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SPECIAL SECTION: PANCREATITIS

Endoscopic and surgical treatment options for chronic pancreatitis: an imaging perspective

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Abstract
Chronic pancreatitis is a chronic fibro-inflammatory syndrome characterized by chronic pancreatic inflammation leading to fibrosis and scarring. Patients with this multifactorial debilitating illness often require endoscopic or surgical intervention for treatment. Radiologists play a crucial role in pre-therapeutic workup as well as post-treatment imaging of chronic pancreatitis. This review summarizes the most common surgical and endoscopic treatment options that are currently available for chronic pancreatitis, including the implications on imaging.

Keywords Chronic Pancreatitis · Endoscopic management · Puestew Procedure

Introduction

Chronic pancreatitis (CP) is an irreversible chronic progressive disease characterized by chronic inflammation leading to fibrosis and scarring. While this disease process has been described over two centuries ago, the term 'chronic pancreatitis' was formed only in 1946 [1]. Majority of the patients with this debilitating illness present with abdominal pain, which can be the sole manifestation of the disease. Other clinical manifestations of chronic pancreatitis result from loss of exocrine (acinar) and endocrine (islet cell) tissue, and include steatorrhea, impaired glucose tolerance, and diabetes mellitus [2]. Chronic pancreatitis is a multifactorial disease, and a major source of morbidity worldwide, including the United States and Europe. Reported annual worldwide incidence and prevalence is 5–14 per 100,000 individuals and 30–50 per 100,000 individuals, respectively [2, 3]. The true prevalence is likely underestimated due to several reasons, including patient compliance, variable definition of the disease, and inaccurate diagnosis. CP is predominantly seen in middle-age or older patients, with median age at diagnosis of ranging from 51 to 58 years [2, 3]. Traditionally, thought to be significantly more common in men, prevalence of CP is increasingly reported in women. Cambridge classification is widely used in imaging diagnosis and grading of chronic pancreatitis (as mild, moderate, and severe) based on the appearance of the main pancreatic duct including the presence and severity of ductal strictures and dilatation of side branches [4].

Besides being a debilitating illness with resultant chronic symptoms, CP increases the risk of developing pancreatic cancer, necessitating a timely and accurate treatment of this entity. The estimated standardized incidence ratio of developing pancreatic cancer in patients with sporadic pancreatitis is 14.4, with up to 5.9% of patients with CP developing cancer 20 years after the diagnosis [5]. Patients with hereditary chronic pancreatitis are diagnosed at a much younger age and have a significantly high chances of developing cancer, with up to 48.1% of the patients developing cancer 60 years after the onset of symptoms [6]. Cancer is the most common cause of death in chronic pancreatitis [7].

The aim of management in chronic pancreatitis is as follows: a. Alleviation of symptoms (pain, exocrine, and endocrine insufficiency) b. Prevention of disease progression c. Prevention/treatment of complications such as pancreatic/biliary duct strictures, and pseudocysts [3]. Various treatment options are available for CP, and an estimated 30%–60% of patients with CP require endoscopic or surgical

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intervention for treatment [8]. Imaging plays a crucial role in preoperative workup as well as postoperative evaluation. In this review, we discuss the most common surgical and endoscopic treatment options available for chronic pancreatitis and its implications on imaging.

Surgical treatment options for chronic pancreatitis

Higher mortality and morbidity associated with pancreatic surgeries including pancreatectomies limit their role in the management of non-neoplastic pancreatic pathologies. Advances in surgical and anesthetic techniques resulting in reduction in mortality/morbidity have led to more frequent utilization of surgical options for management of chronic pancreatitis (CP). Recent studies demonstrate a reduction in mortality related to Whipple's surgery from 25% to 1% [9, 10]. Although controversy exists, recent trends point to superior pain control, better exocrine function with surgery in comparison to endoscopic procedures for select CP patients [11, 12]. Surgical drainage is also thought to be more effective than endoscopic approach in the presence of ductal dilatation [13]. The most common indication for surgery in patients with CP remains intractable chronic pain, not responsive to other measures and imaging concerns for the presence of a malignancy. Up to 15% of patients with CP have been reported to have pancreatic cancer, and CP has been considered a risk factor in developing pancreatic ductal adenocarcinoma [14–16]. Robust evidence for optimal timing of surgical intervention is limited and is tailored after evaluating patient characteristics, anatomic considerations including pancreatic tissue morphology and ductal system, and presence of associated inflammatory mass or concerns for underlying malignancy. Surgical options in CP can be broadly categorized into (a) decompression, (b) resection, and (c) mixed techniques [4]. The following sections provide a detailed account of various surgical options in chronic pancreatitis.

Decompression techniques

Ever since the first description of a Roux-en-Y, side-to-side pancreatojejunostomy in 1947 by Cattell for a single pancreatic stricture, several decompression techniques have been described [17]. Duval and Zollinger separately described caudal end-to-end pancreatojejunostomy in 1954 [18, 19]. Despite early promise, the progressive nature of CP often leads to additional PD strictures which renders these procedures ineffective. To counter the challenges of multiple strictures in CP, Puestow Gillesby procedure describes a longitudinal opening of the duct to a Roux-en-Y loop of jejunum, with resection of the tail of pancreas and spleen (Fig. 1). Subsequently, several modifications of this procedure have been described [20, 21].

Decompression techniques are often suitable for patients with relative conservation of pancreatic head, lack of inflammatory mass in the head, absence of biliary ductal dilation, and with PD dilation measuring at least 6–8 mm to enable mucosal anastomosis [22–24]. Significant post-surgical improvement in CP-related pain has been reported in up to 82% of patients with drainage techniques. However, on long-term follow-up, nearly 50% of patients demonstrate recurrence of pain which worsens over time especially in patients who do not abstain from alcohol [25, 26].

On imaging, following refined Puestow procedure, the roux-en-Y loop can be noted along the anterior aspect of the pancreatic head, which is intimately related to the anterior body and tail of the pancreas (Fig. 2). The retrograde pancreatojejunostomy loop can be filled with contrast, fluid or air, and should not be confused with abscess. Administration of oral contrast helps differentiate collapsed loops of bowel in the surgical bed from other loops of bowel. Post-surgical soft tissue stranding is an expected finding and transient fluid collections (seroma or hematoma) might be encountered. Concerns for pancreatitis or infectious process should be evaluated based on clinical or aspiration findings. The presence of calculi, especially in the head and uncinate process, is a common finding. Unlike other forms of pancreatic resection, pneumobilia is not an expected finding unless there is a prior biliary intervention [27].

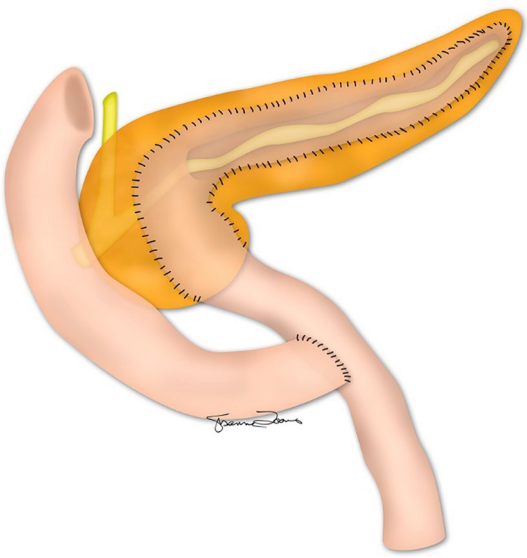

Fig. 1 Schematic illustration of Puestow Procedure which includes longitudinal opening of the pancreatic duct and anastomosis of the pancreatic duct with Roux-en-Y loop of jejunum

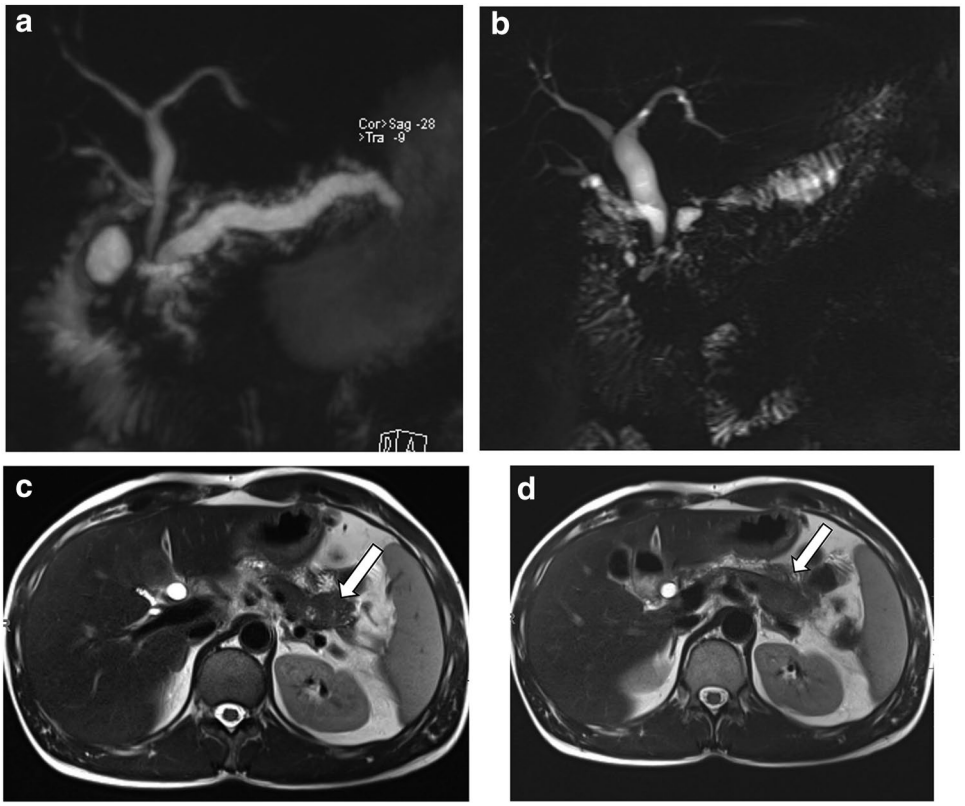

Fig. 2 Imaging before and after Puestow procedure. Coronal MRCP image (**a**) demonstrates findings of chronic pancreatitis with diffusely dilated duct and side branches. The patient underwent Puestow procedure. The post-procedure MRCP (**b**), and axial T2-weighted images (**c**, **d**) demonstrate decompression of the pancreatic duct with roux-en-Y loop along the anterior aspect of the pancreatic head (arrows in **c** and **d**), intimately related to the anterior body and tail of the pancreas

Resection procedures

Surgical decompression techniques for CP were predominantly developed to alleviate pain and are based on the hypothesis that the pain is attributable to ductal hypertension. While chronic pain in CP could be partly related to ductal hypertension, the association has been inconsistently demonstrated [28, 29]. The dominant factor contributing to pain is likely neural in origin, and chronic inflammatory damage to sensory nerves is presumed to be the primary source of pain in these patients [30]. The head of the pancreas is often considered to be the pacemaker of the disease [31]. In addition, despite their benefits, decompression techniques are neither effective nor feasible in those without PD dilation. These factors led to development of resection procedures, prototype of which is the Whipple resection.

First described by Allen O. Whipple in 1912, Whipple procedure is primarily used in the management of malignant neoplasms of pancreatic head, and is now being increasingly used in treatment of CP [9]. It consists of pancreaticoduodenectomy, gastrojejunostomy, pancreatojejunostomy/gastrostomy, and choledochojejunostomy (Fig. 3). Other resection procedures that have been described include, pylorus-preserving pancreaticoduodenectomy (Fig. 4a), denervated pancreatic flap (resection of head and uncinate and leaving a thin rim of tissue near duodenum) (Fig. 4b), subtotal pancreatectomy (excision

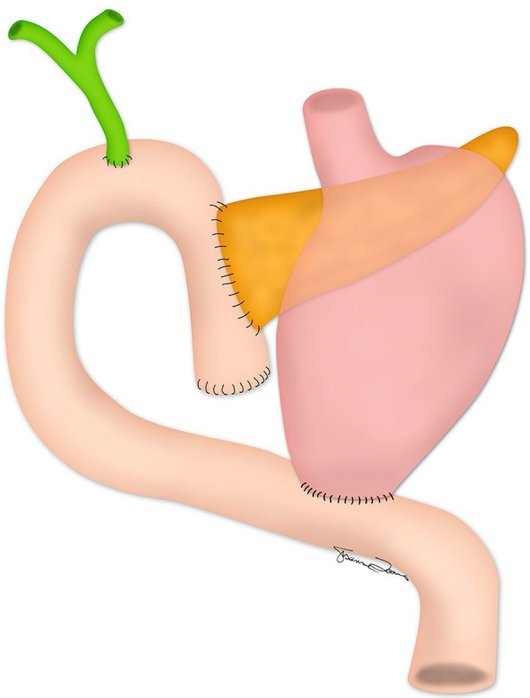

Fig. 3 Schematic illustration of Whipple procedure which includes pancreaticoduodenectomy, gastrojejunostomy, pancreatojejunostomy/gastrostomy, and choledochojejunostomy

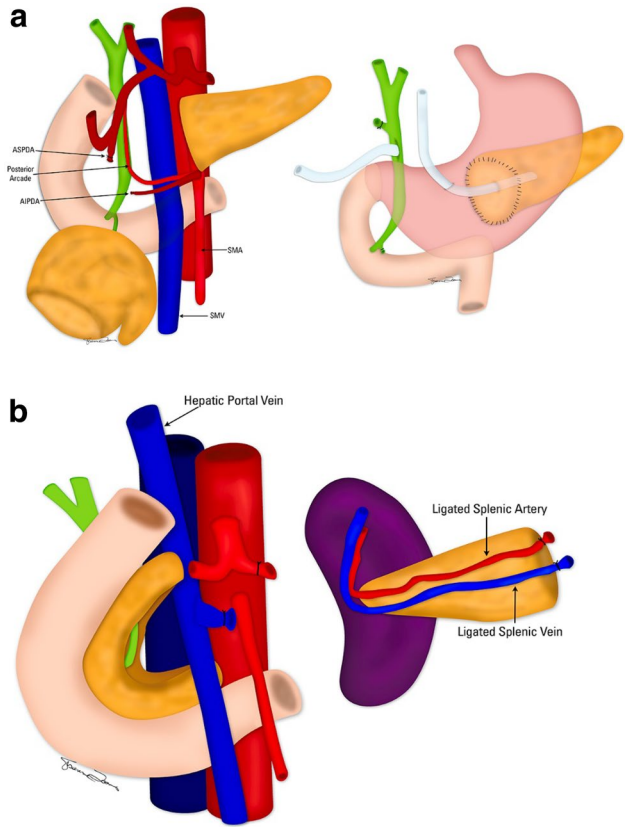

Fig. 4 Schematic illustration of duodenum-preserving pancreatic head resection (**a**) and denervated pancreatic head flap (**b**)

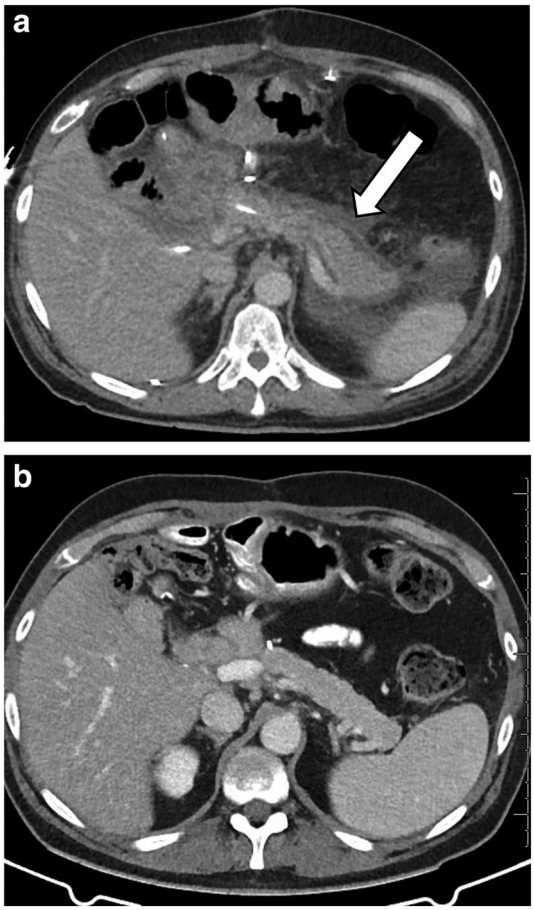

Fig. 5 Normal post-surgical findings after resection procedures for chronic pancreatitis. Axial contrast-enhanced CT images immediately (**a**) and 3 months post Whipple resection shows moderate peripancreatic fluid (arrow in **a**), which resolved on follow-up (**b**)

of portions of tail and body), Child's resection (95% distal pancreatectomy involving the uncinated process, body, tail, and the spleen), and total pancreatectomy. Subtotal and total pancreatectomy procedures are associated with high incidence of endocrine dysfunction (50–72%) and exocrine dysfunction (30%), and are reserved for patients who are resistant to other forms of treatment [32, 33]. Islet-cell auto-transplantation (Total pancreatectomy and Islet-cell auto-transplantation; TPIAT) was therefore introduced to overcome the catastrophic consequences of total pancreatectomy [34]. In this procedure, total pancreatectomy is performed followed by infusion of the islet cells from the pancreas into the recipient's portal vein, where they engraft into the liver. The evidence is still limited, but they indicate improving quality of life and more prolonged narcotic independence [35, 36]. TPIAT is suitable for patient with the hereditary form of CP due to early onset and severity of disease and less encouraged for CPs as a result of chronic alcoholism because of the lower yield of islet cell harvest [34, 37]. Recurrent pain (up to 20% patients) and prolonged insulin-dependency (50–90% of patients) are also described after TPIAT [34, 38].

Imaging after resection procedures

Routine surveillance imaging is not often indicated in the early postoperative period. Multidetector CT (MDCT) is most preferred modality for evaluation of postoperative anatomy and detection of complications. Accurate interpretation of postoperative imaging needs a detailed knowledge of the precise procedure performed, and resultant altered anatomy. In the early postoperative period, all anastomosis should be carefully scrutinized. In pancreatojejunostomy, a jejunal loop is located anterior to the superior mesenteric artery and anastomosed to the right of pancreatic tissue, while in pancreaticogastrostomy, pancreatic tissue is anastomosed to the posterior gastric wall [39]. In a patient with total pancreatectomy, gastrojejunostomy is usually found in the left abdomen [40]. Pneumobilia is a common normal imaging finding in patients with hepaticojejunostomy. Anastomotic edema is also common and leads to the dilation of pancreatic and biliary ducts, which regress over time [39].

Fat stranding and fluid collection can be seen in up to 50% and 28.5% of patients, respectively, and often regress by 3–6 months [41, 42] (Fig. 5). Assessment of pancreatitis in the postoperative phase can be challenging. Fat stranding, inflammatory changes, and elevation of lipase and amylase all can typically be seen in the postoperative period. Nevertheless, imaging can be of value by displaying disproportionate inflammatory changes in the proximity of remnant pancreas.

Anastomotic leaks usually appear in the early postoperative period [10]. The occurrence of the leak or fistula from pancreatic anastomosis is much more common than gastrojejunostomy site (10–30 vs. < 1%), and associated with significant morbidity and mortality [41]. Pancreatic leak is characterized by persistent drainage of fluid on the 3rd postoperative day or later, with the fluid demonstrating amylase content ≥ 3 times the upper normal serum values [43]. Imaging findings that are suggestive of fistula or anastomosis leak are the presence of fluid or air collection in the pancreatojejunostomy site, pancreatic bed, or at the level of the resection margin (Fig. 6). If leak from the gastrojejunostomy is suspected, water-soluble positive oral contrast should be used to evaluate for contrast extravasation. Occasionally, fistulography may need to be performed to evaluate for pancreatic fistulas [44]. Total pancreatectomy is less commonly associated with leak or dehiscence (0.4%) [40]. Biliary anastomotic leaks can also occur and are characterized by bilirubin concentration of fluid collections ≥ 3 times serum bilirubin concentration on or after postoperative day 3 [45]. Hepatobiliary contrast agents (Gadoextic acid) can be used to detect the presence and location of active bile leaks in MRI [46]. A fluid collection with air-bubble, thick enhancing walls with our without significant surrounding inflammation suggests infection [47]. The presence of severely distended stomach, in a proper clinical setting and especially in pylorus-preserving PD, is indicative of delayed gastric emptying [39].

Vascular complications including postoperative hemorrhage can occur (2–10%) and be a source of significant mortality (38%) [48, 49]. Hemorrhage can be divided into intraluminal and extraluminal or early (< 24 h) and late (> 24 h) [49, 50]. GDA stump is the most common site of both early and late hemorrhage. Early hemorrhage is frequently resulted from inadequate ligation while vascular erosion and anastomotic breakdown are also common pathologies in late hemorrhage [49, 50]. Extraluminal hemorrhage is twice more common than intraluminal, which presents with a drop in hematocrit or blood in abdominal drains [48]. On imaging, unenhanced CT is useful in demonstrating high-density fluid/blood, and contrast-enhanced CT might show the site of extravasation or the presence of vascular abnormalities such as pseudoaneurysm (Fig. 7). Dual-energy CT data sets including virtual non-contrast image reconstructions and

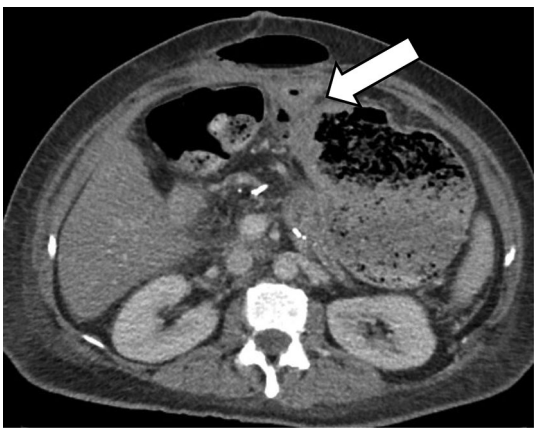

Fig. 6 Anastomotic leak after Whipple resection. Axial contrast-enhanced CT in a patient with gastrojejunostomy anastomotic leak resulting in anterior abdominal wall collection, shows focal wall thickening at the anastomosis, with air-filled tract extending into the anterior abdominal wall (arrow). Note moderate gastric distension with retained residue

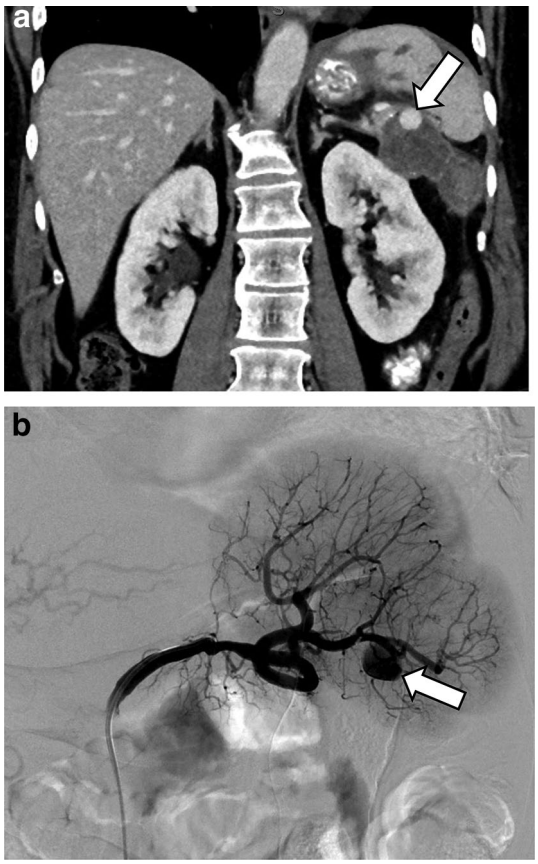

Fig. 7 Vascular complications after partial resection of pancreas for chronic pancreatitis. Coronal contrast-enhanced CT image (**a**) and angiographic image from splenic artery injection (**b**) in a patient with distal pancreatectomy, complicated by pancreatic leak and secondary splenic artery pseudoaneurysm (arrows in **a** and **b**)

iodine specific images offer additional value in detecting hemorrhage while reducing radiation dose by eliminating the need for true non-contrast acquisitions [51]. Thrombosis of the portal vein and superior mesenteric vein (SMV) has been reported with increasing frequency (up to 17%) [52]. Contrast-enhanced CT in the portal venous phase and coronal reconstruction are valuable in diagnosing thrombosis. Hepatic infarction is an unusual consequence seen in less than 1% of patients.

Anastomotic stricture is the most common late complication after CP surgeries. It is best evaluated by MRI and MRCP. Both hepaticojejunostomy and pancreatojejunostomy sites can undergo progressive fibrosis that leads to stricture. Long standing stricture can result in progressive pancreas atrophy and ductal dilation readily detectable on MDCT and MRI.

Imaging consideration for total pancreatectomy and islet auto-transplantation (TPIAT)

TPIAT involves total pancreatectomy, islet cell isolation, and infusion of islet cells into the recipient's portal vein. The infused islet cells engraft into the liver and functions similar to the endocrine pancreas. Preprocedural ultrasound is performed to ensure patency of the haptic and portal veins, evaluate for hepatic parenchymal abnormalities (steatosis or cirrhosis), and to exclude focal hepatic lesions [53]. The success and outcome of this procedure is affected by patency of the vein and the presence of liver parenchymal fibrosis [54]. Postoperative Liver Doppler is performed at 24 h to assess for hemorrhage (parenchymal, subcapsular or peritoneal), patency of the portal vein, and direction of flow. Hemorrhage has been reported to occur in up to 11% of patients and portal vein thrombosis in 3% of patients [53]. Hepatic steatosis, presumably related to abnormal local utilization of insulin, has been reported in up to 20–40% of patients as early as six months post TPIAT, and can regress in up to 50% of patients [55, 56]. Hepatic steatosis in this setting is typically described as granular and heterogeneous, often periportal in distribution [57].

Mixed techniques

Mixed techniques involve a combination of resection and decompression techniques and often involve resection of inflammatory mass in the pancreas head and drainage of the obstructed duct. The most widely used techniques are duodenal preservation techniques (Duodenum-preserving pancreatic head resection, DPPHR). Beger was the first to introduce a duodenum-preserving resection, and its method involves subtotal resection of the head that leaves a small rim of pancreatic tissue between common bile duct and duodenum [58]. The remnant tissue is anastomosed by the side-to-side fashion. Body and tail are drained either by an end-to-end pancreatojejunostomy or a side-to-side anastomosis. Frey technique is less technically demanding and involves excavating the pancreatic head and leaving at least 5 mm of pancreatic tissue, preserving the integrity of the biliary duct. Longitudinal opening of the pancreatic duct and pancreatojejunostomy is followed [59] (Fig. 8). Several other similar modifications of DPPHR techniques have also been described [60, 61].

Postoperative imaging findings after these 'mixed' techniques are similar to the Puestow procedure (Fig. 9). Several complications described above, with 'resection' techniques can also occur with 'mixed' techniques. Fluid collection in the proximity of the head of the pancreas, which corresponds to resection site and biliary dilation are common findings in the postoperative period. As with Puestow procedure, gas in the biliary system is not a normal finding; however, gas in the main pancreatic duct (MPD) is a common finding.

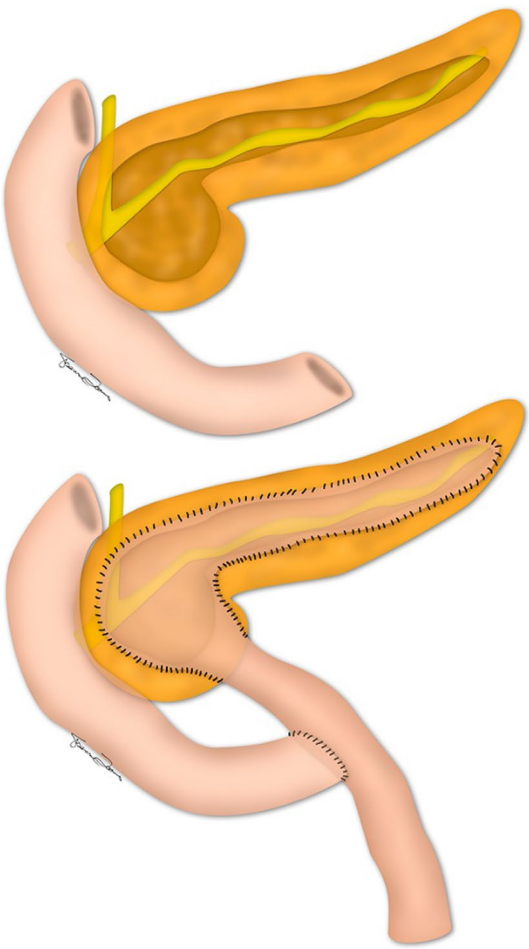

Fig. 8 Schematic illustration of Frey procedure which includes excavation of the pancreatic head, leaving at least 5 mm of pancreatic tissue to preserve the integrity of the bile duct, longitudinal opening of the pancreatic duct and pancreatojejunostomy

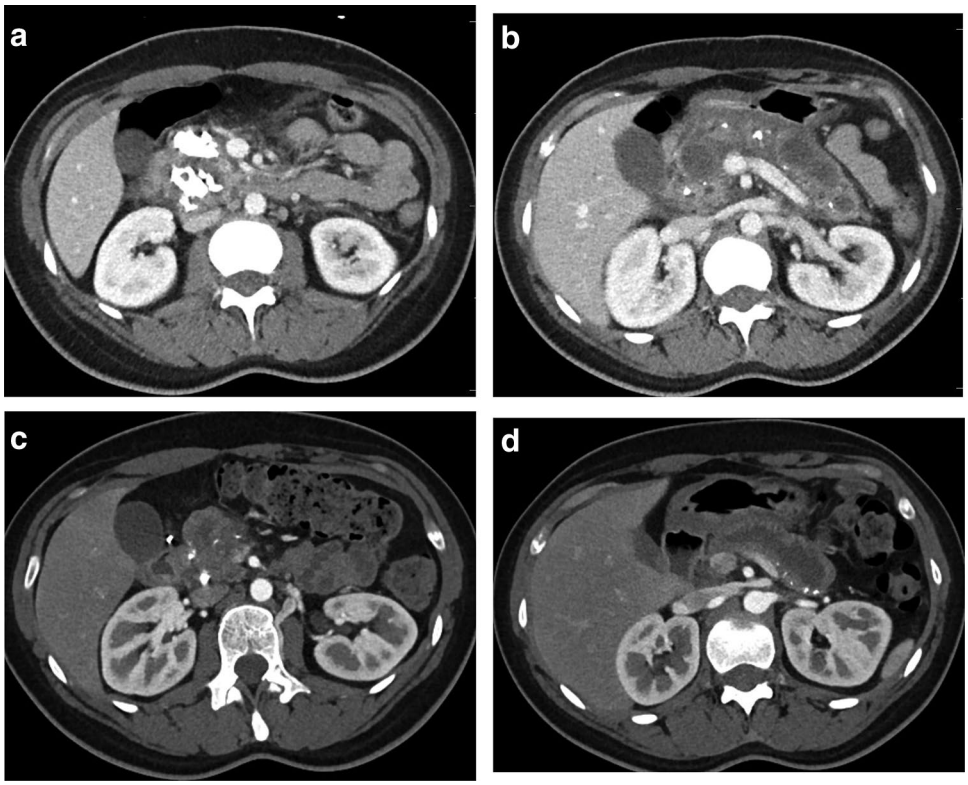

Fig. 9 Frey procedure. Axial contrast-enhanced CT images (**a**, **b**) in a patient with chronic pancreatitis demonstrate diffuse pancreatic ductal dilation with multiple calculi in the head, and diffuse pancreatic atrophy. The patient underwent Frey's procedure. Post-procedure axial contrast-enhanced CT (**c**, **d**) demonstrates normal post- operative anatomy (excavation of the pancreatic head, with preservation of bile duct and pancreaticojejunostomy). The pancreatic duct is no longer dilated

Roux-en-Y loop lies between the pancreatic tail and head. After Frey procedure, large cavity might been seen that corresponds to excavation site of pancreatic head, which will regress in size with time [10]. An important consideration is the ischemia of CBD and duodenum, which might ensue due to extensive head removal and sacrificing the pancreaticoduodenal arteries, potentially resulting in biliary dilatation/leak or bowel obstruction. Prolonged, extensive dilation of CBD and rising bilirubin level should raise concern for the ischemia [2].

Endoscopic treatment options for chronic pancreatitis

Although surgery has shown to be better than endoscopic interventions in long-term pain control [62], endoscopic management is still considered as the first-line therapy for chronic pancreatitis in general, and has also been recommended by United European Gastroenterology evidence-based guidelines for initial treatment [63]. The presence of stone-predominant vs. stricture-predominant disease dictates the choice of endoscopic therapy for pain management.

Pancreatic stone disease

Pancreatic ductal stones can be retrieved via the endoscopic cannulation of papilla or fragmented with extracorporeal shock wave lithotripsy (ESWL). ESWL has high success rate in stone fragmentation [64, 65]. Successful stone fragmentation depends on accurate preprocedural mapping of the size and location of stones. In general, stones ≥ 4 mm are targeted. If multiple stones are present, the stones closest to the papilla are first targeted. Stones which are further upstream are successively targeted after the more downstream stones are fragmented and cleared [63]. Stones in the tail and the side ducts are usually not targeted, because they only drain a small portion of the pancreas, they may not be significant in causing pain and stone fragments might migrate into the more proximal MPD resulting in obstruction [66]. The aim of ESWL is to fragment stones to less than 3 mm in size, to facilitate expulsion via the papilla. Expulsion of fragments can be facilitated by secretin administration [67] or by endoscopic extraction. Primary endoscopic stone extraction prior to ESWL is usually less successful and only reserved in select patients with radiolucent stones or stone size < 5 mm [68], preferably in a small number and located in the head or body of pancreas. Complications of ESWL include pancreatitis, biliary sepsis, and gastric submucosal hemorrhage. Other methods of stone removal include Intraductal lithotripsy which is only considered as a second-line procedure [69] and chemical stone dissolution with trimethadione or other products which have not been well evaluated.

Preoperatively, unenhanced CT is recommended to map the number, size, position (head vs. body vs. tail) as well as location (main duct vs. side duct) of the stone (Fig. 10).

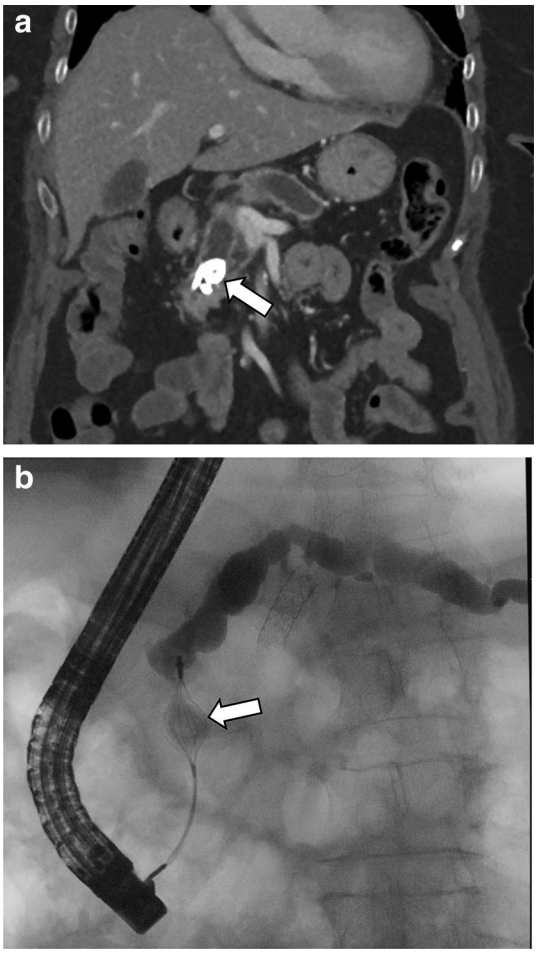

Fig. 10 Coronal contrast-enhanced CT demonstrates multiple calculi within the pancreatic duct in the head of the pancreas (arrow in **a**) with diffusely dilated main pancreatic duct and diffuse pancreatic atrophy, findings compatible with chronic pancreatitis (**a**). Endoscopic retrieval of stone was performed (arrows in **b**)

Stones with attenuation of < 820.5 HU show higher success rates on ESWL [70]. The stones are then correlated and mapped on radiographs, because ESWL is usually done under fluoroscopic guidance. Post-procedure imaging can help ensure stone clearance (fragmentation may become more evident after a few days), to detect the presence of persistent fragments occluding MPD (which may require ERCP for clearance) and to exclude complications of ESWL.

Pancreatic stricture disease

Pancreatic strictures are high-grade narrowing of MPD with upstream ductal dilation > 6 mm [68]. Dilation alone is usually unsuccessful, and the stricture is usually stented with single large (10Fr) stent or multiple stents [63, 68] (Fig. 11). Newer fully covered self-expandable metal stents have shown promise [71] but require further evaluation with larger studies. In selected cases where ERCP is unsuccessful, MPD drainage via antegrade puncture through stomach or duodenum using EUS guidance has been described. After puncture, either a pancreaticogastrostomy fistula can be created with insertion of stents or the guidewire can be advanced into the ampulla and then via a transpapillary (rendezvous) technique MPD stenting can be done [72].

Complications primarily include stent obstruction or migration (either proximal or distal). In addition, pancreatic and ductal changes resembling the lesions of CP have been reported in a high proportion of stented patients [73, 74]. It is unclear if the stent induces ductal changes or if the ductal decompression unmasks the other stenosis which was less obvious previously [75].

Secretin-enhanced MRCP can best assess hydro-dynamically significant strictures and accurately assess the length and location [76]. Malignancy needs to be excluded in the presence of main pancreatic duct stricture, especially in the absence of calcification, as CP is associated with an increased risk of pancreatic cancer. Duct-penetrating sign is useful sign in excluding malignancy on MRCP or secretin-enhanced-MRCP.

Pseudocyst management

Pseudocysts complicate 30% of patients with CP [77]. Endoscopic treatment, after 4–6 weeks of the episode of acute pancreatitis, is the preferred first-line treatment for pseudocyst, with similar long-term success when compared with surgery with lower cost, shorter hospitalization, and better quality of life [63, 68]. Spontaneous resolution of pseudocysts has been documented, especially when size < 3 cm [77]. Treatment is indicated only in symptomatic, or complicated pseudocysts (compression of large vessels, obstruction of the stomach/duodenum, MPD stenosis, pleuro-pancreatic fistula). In patients with co-existent arterial pseudoaneurysm, embolization of the aneurysm should be performed prior to drainage of pseudocysts [68].

Transpapillary/transductal drainage of cysts with stent placement can be considered for small (< 5 cm) pseudocysts communicating with MPD in the head/body of the pancreas [68]. Percutaneous drainage of pseudocysts is now largely abandoned. Most pseudocysts are preferably drained endoscopically by creating transmural cysto-enteric fistulas (duodenal fistula is preferred over gastric fistula) and placing multiple stents (Fig. 12). If a clear bulge is not identified or if the patient has portal hypertension or splenic vein thrombosis (with multiple large venous collaterals), guidance with endoscopic ultrasound (EUS) is required to safely drain the pseudocysts [78]. EUS accurately measures distance to from the lumen to the cyst, and a distance > 1 cm is considered a contraindication [79]. A nasocystic cathether can be placed if there is debris within the pseudocyst to facilitate irrigation

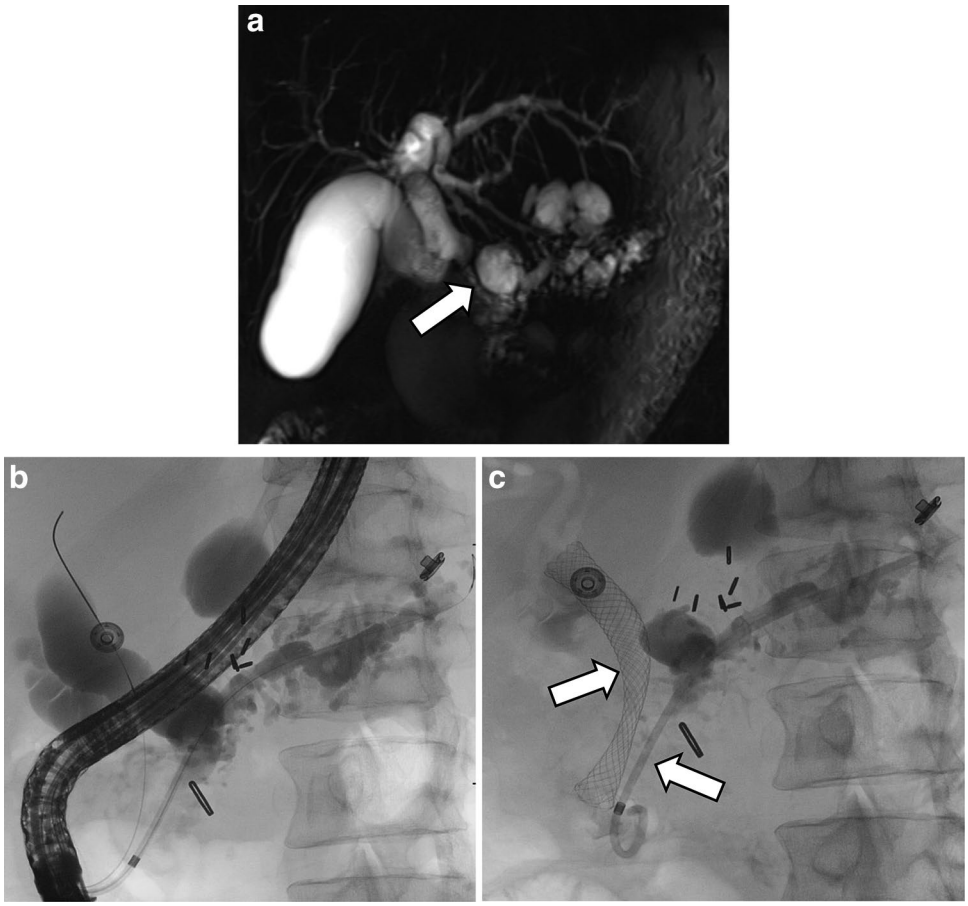

Fig. 11 Stenting of pancreatic duct stricture and biliary stricture. Coronal MRCP image in a patient with chronic pancreatitis with symptomatic biliary obstruction demonstrates diffusely dilated pancreatic duct with dilation of side branches, and dilated common bile duct secondary to chronic pancreatitis related distal CBD stricture (arrow in **a**). Coronal ERCP images (**b**, **c**) demonstrate cannulation of bile duct and pancreatic ducts with placement of stents across the biliary and pancreatic duct strictures (arrows in **c**)

of the cyst. The stents are placed for ≥ 6 weeks before they are successfully removed [68]. Removal is contraindicated if there is complete transection of the MPD (with unstented MPD), as removal of the cysto-enteric stent would inevitably result in re-accumulation from the functioning parenchyma in the tail of the pancreas [80]. Complications of endoscopic drainage include bleeding, infection, stent migration, and bowel perforation resulting in pneumoperitoneum [81]. Perforation at the site of the stenting is more common with pseudocysts in the uncinate process.

On imaging, cystic neoplasms of the pancreas should be excluded prior to decision to treat the pseudocyst. Diagnosis of pseudocyst is made according to Revised Atlanta classification guidelines [82], and needs to be differentiated from 'walled-off necrosis (WON)' as results from endoscopic drainage of WON are worse [83]. MRI is superior to CT in identification of internal non-liquefied component which differentiate pseudocyst from WON [84]. Also, MRCP can identify communication with the MPD (for planning transpapillary vs transmural drainage) as well as the presence of MPD transection (which has significant bearing on potential stent removal) [68]. The presence of bulge on stomach/duodenum and presence of large venous collaterals can be identified on cross-sectional imaging, which impact decision to use endoscopic ultrasound guidance.

Biliary strictures

Biliary obstruction complicates CP in 3–23% of patients and may be secondary to peribiliary inflammation/fibrosis or compression by pseudocyst [85]. Treatment is indicated only if the patient is symptomatic or if there is asymptomatic alkaline phosphate elevation > 2 times normal values for > 1 month [68]. Single plastic stent or uncovered metallic stents are no longer used due to high failure rate which may result in life threatening cholangitis. Multiple plastic stents or covered self-expanding metallic stents are generally used [86]. Imaging, specifically, MRI with MRCP, plays a crucial role in establishing the presence of biliary obstruction, determining the length of the stricture, and detection of associated choledocholithiais or obstructing mass. Large pseudocyst causing biliary compression that requires drainage to relieve biliary obstruction can be readily identified on MRI.

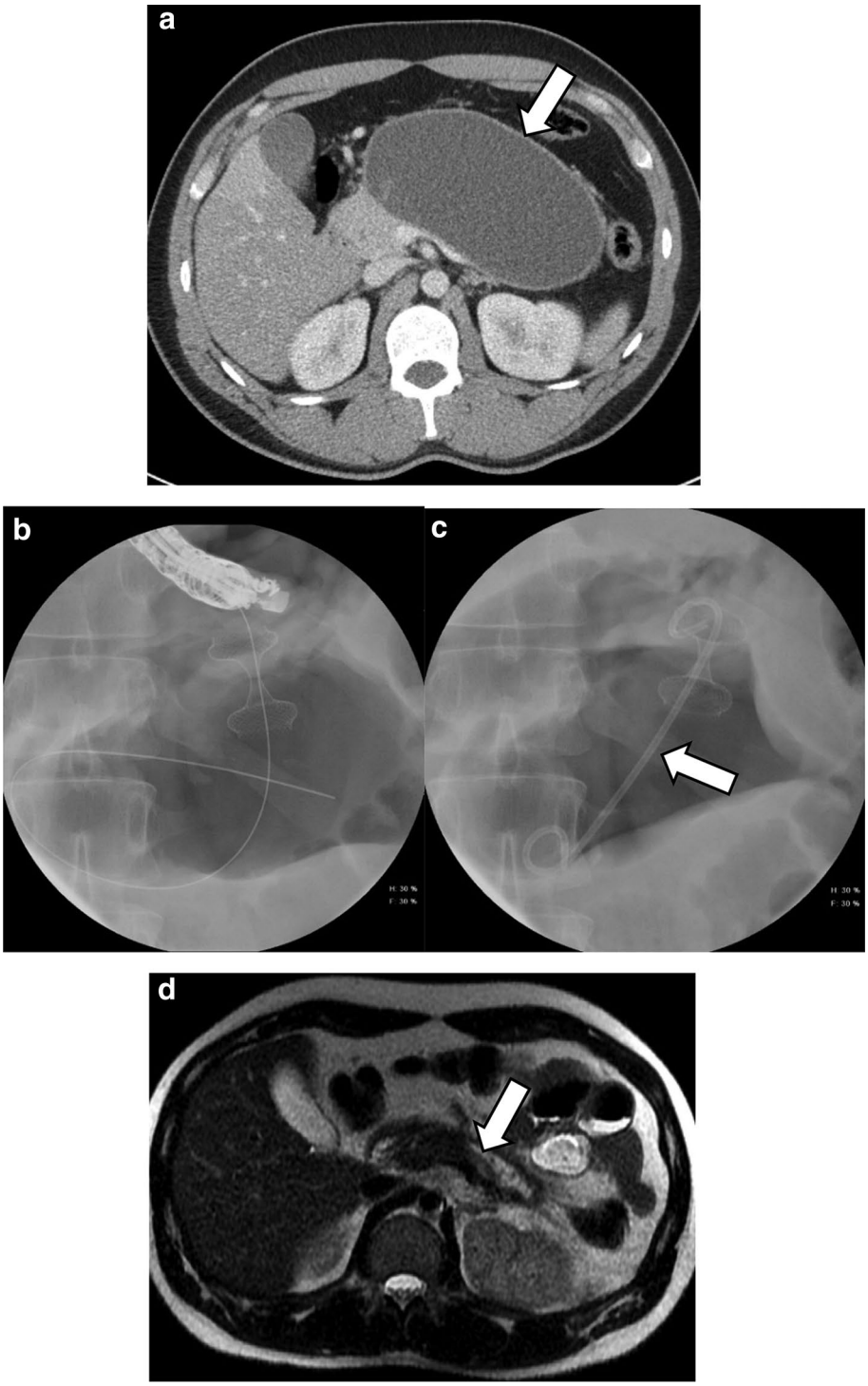

Fig. 12 Axial contrast-enhanced CT showing a large pseudocyst replacing the pancreatic body and tail (arrow in **a**). ERCP images (**b**, **c**) show endoscopic drainage of the pseudocyst with placement of a cystogastrostomy catheter (arrow in **c**). MRI obtained 6 months after the procedure demonstrates complete resolution of the pseudocyst with diffuse pancreatic atrophy compatible with chronic pancreatitis (arrow in **d**)

Celiac block/neurolysis

Pain from CP is mediated by afferent visceral fibers in celiac plexus. Imaging-guided injection of steroid temporarily inhibits pain transmission while injection of alcohol causes neurolysis. They are considered as second-line treatment for pain relief in resistant cases. Anterior approach via endoscopic ultrasound guidance is currently the preferred approach as posterior approach via ultrasound or CT guidance has propensity to be complicated by paraplegia (affecting the dura matter) or pneumothorax (affecting the pleura) [75].

References

1. Comfort MW, Gambill EE, Baggenstoss AH. Chronic relapsing pancreatitis; a study of 29 cases without associated disease of the biliary or gastrointestinal tract. Gastroenterology. 1946;6:376-408.
2. Petrov MS, Yadav D. Global epidemiology and holistic prevention of pancreatitis. Nat Rev Gastroenterol Hepatol. 2019;16(3):175-84.
3. Kleeff J, Whitcomb DC, Shimosegawa T, Esposito I, Lerch MM, Gress T, et al. Chronic pancreatitis. Nat Rev Dis Primers. 2017;3:17060.
4. Hafezi-Nejad N, Singh VK, Johnson SI, Makary MA, Hirose K, Fishman EK, et al. Surgical approaches to chronic pancreatitis: indications and imaging findings. Abdom Radiol (NY). 2016;41(10):1980-96.
5. Lowenfels AB, Maisonneuve P, Cavallini G, Ammann RW, Lankisch PG, Andersen JR, et al. Pancreatitis and the risk of pancreatic cancer. International Pancreatitis Study Group. N Engl J Med. 1993;328(20):1433–7.
6. Lowenfels AB, Maisonneuve P, Cavallini G, Ammann RW, Lankisch PG, Andersen JR, et al. Pancreatitis and the risk of pancreatic cancer. International Pancreatitis Study Group. N Engl J Med. 1993;328(20):1433-7.
7. Machicado JD, Yadav D. Epidemiology of Recurrent Acute and Chronic Pancreatitis: Similarities and Differences. Dig Dis Sci. 2017;62(7):1683-91.
8. Hoffmeister A, Mayerle J, Beglinger C, Buchler MW, Bufler P, Dathe K, et al. English language version of the S3-consensus guidelines on chronic pancreatitis: Definition, aetiology, diagnostic examinations, medical, endoscopic and surgical management of chronic pancreatitis. Z Gastroenterol. 2015;53(12):1447-95.
9. Whipple AO. THE RATIONALE OF RADICAL SURGERY FOR CANCER OF THE PANCREAS AND AMPULLARY REGION. Annals of Surgery. 1941;114(4):612-5.
10. Yamauchi FI, Ortega CD, Blasbalg R, Rocha MS, Jukemura J, Cerri GG. Multidetector CT Evaluation of the Postoperative Pancreas. RadioGraphics. 2012;32(3):743-64.
11. Ahmed Ali U, Pahlplatz JM, Nealon WH, van Goor H, Gooszen HG, Boermeester MA. Endoscopic or surgical intervention for painful obstructive chronic pancreatitis. Cochrane Database of Systematic Reviews. 2015.
12. Löhr JM, Dominguez-Munoz E, Rosendahl J, Besselink M, Mayerle J, Lerch MM, et al. United European Gastroenterology evidence-based guidelines for the diagnosis and therapy of chronic pancreatitis (HaPanEU). United European Gastroenterology Journal. 2017;5(2):153-99.
13. Cahen DL, Gouma DJ, Nio Y, Rauws EAJ, Boermeester MA, Busch OR, et al. Endoscopic versus Surgical Drainage of the Pancreatic Duct in Chronic Pancreatitis. New England Journal of Medicine. 2007;356(7):676-84.
14. Ammann RW, Akovbiantz A, Largiader F, Schueler G. Course and outcome of chronic pancreatitis. Longitudinal study of a mixed medical-surgical series of 245 patients. Gastroenterology. 1984;86(5 Pt 1):820-8.
15. Lankisch PG, Löhr-Happe A, Otto J, Creutzfeldt W. Natural course in chronic pancreatitis. Pain, exocrine and endocrine pancreatic insufficiency and prognosis of the disease. Digestion. 1993;54(3):148-55.
16. Rattner DW, Castillo CF-d, Warshaw AL. Pitfalls of distal pancreatectomy for relief of pain in chronic pancreatitis. The American Journal of Surgery. 1996;171(1):142-6.
17. Cattell RB. Anastomosis of the duct of Wirsung; its use in palliative operations for cancer of the head of the pancreas. The Surgical Clinics of North America. 1947;27(3):636-43.
18. DuVal MK. Pancreaticojejunostomy for chronic pancreatitis. Surgery. 1957;41(6):1019-28.
19. Zollinger RM, Keith LM, Ellison EH. Pancreatitis. The New England Journal of Medicine. 1954;251(13):497-502.
20. Bapat RD, Jadhav RN, Mohite JD, Rohandia OS. Modified Partington's procedure for pancreaticojejunostomy in chronic pancreatitis. Indian Journal of Gastroenterology: Official Journal of the Indian Society of Gastroenterology. 1997;16(2):54-5.
21. Kurian MS, Gagner M. Laparoscopic side-to-side pancreaticojejunostomy (Partington-Rochelle) for chronic pancreatitis. Journal of Hepato-Biliary-Pancreatic Surgery. 1999;6(4):382-6.
22. Cunha JEM, Penteado S, Jukemura J, Machado MCC, Bacchella T. Surgical and Interventional Treatment of Chronic Pancreatitis. Pancreatology. 2004;4(6):540-50.
23. Han S-L, Chen J, Zhou H-Z, Lan S-H, Zhang P-C, Zhu G-B. Indications and surgical treatment of chronic pancreatitis. Hepatobiliary & pancreatic diseases international: HBPD INT. 2008;7(6):638-42.
24. Rios GA, Adam DB, Yeoh K-G, Tamasky PR, Cunningham JT, Hawes RH. Outcome of lateral pancreaticojejunostomy in the management of chronic pancreatitis with nondilated pancreatic ducts. Journal of Gastrointestinal Surgery. 1998;2(3):223-9.
25. Isaji S. Has the Partington procedure for chronic pancreatitis become a thing of the past? A review of the evidence. Journal of Hepato-Biliary-Pancreatic Sciences. 2010;17(6):763-9.
26. Lerut JP, Gianello PR, Otte JB, Kestens PJ. Pancreaticoduodenal resection. Surgical experience and evaluation of risk factors in 103 patients. Annals of Surgery. 1984;199(4):432-7.
27. Freed KS, Paulson EK, Frederick MG, Keogan MT, Pappas TN. Abdomen after a Puestow procedure: postoperative CT appearance, complications, and potential pitfalls. Radiology. 1997;203(3):790-4.
28. Manes G, Büchler M, Piemmico O, Sebastiano PD, Malfertheiner P. Is increased pancreatic pressure related to pain in chronic pancreatitis? International Journal of Pancreatology. 1994;15(2):113-7.
29. Proca DM, Ellison EC, Hibbert D, Frankel WL. Major pancreatic resections for chronic pancreatitis. Archives of Pathology & Laboratory Medicine. 2001;125(8):1051-4.
30. Büchler M, Weihe E, Friess H, Malfertheiner P, Bockman E, Müller S, et al. Changes in peptidergic innervation in chronic pancreatitis. Pancreas. 1992;7(2):183-92.
31. Jimenez RE, Fernandez-del Castillo C, Rattner DW, Chang Y, Warshaw AL. Outcome of Pancreaticoduodenectomy With Pylorus Preservation or With Antrectomy in the Treatment of Chronic Pancreatitis. Annals of Surgery. 2000;231(3):293-300.
32. Braasch JW, Vito L, Nugent FW. Total pancreatectomy of end-stage chronic pancreatitis. Annals of Surgery. 1978;188(3):317-22.
33. Gall FP, Mühe E, Gebhardt C. Results of partial and total pancreaticoduodenectomy in 117 patients with chronic pancreatitis. World Journal of Surgery. 1981;5(2):269-73.
34. Bellin MD, Freeman ML, Gelrud A, Slivka A, Clavel A, Humar A, et al. Total pancreatectomy and islet autotransplantation in chronic pancreatitis: Recommendations from PancreasFest. Pancreatology. 2014;14(1):27-35.
35. Morgan K, Owczarski SM, Borckardt J, Madan A, Nishimura M, Adams DB. Pain Control and Quality of Life After Pancreatectomy with Islet Autotransplantation for Chronic Pancreatitis. Journal of Gastrointestinal Surgery. 2012;16(1):129-34.
36. Walsh RM, Saavedra JRA, Lentz G, Guerron AD, Scheman J, Stevens T, et al. Improved Quality of Life Following Total Pancreatectomy and Auto-islet Transplantation for Chronic Pancreatitis. Journal of Gastrointestinal Surgery. 2012;16(8):1469-77.
37. Dunderdale J, McAuliffe JC, McNeal SF, Bryant SMJ, Yancey BD, Flowers G, et al. Should Pancreatectomy with Islet Cell Autotransplantation in Patients with Chronic Alcoholic

Pancreatitis Be Abandoned? Journal of the American College of Surgeons. 2013;216(4):591-6.
38. Bramis K, Gordon-Weeks AN, Friend PJ, Bastin E, Burls A, Silva MA, et al. Systematic review of total pancreatectomy and islet autotransplantation for chronic pancreatitis. BJS. 2012;99(6):761-6.
39. Chincarini M, Zamboni GA, Pozzi Mucelli R. Major pancreatic resections: normal postoperative findings and complications. Insights into Imaging. 2018;9(2):173-87.
40. Haider M, Makary MA, Singh VK, Hirose K, Fishman EK, Zaheer A. Total pancreatectomy and islet autotransplantation for chronic pancreatitis: spectrum of postoperative CT findings. Abdominal Imaging. 2015;40(7):2411-23.
41. Mortelé KJ, Lemmerling M, de Hemptinne B, De Vos M, De Bock G, Kunnen M. Postoperative findings following the Whipple procedure: determination of prevalence and morphologic abdominal CT features. European Radiology. 2000;10(1):123-8.
42. Raman SP, Horton KM, Cameron JL, Fishman EK. CT After Pancreaticoduodenectomy: Spectrum of Normal Findings and Complications. American Journal of Roentgenology. 2013;201(1):2-13.
43. Bassi C, Dervenis C, Butturini G, Fingerhut A, Yeo C, Izbicki J, et al. Postoperative pancreatic fistula: An international study group (ISGPF) definition. Surgery. 2005;138(1):8-13.
44. Faccioli N, Foti G, Molinari E, Hermans JJ, Comai A, Talamini G, et al. Role of fistulography in evaluating pancreatic fistula after pancreaticoduodenectomy. The British Journal of Radiology. 2012;85(1011):219-24.
45. Koch M, Garden OJ, Padbury R, Rahbari NN, Adam R, Capussotti L, et al. Bile leakage after hepatobiliary and pancreatic surgery: A definition and grading of severity by the International Study Group of Liver Surgery. Surgery. 2011;149(5):680-8.
46. Cieszanowski A, Stadnik A, Lezak A, Maj E, Zieniewicz K, Rowinska-Berman K, et al. Detection of active bile leak with Gd-EOB-DTPA enhanced MR cholangiography: Comparison of 20–25min delayed and 60–180min delayed images. European Journal of Radiology. 2013;82(12):2176-82.
47. Schulick RD. Complications after pancreaticoduodenectomy: intraabdominal abscess. Journal of Hepato-Biliary-Pancreatic Surgery. 2008;15(3):252-6.
48. Lermite E, Sommacale D, Piardi T, Arnaud J-P, Sauvanet A, Dejong CHC, et al. Complications after pancreatic resection: Diagnosis, prevention and management. Clinics and Research in Hepatology and Gastroenterology. 2013;37(3):230-9.
49. Yekebas EF, Wolfram L, Cataldegirmen G, Habermann CR, Bogoevski D, Koenig AM, et al. Postpancreatectomy Hemorrhage: Diagnosis and Treatment. Annals of Surgery. 2007;246(2):269-80.
50. Wente MN, Veit JA, Bassi C, Dervenis C, Fingerhut A, Gouma DJ, et al. Postpancreatectomy hemorrhage (PPH)–An International Study Group of Pancreatic Surgery (ISGPS) definition. Surgery. 2007;142(1):20-5.
51. Aran S, Daftari Besheli L, Karcaaltincaba M, Gupta R, Flores EJ, Abujudeh HH. Applications of Dual-Energy CT in Emergency Radiology. American Journal of Roentgenology. 2014;202(4):W314-W24.
52. Smoot RL, Christein JD, Farnell MB. Durability of portal venous reconstruction following resection during pancreaticoduodenectomy. Journal of Gastrointestinal Surgery. 2006;10(10):1371-5.
53. Dixon S, Tapping CR, Walker JN, Bratby M, Anthony S, Boardman P, et al. The role of interventional radiology and imaging in pancreatic islet cell transplantation. Clinical Radiology. 2012;67(9):923-31.
54. Sutherland DER, Gruessner AC, Carlson AM, Blondet JJ, Balamurugan AN, Reigstad KF, et al. Islet Autotransplant Outcomes After Total Pancreatectomy: A Contrast to Islet Allograft Outcomes. Transplantation. 2008;86(12):1799-802.
55. Bhargava R, Senior PA, Ackerman TE, Ryan EA, Paty BW, Lakey JRT, et al. Prevalence of Hepatic Steatosis After Islet Transplantation and Its Relation to Graft Function. Diabetes. 2004;53(5):1311-7.
56. Jackson S, Mager DR, Bhargava R, Ackerman T, Imes S, Hubert G, et al. Long-term follow-up of hepatic ultrasound findings in subjects with magnetic resonance imaging defined hepatic steatosis following clinical islet transplantation. Islets. 2013;5(1):16-21.
57. Low G, Hussein N, Owen RJ, Toso C, Patel VH, Bhargava R, et al. Role of imaging in clinical islet transplantation. Radiographics. 2010;30(2):353-66.
58. Beger H, Kunz R, Poch B. The beger procedure?duodenum-preserving pancreatic head resection. Journal of Gastrointestinal Surgery. 2004;8(8):1090-7.
59. Frey CF, Amikura K. Local resection of the head of the pancreas combined with longitudinal pancreaticojejunostomy in the management of patients with chronic pancreatitis. Annals of Surgery. 1994;220(4):492-507.
60. Gloor B, Friess H, Uhl W, Büchler MW. A modified technique of the Beger and Frey procedure in patients with chronic pancreatitis. Digestive Surgery. 2001;18(1):21-5.
61. Izbicki JR, Bloechle C, Broering DC, Kuechler T, Broelsch CE. Longitudinal V-shaped excision of the ventral pancreas for small duct disease in severe chronic pancreatitis: prospective evaluation of a new surgical procedure. Annals of Surgery. 1998;227(2):213-9.
62. Cahen DL, Gouma DJ, Laramee P, Nio Y, Rauws EA, Boermeester MA, et al. Long-term outcomes of endoscopic vs surgical drainage of the pancreatic duct in patients with chronic pancreatitis. Gastroenterology. 2011;141(5):1690-5.
63. Dominguez-Munoz JE, Drewes AM, Lindkvist B, Ewald N, Czako L, Rosendahl J, et al. Recommendations from the United European Gastroenterology evidence-based guidelines for the diagnosis and therapy of chronic pancreatitis. Pancreatology. 2018;18(8):847-54.
64. Guda NM, Partington S, Freeman ML. Extracorporeal shock wave lithotripsy in the management of chronic calcific pancreatitis: a meta-analysis. JOP. 2005;6(1):6-12.
65. Moole H, Jaeger A, Bechtold ML, Forcione D, Taneja D, Puli SR. Success of Extracorporeal Shock Wave Lithotripsy in Chronic Calcific Pancreatitis Management: A Meta-Analysis and Systematic Review. Pancreas. 2016;45(5):651-8.
66. Nguyen-Tang T, Dumonceau JM. Endoscopic treatment in chronic pancreatitis, timing, duration and type of intervention. Best Pract Res Clin Gastroenterol. 2010;24(3):281-98.
67. Choi EK, McHenry L, Watkins JL, Sherman S, Fogel EL, Cote GA, et al. Use of intravenous secretin during extracorporeal shock wave lithotripsy to facilitate endoscopic clearance of pancreatic duct stones. Pancreatology. 2012;12(3):272-5.
68. Dumonceau JM, Delhaye M, Tringali A, et al. (2019) Endoscopic treatment of chronic pancreatitis: European Society of Gastrointestinal Endoscopy (ESGE) Guideline - Updated August 2018. Endoscopy. 51(2):179–193
69. Beyna T, Neuhaus H, Gerges C. Endoscopic treatment of pancreatic duct stones under direct vision: Revolution or resignation? Systematic review. Dig Endosc. 2018;30(1):29-37.
70. Ohyama H, Mikata R, Ishihara T, Tsuyuguchi T, Sakai Y, Sugiyama H, et al. Efficacy of stone density on noncontrast computed tomography in predicting the outcome of extracorporeal shock wave lithotripsy for patients with pancreatic stones. Pancreas. 2015;44(3):422-8.
71. Shen Y, Liu M, Chen M, Li Y, Lu Y, Zou X. Covered metal stent or multiple plastic stents for refractory pancreatic ductal strictures in chronic pancreatitis: a systematic review. Pancreatology. 2014;14(2):87-90.

72. Deviere J. EUS-guided pancreatic duct drainage: a rare indication in need of prospective evidence. Gastrointest Endosc. 2017;85(1):178-80.
73. Bakman Y, Freeman ML. Pancreatic Stent-Induced Ductal Injury: Clinical Presentation and Outcomes of Endoscopic Therapy. Gastrointestinal Endoscopy. 2009;69(5):AB125.
74. Freeman ML. Pancreatic Stents for Prevention of Post–Endoscopic Retrograde Cholangiopancreatography Pancreatitis. Clinical Gastroenterology and Hepatology. 2007;5(11):1354-65.
75. Seicean A, Vultur S. Endoscopic therapy in chronic pancreatitis: current perspectives. Clin Exp Gastroenterol. 2015;8:1-11.
76. Manfredi R, Pozzi Mucelli R. Secretin-enhanced MR Imaging of the Pancreas. Radiology. 2016;279(1):29-43.
77. Andren-Sandberg A, Dervenis C. Pancreatic pseudocysts in the 21st century. Part I: classification, pathophysiology, anatomic considerations and treatment. JOP. 2004;5(1):8-24.
78. Panamonta N, Ngamruengphong S, Kijsirichareanchai K, Nugent K, Rakvit A. Endoscopic ultrasound-guided versus conventional transmural techniques have comparable treatment outcomes in draining pancreatic pseudocysts. Eur J Gastroenterol Hepatol. 2012;24(12):1355-62.
79. Fockens P. EUS in drainage of pancreatic pseudocysts. Gastrointest Endosc. 2002;56(4 Suppl):S93-7.
80. Arvanitakis M, Delhaye M, Bali MA, Matos C, De Maertelaer V, Le Moine O, et al. Pancreatic-fluid collections: a randomized controlled trial regarding stent removal after endoscopic transmural drainage. Gastrointest Endosc. 2007;65(4):609-19.
81. Varadarajulu S, Christein JD, Wilcox CM. Frequency of complications during EUS-guided drainage of pancreatic fluid collections in 148 consecutive patients. J Gastroenterol Hepatol. 2011;26(10):1504-8.
82. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62(1):102-11.
83. Baron TH, Harewood GC, Morgan DE, Yates MR. Outcome differences after endoscopic drainage of pancreatic necrosis, acute pancreatic pseudocysts, and chronic pancreatic pseudocysts. Gastrointestinal Endoscopy. 2002;56(1):7-17.
84. Kamal A, Singh VK, Akshintala VS, Kawamoto S, Tsai S, Haider M, et al. CT and MRI assessment of symptomatic organized pancreatic fluid collections and pancreatic duct disruption: an interreader variability study using the revised Atlanta classification 2012. Abdom Imaging. 2015;40(6):1608-16.
85. Abdallah AA, Krige JE, Bornman PC. Biliary tract obstruction in chronic pancreatitis. HPB (Oxford). 2007;9(6):421-8.
86. Regimbeau JM, Fuks D, Bartoli E, Fumery M, Hanes A, Yzet T, et al. A comparative study of surgery and endoscopy for the treatment of bile duct stricture in patients with chronic pancreatitis. Surg Endosc. 2012;26(10):2902-8.