

Introduction

ERCP was first introduced as a realistic endoscopic procedure in the early 1970s. Since then, the diagnostic and therapeutic clinical applications have changed significantly in parallel with improvements in noninvasive and invasive visualization of the biliary and pancreatic ductal systems. What was once predominantly a combined diagnostic endoscopic and radiographic modality, ERCP has taken on new roles as a more sophisticated diagnostic and therapeutic set of procedures including direct visualization of the ducts, tissue interrogation and sampling, and treatment of a wide variety of biliary and pancreatic disorders (Fig. 2.1a, b, c, d, e, f, g, h and i). In the USA, over 500,000 ERCPs were performed in 2008. In 2009, there were an estimated 1.1–1.3 million cases worldwide. The number of diagnostic ERCPs decreased 6% while therapeutic ERCPs increased by 12% up to 2001 [1]. This interventional shift is attributed to the introduction, improvement, and acceptance of other diagnostic modalities such as endoscopic ultrasound (EUS), computed tomography (CT), and magnetic resonance cholangiopancreatography (MRCP). EUS combined with ERCP has become an appropriate

alternative to percutaneous radiological access to an obstructed duct when ERCP alone fails or is not possible.

Despite these changes in the role and range of therapeutic possibilities of ERCP, the basic indications have not. These can be divided into three main categories for the evaluation and treatment of:

1. Stone disease (jaundice, biliary pain, cholangitis, biliary pancreatitis, pancreatic duct stones)
2. Ampullary/papillary abnormalities (Sphincter of Oddi dysfunction (SOD), ampullary cancer)
3. Biliary and pancreatic ductal abnormalities (leaks, strictures, malignancies)

As we shall discuss later in this chapter, there are significant complications of ERCP that one must consider before considering this procedure. Therefore, it is of paramount importance to have an appropriate indication for proceeding.

Stone Disease

Choledocholithiasis

This is still the most common reason for undertaking ERCP (Figs. 2.2, 2.3, 2.4, 2.5). Gallstone disease affects approximately 20 million adults in the USA with an estimated annual healthcare cost of \$ 5.8 billion [2]. Biliary stone disease is responsible for a spectrum of clinical presentations from asymptomatic (detected by imaging) to biliary obstruction, cholangitis, and acute biliary pancreatitis. Choledocholithiasis is seen in up to 15% of patients with cholelithiasis, 10–20% of

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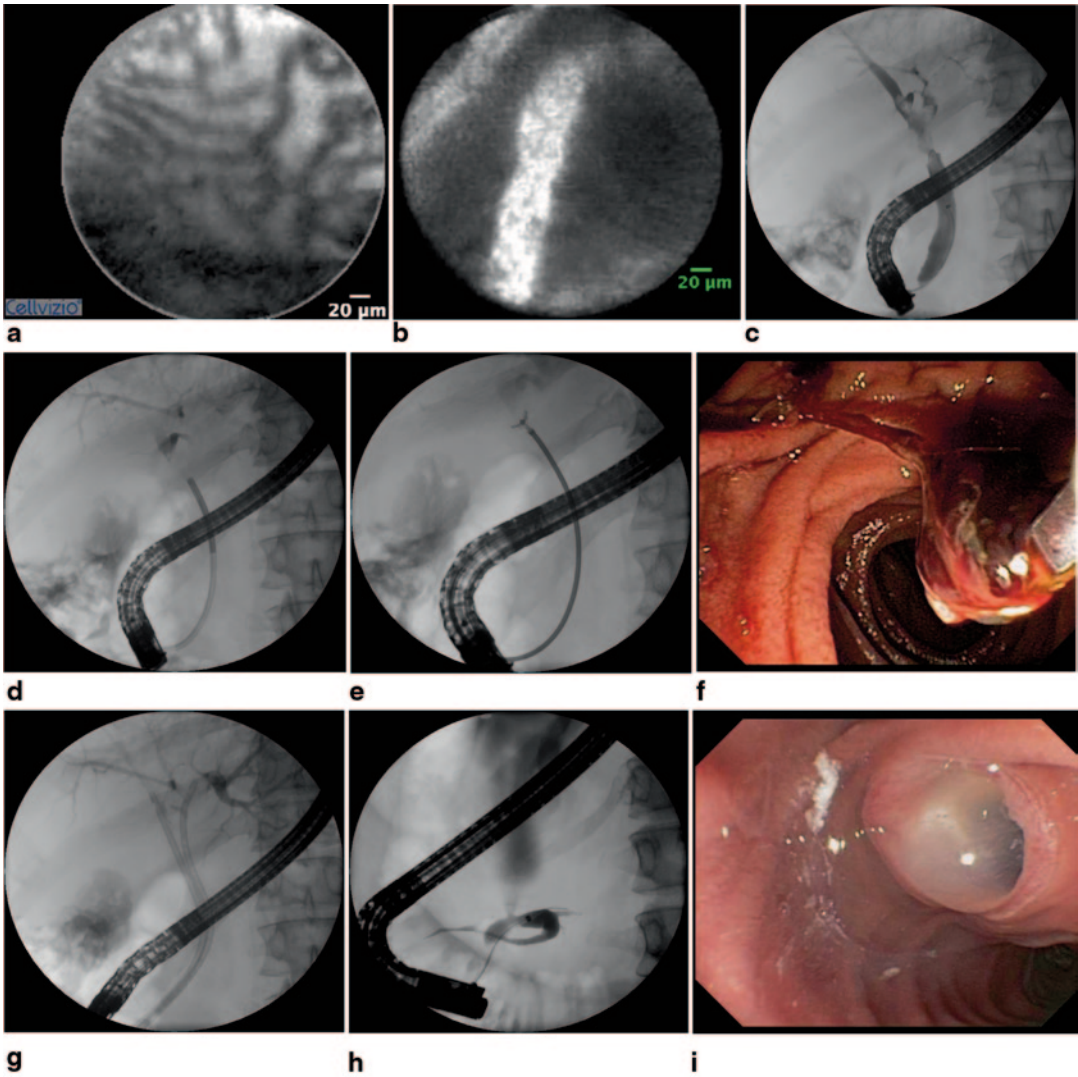


Fig. 2.1 **a** Probe-confocal laser endomicroscopy (pCLE) image of normal bile duct with reticular network of *thin dark branching bands* and *light gray* background. **b** pCLE image of bile duct malignancy with *thick dark bands* and *thick white band* (two criteria for malignant stricture). **c** Endoscopic retrograde cholangiography (ERC) showing filling defect at the hepatic duct confluence. **d** Fluoroscopy

showing cholangioscope advanced to the lesion in **c**. **e** Biopsy of the lesion in **c**. **f** Tissue removed with biopsy forceps (*intraluminal cholangiocarcinoma*) from lesion in **c**. **g** Bilateral plastic stents in the same patient with hilar cholangiocarcinoma. **h** Anomalous union of the bile and pancreatic ducts with type 1 choledochal cyst. **i** Mucus at the papilla in main duct intraductal papillary mucinous neoplasm

those undergoing cholecystectomy, and up to 21% presenting with gallstone pancreatitis [2, 3]. The necessity of expediently diagnosing symptomatic choledocholithiasis is important, as the consequences of failing to do so may result in unfavorable outcomes. Predictors of a high likelihood of choledocholithiasis include jaundice, cholangitis,

severe pancreatitis, alkaline phosphatase (AP) more than twice the upper limit of normal (ULN), increased gamma glutamyl transferase (GGT), and increased alanine aminotransferase (ALT) and/or aspartate aminotransferase (AST) [4]. One study categorized the likelihood of having ongoing choledocholithiasis as “moderate,” “strong” or

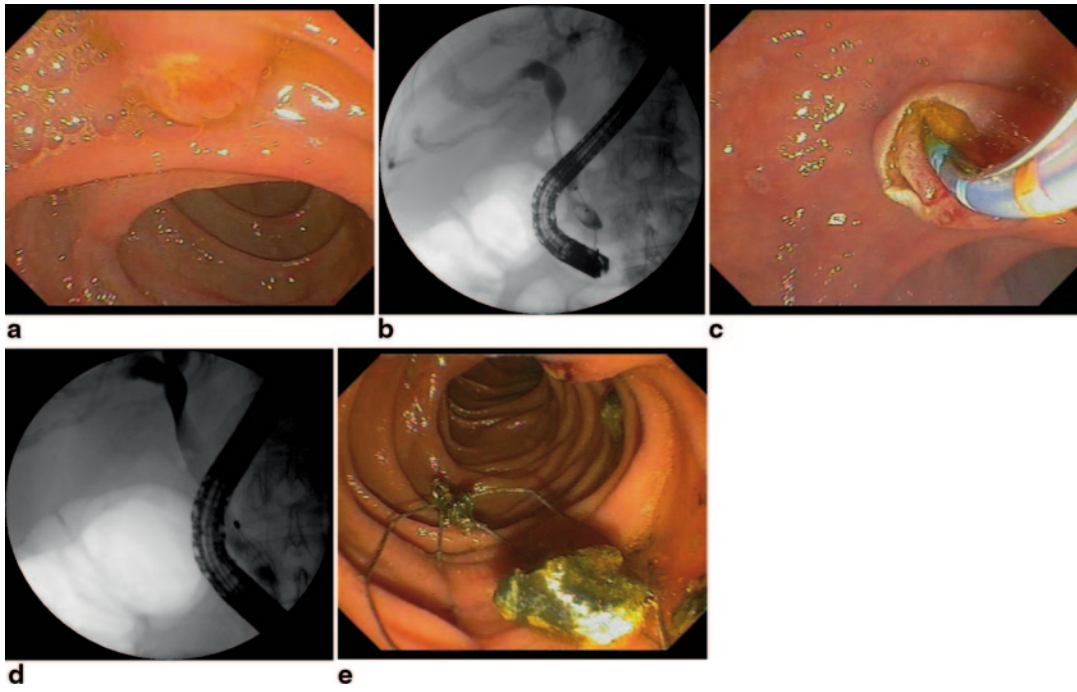


Fig. 2.2 a Sequence from normal papilla. b ERC with distal CBD stones. c Sphincterotomy. d, e Basket extraction of stones. *ERC* endoscopic retrograde cholangiography

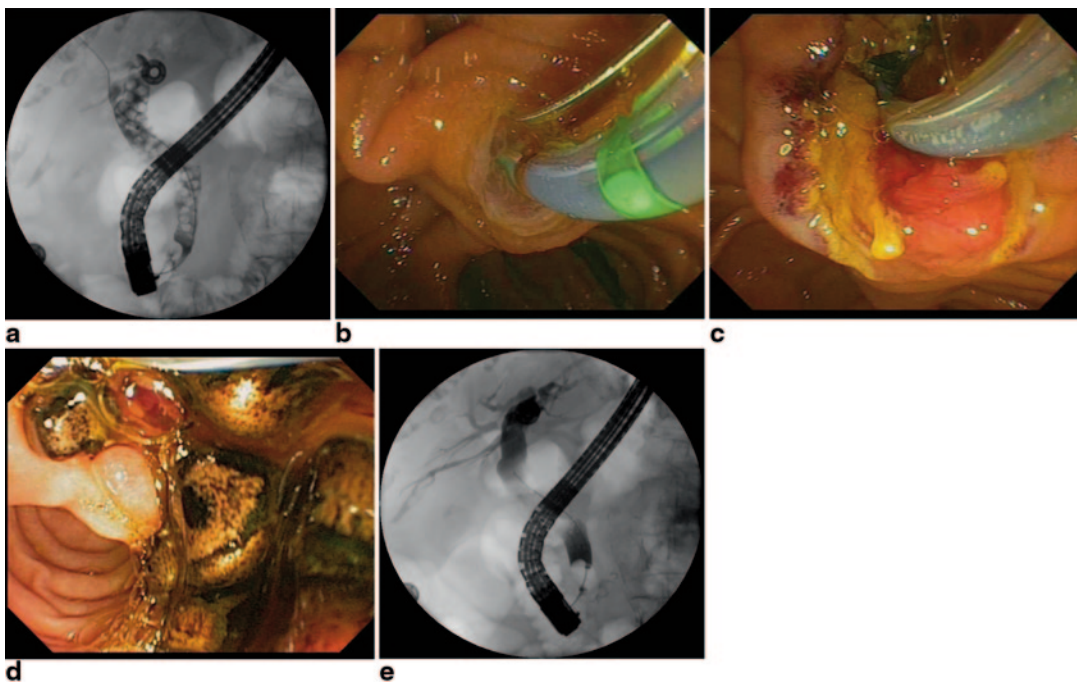


Fig. 2.3 a Sequence of ERC showing multiple stones filling extrahepatic bile duct; b, c Sphincterotomy in the 11 o'clock direction; d balloon extraction; and e Occlusion cholangiogram with biliary stone extraction balloon inflated in distal CBD showing no residual stones. *ERC* endoscopic retrograde cholangiography, *CBD* common bile duct

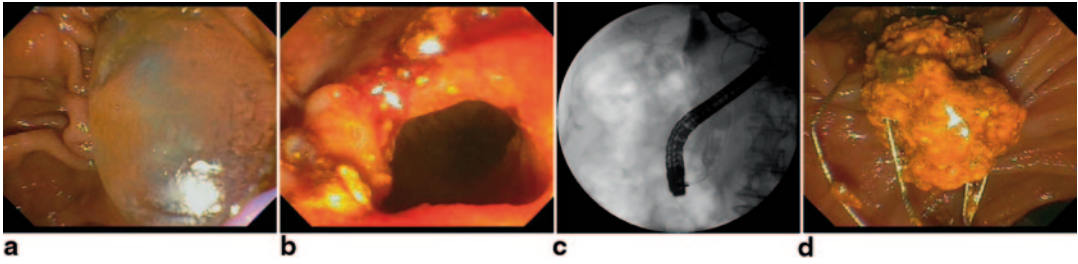


Fig. 2.4 **a, b** Large diameter balloon dilation of the papilla after sphincteromy with **c, d** basket extraction of stone material

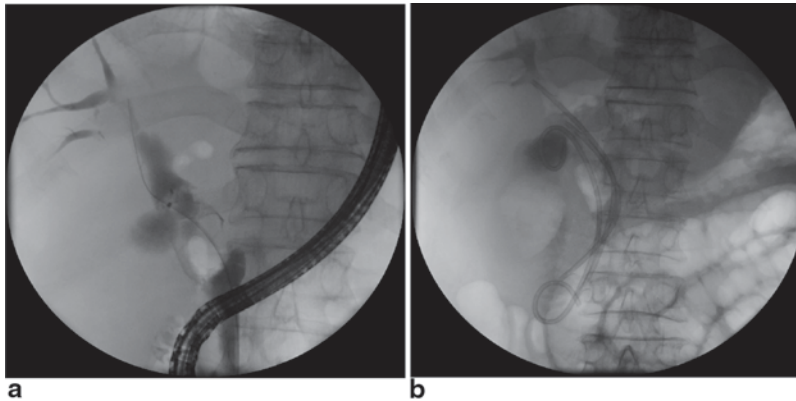


Fig. 2.5 **a** Stone in a cholecystocholedochal fistula, **b** causing biliary obstruction (*Mirizzi syndrome*) treated by CBD and gallbladder stent placement preoperatively. *CBD* common bile duct

“very strong.” Those included in the “very strong” category included visualized choledocholithiasis on transabdominal ultrasound, clinical cholangitis, and a total bilirubin >4 mg/dL. “Strong” indicators included a dilated common bile duct (CBD) >6 mm and total bilirubin between 1.8–4 mg/dL. “Moderate” indicators included abnormal liver tests, age >55 , and clinical gallstone pancreatitis [5]. Based on several prospectively supported algorithms, patients can be risk-stratified into “low,” “intermediate,” or “high” risk for choledocholithiasis [6]. Patients who are “high risk” benefit the most from ERCP as opposed to other noninvasive modalities. In support, the American Society for Gastrointestinal Endoscopy (ASGE) recommends that only patients meeting the criteria for high suspicion undergo an ERCP for choledocholithiasis since it allows for immediate diagnosis and treatment [7]. Sphincterotomy and stone extraction with or without lithotripsy can be performed using the numerous tools now available in order

to relieve biliary or pancreatic ductal obstruction caused by stones.

In 1988, Neoptolemos and Carr-Locke et al. were the first to examine the role of early (less than or equal to 72 h) ERCP in gallstone pancreatitis. Prior to this time, ERCP had been considered contraindicated in this setting. The study demonstrated that only patients predicted to have severe disease, by the modified Glasgow criteria, benefited from ERCP. Although mortality was not affected by early ERCP, overall complications were significantly decreased in the ERCP group (24%) compared to those who received conventional supportive treatment (61%) [8]. In 1993, Fan et al from Queen Mary Hospital, Hong Kong, published a study of 195 patients randomized to either early ERCP within 24 h versus conservative treatment. Morbidity in the ERCP group was significantly decreased compared to patients managed by conservative therapy (16 vs. 33%) [9].

The latest American College of Gastroenterology (ACG) guidelines published in 2013 state that patients with acute pancreatitis and concurrent acute cholangitis should undergo ERCP within 24 h of admission. However, the guidelines further state that “ERCP is not needed in most patients with gallstone pancreatitis who lack laboratory or clinical evidence of ongoing biliary obstruction” [10]. Controversy remains in this area concerning the absolute need for concomitant cholangitis and evidence for biliary obstruction, and there is inconsistency in guidelines for and against this inclusion.

Pancreatic Stones

Nearly always in the setting of chronic pancreatitis, pancreatic duct stones are treated in much the same way as bile duct stones and with the same accessories in symptomatic patients. The treatment of asymptomatic nonobstructing pancreatic duct stones is questionable but an argument can be made for removing stones that are causing complete main duct obstruction in order to improve exocrine function although such patients are not truly asymptomatic. There are differences in approach from biliary stones since the pancreatic duct is a more fragile and tortuous structure, may carry strictures as part of the spectrum of chronic pancreatitis, the stone(s) may be located in the duct and may be impacted, all of which renders the successful extraction of pancreatic stones more problematic compared to their biliary counterparts. Extracorporeal shock wave lithotripsy (ESWL) is a useful adjunct and, if not available, may significantly influence the choice of endoscopic, which may need to be sequential, or surgical therapy.

Ampullary/Papillary Abnormalities

Sphincter of Oddi Dysfunction

The modified Milwaukee classification for biliary SOD, used by many for more than two decades, are:

Type I	Biliary-type pain
	Elevated ALT, AST, or AP on one occasion
	Bile duct diameter > 10 mm
Type II	Biliary-type pain
	One of the other two criteria for type I
Type III	Biliary-type pain only

The approximate frequency of abnormal sphincter of Oddi manometry (SOM) is 65–85, 65, and 59% for type I, II, and III respectively in the post-cholecystectomy patient presenting with presumed biliary pain [11]. Endoscopic biliary sphincterotomy has largely replaced open surgical sphincteroplasty. Regardless of whether SOM is normal or abnormal, 90–95% of type I SOD patients experience pain relief. Therefore, in type I patients, endoscopic sphincterotomy is indicated. In type II SOD patients, the role of endoscopic sphincterotomy is controversial. In patients with suspected type II SOD with abnormal SOM results, 85% will have pain relief with sphincterotomy, but in those with normal SOM results, only 35% will experience pain relief. Regardless, most experienced biliary endoscopists will offer type II SOD a biliary sphincterotomy after discussion of the risks. In type III SOD patients, abnormal SOM has recently been shown not to be predictive of outcome, and empiric sphincterotomy (biliary with or without pancreatic) is not indicated and carries a significant risk. The equivalent pancreatic SOD classification has not been validated as an indication for pancreatic sphincterotomy, but in patients with unexplained recurrent pancreatitis, abnormal pancreatic and/or biliary SOM is often used as an indication for empiric dual sphincterotomy.

Ampullary Cancers/Adenomas

The major duodenal papilla, often interchangeably but erroneously called the ampulla of Vater, can be the source of different types of tumor including adenomas, adenocarcinomas, lipomas, leiomyomas, lymphomas, neuroendocrine tumors, and hamartomas. Adenomas occur sporadically in 0.04–0.12% of the general population, but in those with hereditary polyposis

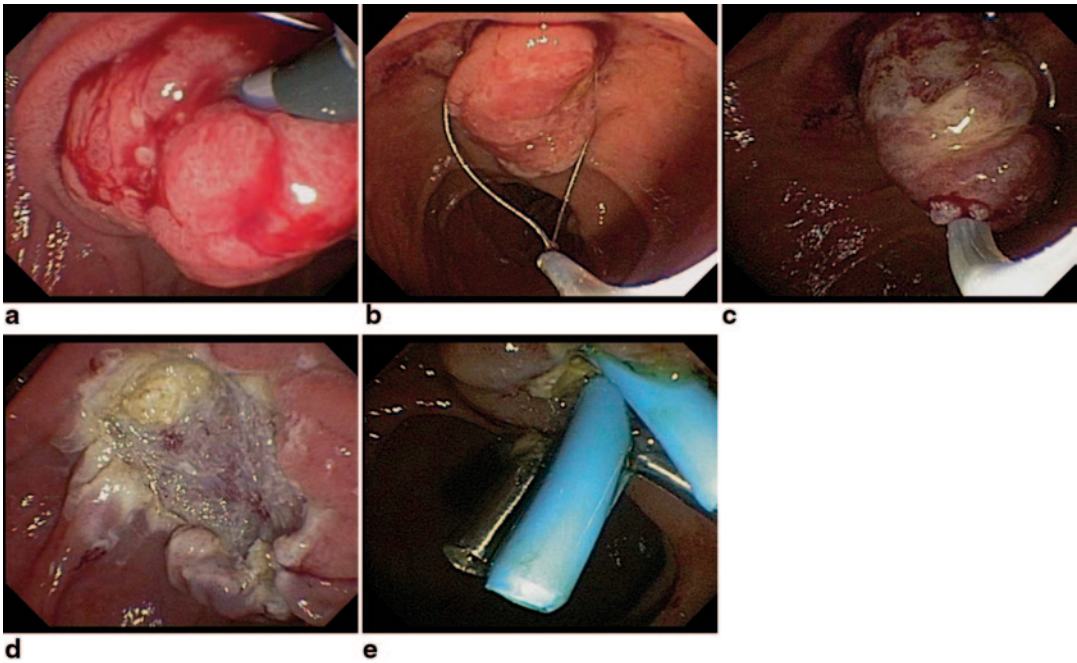


Fig. 2.6 Ampullectomy for adenoma sequence: **a** Canula injecting pancreatic duct with friable adenoma visible; **b, c** Snare cautery en bloc resection of adenoma;

d Postresection with biliary orifice visible in *upper left corner (yellow stain)*; **e** Biliary and pancreatic duct stents inserted with clips visible placed for bleeding

syndromes, the incidence of ampullary adenoma increases to 40–90% [12]. Periampullary adenomas have the potential for malignant transformation into carcinoma at a rate of 30–50% [12] in sporadic cases but the risk in polyposis individuals is also high and this site represents the second highest incidence of cancer after the colon. Two decades ago, the primary treatment of periampullary adenomas was pancreaticoduodenectomy. Due to the increased morbidity and mortality associated with this procedure, especially for a benign disease, the surgery changed to a transduodenal approach with local excision. However, the recurrence rate ranged from 5 to 30% [12]. A review comprising 967 patients undergoing endoscopic ampullectomy reported a recurrence rate of 14% [12]. Endoscopic *en bloc* ampullectomy causes pancreatitis in an unpredictable manner. A prospective randomized controlled trial demonstrated that the placement of a prophylactic pancreatic duct stent conferred a protective benefit against pancreatitis after endoscopic ampullectomy (Fig. 2.6) and should be used in all cases when possible [13].

Cancers in this area can be palliated in the same way as malignant pancreatobiliary strictures (see below) (Fig. 2.7).

Biliary and Pancreatic Ductal Abnormalities

ERCP is of great utility in the diagnosis and management of biliary and pancreatic ductal abnormalities including leaks and strictures. ERCP serves as a platform to access the ductal systems, as it always has, for the purpose of ductography but also to allow sampling by brushing and biopsy. It also permits direct cholangioscopy and pancreatoscopy which further facilitates sampling by directed forceps biopsy and interrogation by confocal laser endomicroscopy and intraductal ultrasound.

Leaks

Leaks from the ductal systems can be treated endoscopically in carefully selected patients.

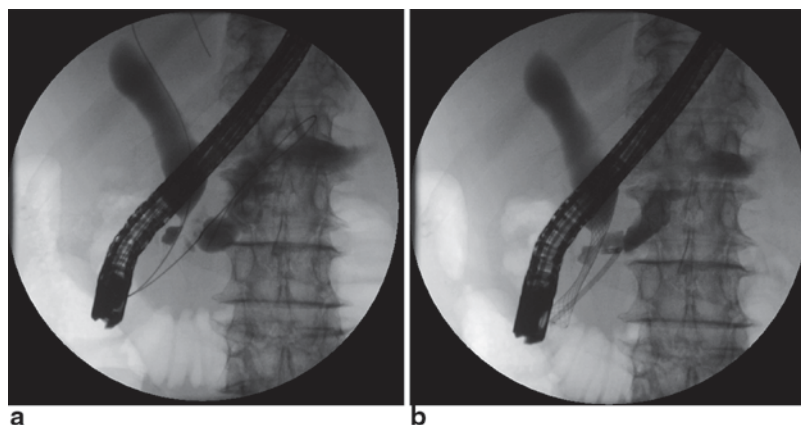


Fig. 2.7 **a** Two wires placed into dilated CBD and PD with distal strictures in patient with ampullary cancer. **b** Metal biliary stent and plastic pancreatic duct stent placed. *CBD* common bile duct, *PD* pancreatic duct

Continuity of the duct to be evaluated and treated is the most important factor determining the feasibility of managing the leak endoscopically [14, 15]. If the bile duct is completely transected or when there is no continuity between the injured segments, endoscopic management is usually not possible. Once duct continuity has been confirmed by cholangiography or pancreatography, the leak can be managed by deploying a stent either across the papilla to reduce intrabiliary pressure in the case of a postoperative biliary leak in an otherwise normal duct, or across the leak itself as in the case of a pancreatic disruption or injury (Fig. 2.8). The types of stent used in these situations continue to evolve as stent technology changes. Two studies performed by Traina

et al and Kahaleh et al. reported resolution of the majority of bile leaks after the use of self-expandable metal biliary stents [16, 17]. However, there were instances of stent migration and stricture formation with the use of these metal stents and cost-effectiveness is questionable. It is hypothesized that the success of biliary stenting in the setting of leaks is attributed to the reduction of transpapillary biliary pressure gradient. The reduction in this pressure gradient diverts flow from the leak site to the intact biliary tree and ultimately into the duodenum. Pancreatic duct (PD) leaks are a result of acute or chronic pancreatitis, trauma, malignancy, and surgery. Varadarajulu et al. demonstrated that successful resolution of a PD disruption was dependent on the type of

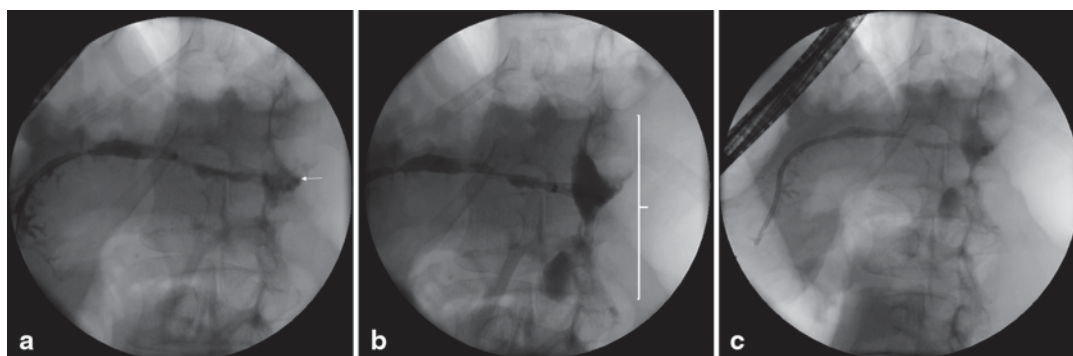


Fig. 2.8 **a, b** PD head stricture and tail disruption (*arrow* and *bracket*) with ascites, **c** treated by stent placement. *PD* pancreatic duct

disruption and the ability to bridge the disruption [18]. A study investigating the role of PD stenting in ductal disruption demonstrated that in 21 out of 28 patients with partial PD disruption who were treated with PD stent alone, the disruption resolved. In six out of eight patients with complete PD disruption, the disruption resolved with PD stenting alone as well [19].

Benign Strictures

The diagnosis of a benign stricture is not always straightforward and usually involves the implementation of the diagnostic sampling tools mentioned above. Once the stricture has been designated as benign and endoscopic therapy chosen as the management plan, either balloon dilation plus stenting or simply stenting alone may be employed. In the case of benign biliary strictures, placement of multiple large bore plastic stents side by side has resulted in good long-term outcomes, [20] but the outcome of self-expandable covered metal stents is being evaluated [21].

Pancreatic duct strictures in the setting of chronic pancreatitis or injury (Fig. 2.9) may also be amenable to endoscopic therapy using the same tools as in biliary applications, but the

pancreatic duct does not necessarily respond in the same way and a plan of sequential pancreatic endotherapy needs to be discussed at the outset. Stents specifically designed for use in the pancreatic duct are available.

Malignant Strictures

In the last 30 years, endoscopic decompression through stent deployment has emerged as the therapeutic procedure of choice in the temporary or permanent palliative management of malignant biliary obstruction (Figs. 2.10, 2.11 and 2.12). Lower hospital costs, shorter hospital stays, and lower morbidity when compared to surgical palliation of malignant biliary strictures have been demonstrated [22]. Biliary decompression can palliate the consequences of obstruction including jaundice, weight loss, cholangitis, secondary cirrhosis, and pruritus thus improving quality of life. Biliary stent therapy, however, has not been shown to have significant survival benefit [23, 24]. Although short-term preoperative biliary drainage with plastic stents is not indicated, metal stents may be cost-effective and, in the potentially resectable patient and/or those undergoing neoadjuvant chemoradiation therapy who have a significant delay between diagnosis and surgery, metal stent placement is indicated.

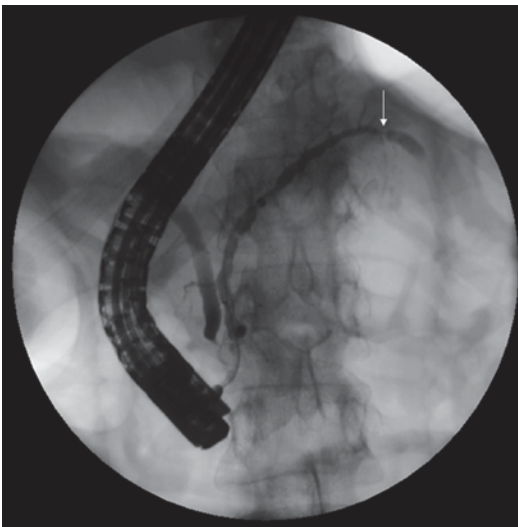


Fig. 2.9 Traumatic PD stricture (*arrow*) from seat belt injury with mild upstream dilation. PD pancreatic duct

When is ERCP Not Indicated or Contraindicated?

Like any invasive procedure, there are circumstances in which ERCP should not be performed. Relative contraindications include:

1. Portal hypertension with esophageal and/or gastric varices
2. Acute pancreatitis except gallstone pancreatitis (this may change)
3. Recent myocardial infarction and/or severe cardiopulmonary disease unless the procedure is life-saving (e.g., cholangitis)
4. Repeated failed attempts at ERCP therapy when alternatives are available

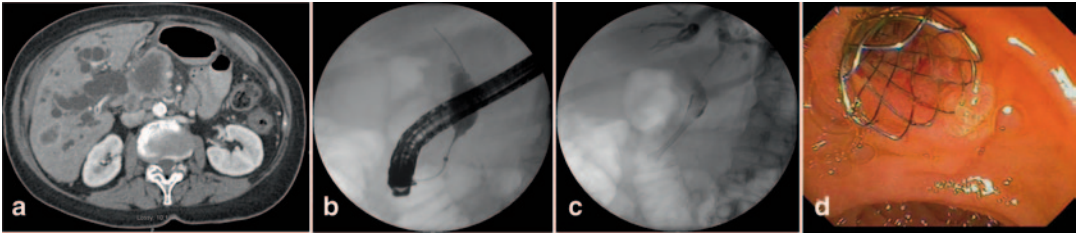


Fig. 2.10 **a** Pancreatic cancer with diffuse intrahepatic biliary dilation on abdominal CT, **b** confirmed by cholangiogram showing distal biliary stricture, **c, d** treated by metal biliary stent placement. *CT* computed tomography

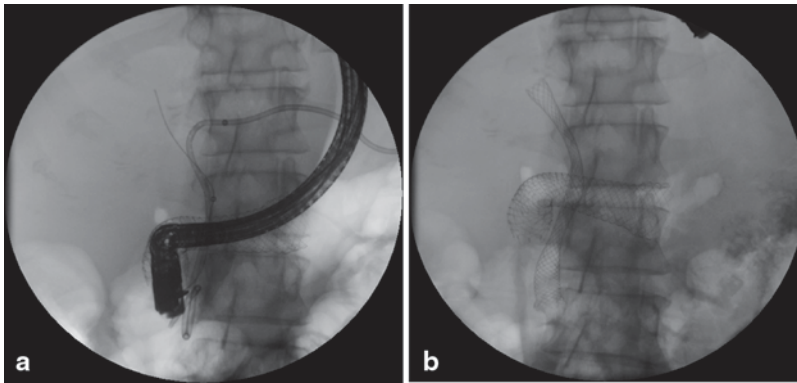


Fig. 2.11 **a** Malignant duodenal stricture treated by metal enteral stent placement and percutaneous biliary drain, **b** exchanged for metal biliary stent

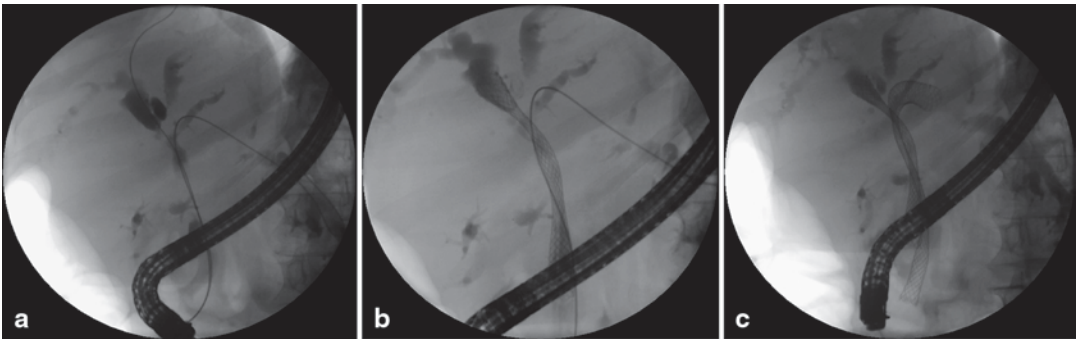


Fig. 2.12 Hilar malignancy treated by bilateral metal biliary stent placement. **a** Two wires advanced into bilateral hepatic ducts. **b** Metal stent placed into right main

hepatic duct. **c** Second metal stent placed alongside into left main hepatic duct

5. Patient cannot be adequately sedated.
6. Anaphylactic reaction to radiographic contrast although this usually refers to reactions after intravenous contrast and there is little to no evidence that ERCP carries the same risk. Local policies will guide this.

Absolute contraindications are the following:

1. Pharyngeal or esophageal obstruction (unless these can be treated simultaneously)
2. Severe uncorrected coagulopathy
3. Inadequate indication, e.g., abdominal pain of unknown cause

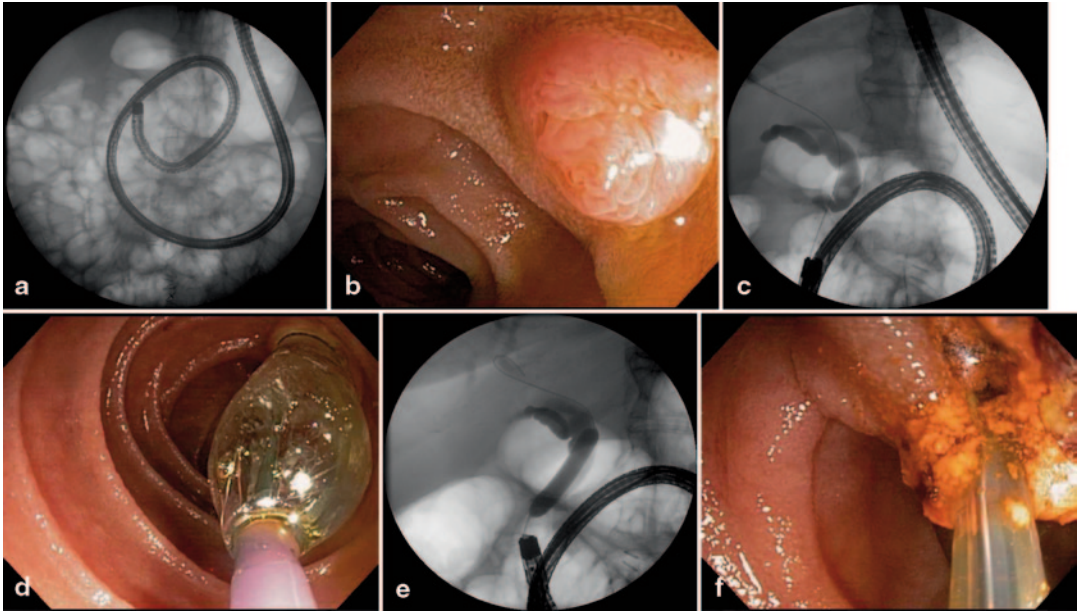


Fig. 2.13 **a** ERC after Roux-en-Y gastric bypass using a colonoscope, **b** showing a normal major papilla, **c** and stone in distal CBD with long guidewire placed. **d**, **e** Bal-

loon dilation of the papilla performed, **f** followed by stone extraction using a biliary stone extraction balloon

4. Altered anatomy (Roux-en-Y, Billroth II, and pancreaticoduodenectomy) without the necessary skills and tools available (Fig. 2.13)
5. Known or suspected perforation
6. Consent cannot be obtained, unless deemed an emergency
7. The risks of the procedure outweigh the potential benefits

Where Do EUS and MRCP Fit in with ERCP?

EUS and MRCP have emerged as diagnostic modalities to aid, or in many cases, completely replace diagnostic ERCP. Both have become well accepted as less invasive and safer diagnostic procedures compared to ERCP that can provide the same information as ERCP without the risks.

MRCP, first developed in 1991, uses heavily T2-weighted sequences to return a high signal from fluid in the biliary and pancreatic ducts, which have long T2 relaxation times [25]. One of the advantages of MRCP is that there is no use of ionizing radiation nor iodinated contrast material

[25]. Another advantage is that MRCP allows for visualization of ductal abnormalities extending into the smaller caliber intrahepatic ducts compared to EUS. Spatial resolution of MRCP compared with ERCP is, however, inferior. Therefore, pathology in nondistended pancreatic side branch or peripheral intrahepatic ducts may be missed [25]. Furthermore, early changes of conditions in chronic pancreatitis and primary sclerosing cholangitis may be missed on MRCP as opposed to ERCP [25].

Where EUS is not readily available, MRCP has become the test of choice in the diagnosis of choledocholithiasis. One study demonstrated that the sensitivity and specificity of diagnosing choledocholithiasis was 100 and 91 % in the EUS group while it was 90 and 100 % in the MRCP group, respectively [4]. Some studies suggest that MRCP is less accurate in detecting smaller diameter stones. For instance, one study reported that the sensitivity of MRCP in the detection of choledocholithiasis decreases from 71 to 33 % as stone diameters fell below 6 mm [2]. Kondo et al. corroborated this by stating that the performance of EUS was superior to

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