



## Review Article

# Post Cholecystectomy Syndrome: Literature Review

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### Abstract

**Background:** The aim of this literature review is to identify any potential interrelated factors between patient clinical presentation, their disease profile, and the presence or absence of Post Cholecystectomy Syndrome (PCS).

**Observations:** This review focuses on aetiologies of PCS both within the biliary tree and extra-biliary. Additionally, there is an examination into the management and investigation of someone presenting with this syndrome.

**Conclusions:** The definition of Post-Cholecystectomy Syndrome (PCS) is broad and varies significantly across the literature, encompassing a range of non-specific symptoms that may or may not be linked to or exacerbated by gallbladder removal surgery. There is no consensus on the timing of symptom onset after cholecystectomy, with definitions varying widely and many symptoms not necessarily related to the biliary system. It remains uncertain whether some of these symptoms are directly attributable to the removal of the gallbladder, thus labelling them as a syndrome may be inaccurate. Post-laparoscopic cholecystectomy patients presenting with pain may actually be suffering from another pathology, and physicians should rule out these conditions before conducting further investigations into the biliary system. The timing of symptom onset is crucial; symptoms developing long after surgery are less likely to be attributed to cholecystectomy. Research indicates that within three years of surgery, non-biliary pathologies are the primary cause of PCS symptoms, whereas biliary-related pathologies become more common beyond three years but are unrelated to the cholecystectomy itself. Therefore, if a patient experiences symptoms of biliary dysfunction after cholecystectomy, the term PCS should prompt further diagnostic evaluation rather than being considered a diagnosis in and of itself.

**Keywords:** General Surgery; Post Cholecystectomy Syndrome; Surgical complications; Surgical outcomes

### Introduction

Post Cholecystectomy Syndrome (PCS) refers to the reappearance of symptoms similar to those experienced before undergoing cholecystectomy. These symptoms typically include upper abdominal pain, particularly in the right upper quadrant, as well as dyspepsia and sometimes jaundice [1]. These symptoms may be a continuation of those attributed to gallbladder pathology or the development of new symptoms that would have been caused by the gallbladder. In addition, PCS may include symptoms resulting from the removal of the gallbladder, such as gastritis and diarrhoea [2]. However, PCS is only a provisional diagnosis and should be replaced with a more specific name once a comprehensive

workup has identified the underlying disease [2]. There has been conjecture that as there is no unifying diagnostic or management algorithm for PCS, the term should be used sparingly if at all [3]. The prevalence of gallstone disease is significant. As a result, the cholecystectomy is one of the most performed abdominal surgeries in the country. In Australia in 2020-21 there were 60393 cholecystectomies performed [4]. Similar figures can be observed globally. Gallstone-related symptoms account for approximately 60,000 cholecystectomies per year in the UK [5]. Most patients recover smoothly after the surgery. However, around 10% of patients may experience symptoms of Post-Cholecystectomy Syndrome (PCS) several weeks or months after the procedure [5].

### Methods

English-language reports related to Post cholecystectomy

syndrome were sought using the PubMed and Scopus databases. This encompassed studies that examined diagnosis, investigations, management, and possible treatment of PCS. The studies that were selected were published between January 1, 2000, and December 31, 2021. Seminal studies published before 2000 were included when relevant to the review and when more recent data was not available. A total of 596 studies were identified. Of these, of 41 were included for analysis.

## **Discussion**

### **Definition**

Womack and Crider were the first to describe PCS in 1947 [6]. This condition is characterized by the continuation of gastrointestinal symptoms after cholecystectomy and has been documented to affect 5-47% of cholecystectomy patients [7-9]. The ongoing occurrence of gastrointestinal symptoms are described as those which resemble the symptoms that an individual experienced before undergoing cholecystectomy. These symptoms may include right upper quadrant pain, fatty food intolerance, nausea, vomiting, heartburn, flatulence, indigestion, diarrhea, jaundice, and intermittent abdominal pain [5]. While common, many of these symptoms are non-specific and depend on the root cause. As a result, they do not provide much diagnostic utility in isolation. Ongoing biliary colic is a usual occurrence, while severe abdominal pain, jaundice, fever, and chills are infrequent but if they appear, they suggest a higher chance of identifying a specific and treatable cause in contrast to patients with mild, dyspeptic, or non-specific symptoms [1]. Despite its prevalence in medical literature, the term Post cholecystectomy syndrome is imprecise as it covers a broad range of both biliary and non-biliary conditions [10]. Half of the patients experience symptoms within a few weeks of surgery, while the remaining experience symptoms anywhere from months to years later [3].

### **Causes Of PCS**

There are numerous potential factors that can cause PCS, with approximately 50% of patients experiencing an organic disorder of the pancreatobiliary or gastrointestinal system [11]. The remaining individuals suffer from various psychosomatic or other extra-biliary causes of their symptoms [3,12]. It's important to note that there are many causes of the symptoms that are unrelated to the biliary system.

### **Aetiological Contributors**

Multiple possible diagnoses for causative pathologies exist in the PCS. This is part of what makes documenting it so difficult. There are many aetiological agents, all of which have their own clinical vignette. The Rome III committee suggested that PCS pain can be classified based on the type and location of symptoms, which

can aid in characterization [13]. Post-surgery, retained stones, formation of new ductal stones, strictures, and sphincter of Oddi dysfunction (SOD) may cause PCS, all of which can be linked to biliary aetiology [14]. Phillips et al., hypothesised that most patients who experience post cholecystectomy syndrome may have extra-biliary or organic disorders, such as gastroesophageal reflux disease, acute pancreatitis, or chronic pancreatitis [15]. Determining whether these disorders were pre-existing and worsened by the post-operative changes in biliary kinetics or are new symptoms resulting from the procedure is a contributor to the diagnostic challenge.

In 2019, Isherwood et al. published a systematic review examining the causes of PCS in their respective study populations. They observed that within the first three years following cholecystectomy, gastric pathologies such as peptic ulcer disease, hiatus hernia, and gastro-oesophageal reflux disease were the most frequently identified causes of PCS, with prevalence rates ranging from 11% to 100%. In the longer term (i.e., more than three years post-cholecystectomy), retained ductal stones within the biliary tree were the most reported cause, found in 4% to 40% of participants. Sphincter of Oddi dysfunction (SOD) accounted for 1.8% to 31% of cases in an unselected population with PCS, according to various studies. However, in a population pre-selected with likely SOD, only 25% to 47% of cases were confirmed to have true SOD based on liver biochemistry, ERCP, or manometry as per the Rome or Milwaukee Criteria [16,17]. In these studies, 4.1% to 50% of cases had no identifiable cause for their symptoms [3].

Isherwood et al. also remarked that the literature on managing post cholecystectomy syndrome is scarce and inconsistent, with limited participant numbers and flawed methodology in the available studies. Stating that their study has several limitations, including the broad definition of PCS, poor and underpowered quality of studies, and variations in modes of investigation, making the synthesis of aetiological data less reliable [3]. The division of contributing pathologies to PCS can be considered as follows: Extra biliary abdominal disease, Organic Biliary disorders, Functional Biliary disorders, and Post Operative complications [1,17]. PCS has been attributed to multiple diagnoses within these subgroups. The breakdown of specific diagnoses is below:

#### **Abdominal - Extra biliary**

The following are examples of extra-biliary aetiologies as described in the literature.

**Peptic ulcer disease, GORD, Hiatus Hernia** [8,18-23]

**Pancreatitis** [10,19,24,25]

**Hepatitis** [20,24]

**Biliary – organic**

**CBD stone/ Microlithiasis** [10,19,21-28]

**Benign/malignant stricture** [10,19,26]

**Biliary – functional**

**Cholangitis** [10,19,23]

**Sphincter of Oddi stenosis** [10,19,23,24,29,30]

**Sphincter of Oddi dysfunction/biliary dyskinesia** [10,19,22,23,26-30].

**Post operative**

**Cystic duct remnant** [14,15,23,24,26].

**Insufficient cholecystectomy** [24].

**Management and Investigation**

There are various management options available for PCS based on the underlying cause, including medical, endoscopic, and surgical interventions with the choice of management contingent on the patient's pre-morbid state and the aetiology of PCS [3]. As management is dependent on the aetiology of PCS, significant efforts should be made to promptly identify a causative agent. While guidelines exist for managing known causes of PCS such as retained or de novo stones, there are currently no consensus published guidelines for investigating the underlying aetiology of PCS when it is unknown [3].

Several studies used a range of blood results, radiology, and endoscopy to investigate and diagnose the cause of PCS. However, up to 50% of patients had PCS symptoms despite normal investigations. PCS has a psychosocial influence and is associated with functional PCS and social status [5]. Time should be taken to examine the common causes of PCS. The most prevalent causes of PCS are easily detectable on endoscopy or routine imaging [31]. The standard workup for post-cholecystectomy syndrome typically includes the following: Full blood examination (FBE) to exclude any infectious factors. Additionally performing a complete metabolic panel including liver enzymes, amylase, lipase, and Prothrombin Time (PT) to assess for possible hepatobiliary or pancreatic pathologies [10]. Liver function tests can be considered however they should be interpreted in conjunction with other investigation findings. Filip et al. found that The LFT parameter that proved most valuable was the analysis of serum bilirubin, but its overall performance was only moderate with an accuracy of 63%. Although serum alkaline phosphatase levels were elevated in the majority of late-PCS patients (with a sensitivity of 89.3%), it exhibited poor specificity for identifying bile duct disease in patients experiencing post-cholecystectomy pain, and its overall accuracy was only 49.8%. On the other hand, Transabdominal

ultrasound (TUS) had an overall accuracy of 76.4%. However, if abnormal LFTs were correlated with TUS, the accuracy increased significantly to 90% [10].

A significant focus in investigating PCS is on imaging of the RUQ to ascertain a cause for the syndrome. The conventional method of imaging for PCS involves ultrasonography and/or CT, with direct cholangiography being the preferred standard [2,10]. Due to its non-invasive nature, accessibility, speed, and utility in differentiating between obstructive and non-obstructive jaundice, TUS is commonly the first imaging technique utilized to assess patients experiencing suspected biliary pathologies in PCS [32]. However, TUS has a lower sensitivity (77.3 vs 96.2) and specificity (74 vs 88.9) than Endoscopic US (EUS) when used for diagnosis of late PCS [10]. Recently, MRCP has emerged as a trustworthy and non-invasive substitute for direct cholangiography in assessing the biliary tract. As a result, there has been a growing trend of using MRCP to evaluate patients with suspected biliary causes of PCS [31]. If technically successful, ERCP is likely the most precise diagnostic test available, with a sensitivity and specificity of 100% and 95.2% respectively, enabling clear visualization of the papilla, pancreas, and biliary system. It also allows for tissue diagnosis and therapeutic interventions in case of any detected pathology [10]. However, ERCP carries a notable risk of complications, the most frequent being acute pancreatitis, which occurs in approximately 5% of low-risk patients and up to 40% of high-risk patients [10].

**Imaging Findings In PCS**

**Lithiasis**

The most prevalent biliary manifestation for PCS is the presence of calculi in either the common bile duct (CBD) or the remaining cystic duct [3]. These calculi can be classified as "retained" or "recurrent" depending on whether they are discovered before or after two years post-surgery, respectively. Retained calculi are likely to have existed during the surgical procedure, while recurrent calculi are typically "secondary calculi" that form due to biliary stasis resulting from co-existing conditions such as strictures, papillary stenosis, or biliary dyskinesia [1]. [10] suggest that TUS is the initial evaluation method as it is useful for differentiating between obstructive and non-obstructive jaundice. They suggest it is possible to accurately detect obstruction in as many as 95% of patients, and the underlying reason for the obstruction can be determined in up to 85% of patients [10]. However, certain diagnoses such as choledocholithiasis have a lower sensitivity, with less than 50% accuracy [33,34]. MRCP is a highly sensitive diagnostic tool with a sensitivity of 95-100% and specificity of 88-89% for detecting CBD calculi and should be considered in cases where TUS/EUS cannot determine a cause for the symptoms [31].

### **Bile duct injury and bile duct leaks**

According to Coakley et al., laparoscopic surgery has a slightly higher incidence of bile duct injuries (0.5%) compared to open surgery (0.15%). With a laparoscopic approach, the gallbladder and cystic duct may not be fully isolated, leading to incomplete isolation of anatomical structures and/or traction injury [35]. The primary causes of ductal injury are improper cutting of bile ducts, unintentionally placed clips or ligatures, periductal bile leakage resulting in fibrosis, and thermal injury due to electrocautery [31]. Biliary leaks often occur at injured ducts, the cystic duct stump, and the gallbladder stump. After cholecystectomy, up to 14% of patients may experience fluid collections in the gallbladder bed, which usually resolve spontaneously; however, collections that persist for more than a week or fluid outside the gallbladder bed may indicate a biliary leak or injury [1]. Bile duct injury can present as a leak, stricture, or transection [36]. On MRCP, strictures and transections appear as a focal narrowing or abrupt interruption of the bile duct, respectively, with or without biliary dilatation upstream [31].

### **Strictures**

Post-cholecystectomy, strictures can occur in up to 0.6% of cases [16]. Typically, if the stricture does not involve previously injured anatomical variations, it arises from injury to the common bile duct (CBD) during clamping or ligation of the cystic duct in close proximity to its insertion point. Strictures are the most frequently occurring late complication of biliary surgery, developing several months to years after cholecystectomy [37]. When viewed on MRCP, they typically present as a constriction of the inner signal and are generally brief with uniformly even edges [31].

### **Biliary Dyskinesia and Papillary Stenosis**

The term “biliary dyskinesia” has commonly been used to refer to sphincter of Oddi dysfunction (SOD) that primarily involves motor function [16]. It is more frequently diagnosed years after cholecystectomy and affects 9-11% of patients with abdominal pain [38]. Papillary stenosis, an organic variant of SOD, is related to fibrotic narrowing of the sphincter in response to inflammation caused by pancreatitis or gallstone migration through the papilla [38]. In these patients, abdominal pain is thought to result from impeded flow, leading to ductal hypertension, inflammation, and distension. Sphincter of Oddi Manometry (SOM) is still considered the standard diagnostic tool for SOD and to guide therapy [16]. However, abnormal SOM findings are uncommon in SOD Types II and III and are not reliably different between primary motor and stenotic forms [39]. The role of MRCP in diagnosing SOD is unclear because correlation with ERCP or biliary manometry has been poorly investigated, and differentiation between stenotic or spastic papilla is challenging [16]. Advanced MRCP techniques

may serve as a non-invasive first-line tool to demonstrate biliary abnormalities in patients with possible SOD and could ultimately guide diagnostic investigation.

### **Utility of MRCP in PCS**

The reliability of MRCP techniques in identifying biliary causes of PCS has been established. It is crucial to accurately determine the location and type of biliary abnormality to customize patient therapy and create a plan for interventional procedures. One of the primary limitations of MRCP in assessing PCS is its ability to diagnose SOD [31]. To improve the diagnostic accuracy of MRCP in patients with SOD and flow obstruction, secretin-enhanced MRCP has been proposed as a promising non-invasive diagnostic tool. Secretin, a polypeptide hormone, stimulates the secretion of bicarbonate-rich fluid and temporarily increases the tone of the sphincter of Oddi [39]. Currently, there is limited data on the ability of secretin-enhanced MRCP to predict SOD by evaluating main pancreatic duct dilation (degree and timing pre- and post-stimulation) and duodenal filling before and after stimulation in patients with suspected SOD [39]. However, it is generally believed that secretin-enhanced MRCP could be a useful initial non-invasive tool to rule out organic underlying disorders like chronic pancreatitis in patients with PCS [39].

### **Imaging Algorithm**

The initial diagnostic tests for suspected PCS are ultrasound (US) and liver function tests (LFTs). If the common bile duct (CBD) measures 10mm or greater on US, but the cause is unclear, magnetic resonance cholangiopancreatography (MRCP) should be considered [2]. However, if both US and LFTs come back normal, there is no need for MRCP. According to [2], the availability of LFTs enhances the diagnostic value of imaging. This finding was also found by [10]. Following this, if further investigation is required, MRCP is the best suited imaging modality as it can identify nearly all common pathologies associated with the biliary tree [31]. That is with the notable exception of SOD which may be best investigated via ERCP and sphincteric manometry [25,39]

### **Conclusion**

The definition of post cholecystectomy syndrome is broad and varies significantly across the literature, encompassing a range of non-specific symptoms that may or may not be linked to or worsened after gallbladder removal surgery. There is no consensus on when symptoms should occur after cholecystectomy, and definitions vary with many of these symptoms not necessarily related to the biliary system, it is uncertain whether some of these symptoms are truly a result of the gallbladder removal. As such, labelling them as a syndrome may not be entirely accurate. After their laparoscopic cholecystectomy, some patients who present with pain may actually be suffering from GORD or peptic

ulcer disease, and physicians should rule out these causes before conducting further investigations into the biliary system. The timing of symptoms is crucial, as symptoms that develop long after surgery are less likely to be attributed to cholecystectomy. Research has shown that within three years of surgery, non-biliary pathologies are the primary cause of PCS symptoms, while biliary-related pathologies are more common beyond three years but are not related to cholecystectomy. Therefore, if a patient experiences biliary symptoms after cholecystectomy, the term PCS should prompt further diagnosis rather than being considered a diagnosis in and of itself.

In the end, the term PCS may become unnecessary and redundant due to its lack of specificity and utility. The significant variation in symptom profiles as well as pathologies associated with the term make its use somewhat unhelpful in standardising management guidelines.

## References

1. Schofer JM (2010) Biliary Causes of Postcholecystectomy Syndrome. *The Journal of Emergency Medicine* 39: 406-410.
2. Terhaar OA, Abbas S, Thornton FJ, Duke D (2005) Imaging patients with "post-cholecystectomy syndrome": an algorithmic approach. *Clinical Radiology* 60: 78-84.
3. Isherwood J, Oakland K, Khanna A (2019) A systematic review of the aetiology and management of post cholecystectomy syndrome. *The Surgeon* 17: 33-42.
4. Australian Institute of Health and Welfare (2021) Principal Diagnosis data cube under ICD-10-AM Edition 11, 2020-21. Admitted patient care NMDS 2020-21.
5. Jaunoo S, Mohandas S, Almond L (2010) Postcholecystectomy syndrome (PCS) *International Journal of Surgery* 8: 15-17.
6. Womack N, Crider R (1947) The Persistence of Symptoms Following Cholecystectomy. *Annals of Surgery* 126: 31-55.
7. Anand AC, Sharma R, Kapur BM, Tandon RK (1995) Analysis of symptomatic patients after cholecystectomy: is the term post-cholecystectomy syndrome an anachronism? *Tropical Gastroenterology : Official Journal of the Digestive Diseases Foundation* 16: 126-131.
8. Bisgaard T, Rosenberg J, Kehlet H (2005) From acute to chronic pain after laparoscopic cholecystectomy: A prospective follow-up analysis. *Scandinavian Journal of Gastroenterology* 40: 1358-1364.
9. Russello D, Di Stefano A, Scala R, Favetta A, Emmi S, et al. (1997) Does cholecystectomy always resolve biliary disease? *Minerva Chirurgica* 52: 1435-1439.
10. Filip M, Saftoiu A, Popescu C, Gheonea D, Lordache S, et al. (2009) Postcholecystectomy syndrome – an algorithmic approach. *Journal of Gastrointestinal and Liver Diseases* 18: 67-71.
11. Feldman M, Friedman L, Brandt L (2006) Treatment of gallstone disease. In *Sleisenger & Fordtran's gastrointestinal and liver diseases* 8: 1419-1437.
12. Goenka M, Kochhar R, Nagi B, Bhasin D, Chowdhury A, et al. (1996) Endoscopic retrograde cholangiopancreatography in postcholecystectomy syndrome. *The Journal of the Association of Physicians of India*, Feb 44: 119-122.
13. Behar J, Corazziari E, Guelrud M, Hogan W, Sherman S, et al. (2006) Functional Gallbladder and Sphincter of Oddi Disorders. *Gastroenterology* 130: 1498-1509.
14. Zhu J-g, Zhang Z-t (2015) Laparoscopic Remnant Cholecystectomy and Transcystic Common Bile Duct Exploration for Gallbladder/Cystic Duct Remnant with Stones and Choledocholithiasis After Cholecystectomy. *Journal of Laparoendoscopic and Advanced Surgical Techniques* 25: 7-11.
15. Phillips MR, Joseph M, Dellon E, Grimm I, Farrell TM, et al. (2014) Surgical and endoscopic management of remnant cystic duct lithiasis after cholecystectomy – a case series. *Journal of Gastrointestinal Surgery* 1278-1283.
16. Van Hoe L, Vanbeckevoort D, Mermuys D, Van Steenberghe W (2006) Extrahepatic bile ducts - Traumatic, Postoperative, and Iatrogenic Abnormalities. In *MR cholangiopancreatography. Atlas with cross-sectional imaging correlation* 2006: 172-176.
17. Drossman DA (2006) The Functional Gastrointestinal Disorders and the Rome III. *Gastroenterology*, 130: 1377-1390.
18. Caldwell M, McDermott M, Jazrawi S, O'Dowd G, Byrne P, et al. (1995). *Helicobacter Pylori* infection increases following cholecystectomy. *Irish Journal of Medical Science* 164: 52-55.
19. Khuroo M, Zargar S, Yatoo G (1992) Efficacy of nifedipine therapy in patients with sphincter of Oddi dysfunction: a prospective, double-blind, randomized, placebo- controlled, cross over trial. *British Journal of Clinical Pharmacology* 33: 477-485.
20. Soontrapornchai P, Maipang T, Ovartlarnporn B (1997) Postcholecystectomy syndrome after laparoscopic cholecystectomy. *Asian Journal of Surgery* 315.
21. Kochhar R, Malik A, Nijhawan R, Goenka M, Mehta S (1993) H. Pylori in postcholecystectomy symptoms. *Journal of Clinical Gastroenterology* 269.
22. Topazian M, Hong-Curtis J, Wells C, Li J (2004) Improved predictors of outcome in postcholecystectomy pain. *Journal of Clinical Gastroenterology* 38: 692.
23. Zhou P, Liu F, Yao L, Qin X (2003) Endoscopic diagnosis and treatment of post-cholecystectomy syndrome. *Hepatobiliary & Pancreatic Diseases International : HBPD INT* 2: 117-120.
24. Rogy M, Fugger R, Herbst F, Schulz F (1991) Reoperation after cholecystectomy. The role of the cystic duct stump. *HPB Surgery* 129-135.
25. Dilawari J, Chawla Y, Singhal A, Kataria S (1990) Postcholecystectomy syndrome in Northern India. Study on the diagnosis and therapeutic role of ERCP. *Gastroenterology Japan* 1990: 394-399.
26. Druart-Blazy A, Pariente A, Berthelemy P, Arotçarena R (2005) The underestimated roles of opiates in patients with suspected sphincter of Oddi dysfunction after cholecystectomy. *Gastroenterology and Clinical Biology* 29: 1220-1223.
27. Madácsy L, Fejes R, Kurucsai G, Joó I, Székely A, et al. (2006) Characterization of functional biliary pain and dyspeptic symptoms in

- patients with sphincter of Oddi dysfunction: effect of papillotomy. *World Journal of Gastroenterology* 6850-6856.
28. Quallich L, Stern M, Rich M, Chey W, Barnett J, et al. (2002) Bile duct crystals do not contribute to sphincter of Oddi dysfunction. *Gastrointestinal Endoscopy* 55: 163-166.
  29. Kim J, Wu G (2022) Update on Sphincter of Oddi Dysfunction: A Review. *Journal of Clinical and Translational Hepatology* 10: 515-521.
  30. Bennett E, Evans P, Dowsett J, Kellow J (2009) Sphincter of Oddi dysfunction: psychosocial distress correlates with manometric dyskinesia but not stenosis. *World Journal of Gastroenterology* 15: 6080-6085.
  31. Girometti R, Brondani G, Cereser L, Como G, Del Pin M, et al. (2010) Post-cholecystectomy syndrome: spectrum of biliary findings at magnetic resonance cholangiopancreatography. *The British Journal of Radiology* 83: 351-361.
  32. Rogoveanu I, Gheonea D, Saftoiu A, Ciurea T (2006) The role of imaging methods in identifying the causes of extrahepatic cholestasis. *Journal of Gastrointestinal and Liver Diseases* 265-271.
  33. Baron R, Tublin M, Peterson M (2002) Imaging the spectrum of biliary tract disease. *Radiologic Clinics of North America* 40: 1325-1254.
  34. Gandolfi L, Torresan F, Solmi L, Puccetti A (2003) The role of ultrasound in biliary and pancreatic diseases. *European Journal of Ultrasound* 16: 141-159.
  35. Coakley F, Schwartz L, Blumgart L, Fong Y, Jarnagin W, et al. (1998) Complex postcholecystectomy biliary disorders: preliminary experience with evaluation by means of breath-hold MR cholangiography. *Radiology* 209: 141-146.
  36. Khalid T, Casillas V, Montalvo B, Centeno R, Levi J (2001) Using MR cholangiopancreatography to evaluate iatrogenic bile duct injury. *American Journal of Roentgenology* 177: 1347-1352.
  37. Ward J, Sheridan M, Guthrie J, Davies J, Millson C, et al. (2004) Bile duct strictures after hepatobiliary surgery: assessment with MR cholangiography. *Radiology* 231: 101-108.
  38. Piccinni G, Angrisano A, Testini M, Bonomo G (2004) Diagnosing and treating Sphincter of Oddi dysfunction: a critical literature review and reevaluation. *Journal of Clinical Gastroenterology* 38: 350-359.
  39. Aisen A, Sherman S, Jennings G, Fogel E, Li T, et al. (2008) Comparison of secretin-stimulated magnetic resonance pancreatography and manometry results in patients with suspected sphincter of oddi dysfunction. *Academic Radiology* 15: 601-609.