given the clinical overlap with SOD these medicines should be given and titrated to effect.³ Some patients will not respond to conservative measures and represent an even more difficult cohort to treat. These patients often suffer from psychological issues or more refractory symptoms whereby novel treatments, both medical and psychological may be warranted.⁴

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MODERATOR

Paul R. Tarnasky, MD
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Dr. Tarnasky concludes:

Post-cholecystectomy pain (PCP) remains a considerable challenge as illustrated by this patient, one that is rather typical of those often referred for management of potential SOD. Our experts have carefully navigated the salient clinical features as well as the more subtle aspects of this dilemma while stating their respective cases.

As reviewed by our experts, patients with PCP have historically been categorized as Types I, II or III for predicting the likelihood of sphincter dysfunction and a successful response to therapy. We now know that SOM is unreliable and not necessary for diagnosis of Type I, there is considerable overlap for SOM findings in Types II and III, and most importantly, the EPISOD trial appears to have eliminated the Type III subgroup.

One could conclude from EPISOD that “Type III SOD” does not exist because there was no difference in outcomes between those who did or did not undergo sphincterotomy. It should be noted, however, that the definition of success was strict, requiring a marked reduction in disability due to pain at both 9 and 12 months, no reintervention and no narcotics. Our experts also raised valid points regarding the potential confounding variables in study subjects that may have predicted failure. A significant number of study subjects had daily pain, endorsed other functional symptoms, required frequent narcotics and/or were being treated for depression.

Perhaps we should now classify patients with PCP according to likelihood for benefit from sphincterotomy for treatment of suspected sphincter of Oddi disease into only two groups; a third group would comprise the remaining patients with nonspecific post-cholecystectomy symptoms.

Group 1

Sphincter of Oddi Stenosis (SOS): This group is the smallest subset and includes the “Type I” patient that has classic biliary pain (intermittent, not daily, severe, lasts minutes to hours, not related to positional changes, and not relieved by bowel movements), a dilated bile duct ($\geq 10\text{mm}$), and a history of increased serum liver tests (only during and/or shortly after pain attacks). Historically, this was described as papillary stenosis and is most likely related to presence and/or passage of biliary sludge. Patients in this group will most often benefit from sphincterotomy.

Group 2

Sphincter of Oddi Dysfunction (SOD): Patients with suspected SOD represent the most interesting subset and may include some described previously as “Type II” and likely a minority of “Type III” patients. Again, classic biliary pain is required and there should be objective evidence of at least intermittently impaired biliary drainage such as elevated liver chemistries during attacks or abnormal biliary scintigraphy. With time, they may develop bile duct dilation (that is not related to longstanding narcotic use), and may actually progress to having SOS (Group 1) as defined above. While some studies have suggested that SOM can predict a benefit from sphincterotomy, further investigation is required. Clearly, there is need to identify clinical predictors of response to sphincterotomy among this cohort.

Group 3

Other Dysfunction or Disorder (ODD): Patients in this group represent the largest subset and present the greatest challenge. They have post-cholecystectomy symptoms including pain that is not classic for biliary pain, often in the setting of other pain syndromes, have no other objective evidence of biliary obstruction and typically have additional wide-ranging complaints that may be functional in origin. Ironically, it seems to be this subset of patients that most often demand ERCP and intervention despite knowing the considerable risks. This likely reflects their level of desperation and/or psychological distress.

The bottom line...conclusion

It is most important to consider sphincter of Oddi disease as the cause of PCP only in patients with firm clinical evidence that support the diagnosis. Secondly, one must be mindful of the many other possible explanations for pain such as fatty liver, abdominal wall myalgia, functional dyspepsia, chronic pain syndromes, etc.

The principle questions faced when diagnosing SOS or suspected SOD include: 1) is the pain classic for biliary pain 2) are there objective data to suggest impaired biliary obstruction, and 3) are there other predominant associated symptoms and/or diagnoses that will clearly not benefit from sphincterotomy? Currently, ERCP with sphincterotomy is only indicated for clear evidence of SOS. Further studies are needed in patients with suspected SOD to determine clinical indicators for ERCP and outcomes after endotherapy.

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Disclosures: None

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