

Postoperative Pancreatic Biliary Surgical Complications

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ABSTRACT

This review tries to trace the diagnosis and management of complications after surgery of biliary tract and pancreas that emergency care physicians can encounter. These specific complications, relevant after hospital discharge, have a wide range of incidence, from the lowest one following simple cholecystectomy to the highest one after difficult re-hepatectomies, transplants or pancreatic cancer resections. Although we separate those complications as they come from biliary tract or pancreatic surgery, many of them are shared since several surgical procedures involve more than one of these anatomical areas at the same time.

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Key words: Biliary tract surgery complications; Biliary fistulas; Biliary stenosis; Pancreatic surgery complications; Delayed gastric emptying; Pancreatic fistulas

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INTRODUCTION

This chapter is conceived to provide information regarding the diagnosis and management of specific complications after surgery of biliary tract and pancreas that emergency care physicians can encounter. Besides medical and surgical complications common to

any major surgical procedures, which will not be addressed here, a number of specific complications, unique to pancreatobiliary surgery, can be faced by emergency care physicians since these may be clinically relevant after hospital discharge. These complications show a wide range of incidence, from the lowest one following simple cholecystectomy to the highest one after difficult re-hepatectomies, transplants or pancreatic cancer resections. In this review we separate those complications as they come from biliary tract or pancreatic surgery though, because of several procedures involve more than one of these anatomical areas at the same time, many of the complications are accordingly shared.

As a consensus on how to define and grade postoperative complications does not exist yet, hindering comparison of outcome data among different centers and therapies and over time, we here favour the classification proposed by Clavien, based on the therapy used to treat the complication^[1].

COMPLICATIONS IN BILIARY TRACT SURGERY

Diseases of the biliary system can be extremely painful, debilitating, and occasionally life threatening. An absolute knowledge of the anatomic variations of this system with careful dissection and identification of structures at the time of surgery is a minimal requirement for the safe performance of any hepatobiliary operation^[2]. Most of these surgical complications are related to iatrogenic injuries and include bile leak, bile duct obstruction or stricture and infection.

Prevalence of gallstones is 5% to 22% in the Western world, with a 10%-40% of them being symptomatic. Laparoscopic cholecystectomy is associated with a higher incidence of biliary injuries relative to the open procedure (0.2% and 0.8% respectively)^[3-8]. Less than one third of bile duct injuries (15%-30%) are recognized during surgery^[2,9]. These injuries can result in long-term morbidity, recurrent hospital admissions, multiple radiological and surgical interventions, costly litigation, and mortality.

Different mechanisms of injury have been described but two major groups of errors are identified: misidentification of the anatomy of the biliary tract (around 70%) and technical errors leading to bleeding and subsequent clipping of the bile duct/artery or to bile leakage^[10,11]. These injuries have deserved the development of various classifications^[12-16] and the description of some techniques and maneuvers addressed to decrease their incidence^[17,18].

Injuries can present during the operative procedure, a few days

after surgery or weeks or months or even years afterward. Few days after surgery patients have biliary cutaneous-fistula, jaundice, signs of acute abdomen, and sepsis. Injuries that present after weeks or months are usually lesser injuries that preserve biliary function yet heal with stricture formation and patients complaint of recurring fever and rigors indicative of cholangitis. Fatigue and weight loss are common and jaundice is usually not present with sectorial or lobular ductal ligation. Injuries from procedures other than cholecystectomy follow the same guidelines.

Laboratory tests may show hyperbilirubinemia after a few days. Direct bilirubinemia predominates in cases of retained stones, clipped common duct or biliary stenosis. Leucocytosis also may be present^[19]. Elevations of hepatic transaminases, alkaline phosphatase, and gamma-glutamyl transpeptidase may also be pertinent findings in biliary obstruction but are much less prominent in biliary leaks. Laboratory findings, however, may be nonspecific.

Imaging studies are common to most of these complications. The first step in the diagnostic work-up of patients with abdominal complaints after biliopancreatic operations is ultrasonography (US) which is a fast, inexpensive, and noninvasive method for detecting fluid collections, duct dilatations and gross modifications of hepatic or pancreatic parenchyma^[20]. However, this procedure cannot characterize the fluid as a bile collection, a hematoma, a seroma, or a lymphocele. Its effectiveness is limited by abundant bowel gas, which may accompany cases of ileus and obesity. It also facilitates percutaneous aspiration, useful to establish the diagnosis and frequently to place drain. Although it can detect a dilatation of bile ducts and even of pancreatic duct, providing indirect evidence of a stricture, is less accurate for determining the etiology and level of obstruction.

Magnetic resonance cholangiopancreatography (MRCP) is a very useful tool for visualizing the pancreatobiliary ductal system and is considered the most effective imaging technique in showing biliary complications^[21]. Biliary dilation can be accurately detected by MRCP in 97-100% of patients and the level of obstruction is correct in almost 87% of cases^[20,22]. MRCP provides a viable alternative to endoscopic retrograde cholangiopancreatography (ERCP) although, unlike ERCP, it is not therapeutic. Since its introduction, MRCP has rapidly become an important tool for visualizing the biliary system.

Computed tomography (CT) scan may identify biloma, hepatic or pancreatic abscess formation, pancreatic necrosis and atrophy or hypertrophy of the hemi-liver. Although CT easily detects and localizes fluid collections, cannot characterize them. CT should not be the first choice for diagnosing bile duct injuries because CT does not demonstrate details of the biliary tree as well as US does. Obviously, CT can also be used for guided percutaneous aspiration and drainage.

Hepatobiliary scintigraphy with technetium 99 m-labelled hepatic iminodiacetic acid (HIDA) is a safe, highly sensitive, and noninvasive detector of biliary leaks. It is accurate in 83% to 87% of these cases and is more sensitive and specific than CT or US^[19]. When imaging is delayed to 60-90 min, the sensitivity of this diagnostic method is further increased. This procedure can be performed in cases of hyperbilirubinemia. The entry of the agent into the duodenum is an indication of the continuity of the biliary tract and is often used as the first test to exclude a complete transection of the bile ducts (but it can not exclude a minor bile duct injury). It can help determine the clearance of bile across strictures and surgical anastomosis and is also useful for distinguishing cholangitis from cholecystitis. However, HIDA scan is insensitive for helping detect biliary dilatation or the site and cause of bile duct obstruction.

Therefore, because injury may occur anywhere in the biliary tree or even in the small bowel, HIDA is not regularly used to detect injuries and other diagnostic studies are needed to track down the exact location of a leak.

ERCP is required to delineate the ductal system, identify abnormalities and locate its level. It has been regarded as the nonoperative gold standard for biliary imaging until recently. Therapeutic maneuvers such as sphincterotomy, stone extraction, and biliary stent placement may be performed. ERCP can easily detect duct leaks and lacerations. In cases of transections of the biliary tree, however, ERCP may not allow visualization of proximal anatomy, and complete delineation of the injury may not be possible. Attempts at repair of a biliary injury may be ineffective when preoperative cholangiograms are either not performed or incomplete^[23]. The success rate of ERCP is 90-95%, with a complication rate of approximately 3-5%. Besides that, it can collect bile samples or brushings of the ducts.

Percutaneous transhepatic cholangiography (PTC) is the method of choice for most cases of bile duct lesions although ERCP may be easier to obtain in an emergency environment. PTC is an excellent method for illustrating the anatomy of the proximal biliary tree. Furthermore, temporary therapeutic procedures, such as placing drainage catheters and stents, can be performed using this approach as a bridge before definitive repair. Later, these catheters can serve as guides during operative dissection for definitive repair procedures. The success rate of PTC approaches 100% when ducts are dilated. Complications can occur in as many as 10% of the cases.

In patients with an external fistula or a biliary T tube, contrast medium can be injected into the fistula or the biliary system through the tube, which delineates the anatomy of the fistula and the site of stricture. Arteriography may be considered in the preoperative evaluation of patients with suspected vascular complications, although helical CT or MRI angiography can afford enough information. If tumour is suspected, a positron emission tomography (PET) may help distinguish a benign lesion from a new malignant one or a tumour recurrence.

The tenets of initial management are control of sepsis and physiological support followed by definition of anatomy, drainage, and finally repairs. Intra-abdominal sepsis, dehydration, electrolyte depletion, and malnutrition must be addressed on initial presentation, because attempts to repair injuries in suboptimal physiologic conditions can be counterproductive. Around 70%-80% of patients respond to medical therapy and do not need urgent intervention. Patients without a response to empiric antibiotic therapy within 24 h should be considered for immediate biliary decompression, which can be performed surgically, percutaneously, or endoscopically. The timing of repair is an important determinant of long-term outcome. Since the intermediate period is significantly associated with biliary strictures, repairs must be undertaken either in the immediate (0-72 h) or delayed (more than 6 weeks) periods after initial surgery^[24].

Morbidity rate after bile duct reconstruction is 43% but most complications are minor. Mortality rates ranging from 1.7% to 9% have also been reported. Postoperative stricture formation with long-term follow-up occurs in 10% to 19% of patients. Endoscopic or percutaneous decompression is often associated with lower morbidity and should be considered first. The rate of chronic liver disease following bile duct repair has been reported to be between 6% and 22%^[2]. Despite this increased morbidity, need for long-term follow-up, and potential for litigation surrounding bile duct injuries, the rate of successful repair of these injuries in the hands of experienced hepatobiliary surgeons may be greater than 90%^[25].

Biliary leaks

Postoperative bile leaks may occur from any portion of the biliary tract. After cholecystectomy, the Luschka ducts, which arise from the gallbladder bed, are occasionally a source. Biliary leaks may also arise from the cystic duct stump, the common bile duct, hepatic duct lacerations or transections, or small bowel injury. Cystic duct leaks generally occur after inadequate ligation or slippage of clips, although they can also be caused by retained stones or by an injury distal to the clips or ligation. Accumulation of free bile usually occurs in the subhepatic space. Depending on the severity and rate of the leak and the length of time before it is detected, bile may spread freely throughout the peritoneal cavity (bile peritonitis) or may be loculated (biloma). Obviously, bile can flow externally through a drain or a surgical wound. A biloma can exist for a long period of time before symptoms occur.

The early symptoms may be nonspecific and consist of general malaise, nausea, vomiting, prolonged ileus, anorexia, abdominal pain and low-grade fever, leading to a delay in diagnosis. Patients are rarely clinically jaundiced in the immediate postoperative period, but an elevation of bilirubin levels is commonly seen. There is not usually a consistent pattern of direct or indirect hyperbilirubinemia in cases of free bile leakage into the abdomen. Sepsis and jaundice can appear later.

US can identify fluid collections and is useful to aspirate them and to leave a drain if required. It must be noticed that a small fluid collection can be observed in the gallbladder fossa in 10% to 28% of postoperated patients. CT scan may also detect or exclude an intra-abdominal collection. A HIDA scan is useful in confirming the presence of bile extravasation and, chiefly, in assessing the continuity of the biliary tract. Nuclear activity may be apparent in the subhepatic area, right paracolic gutter, or diffusely in the peritoneal cavity. On occasion, a fistulogram can be obtained through the drainage tube or the fistula tract that may delineate the biliary tree and the level of the lesion. Definitive diagnosis is usually made by ERCP or PTC.

A minor leak from an accessory hepatic duct or cystic duct is likely to heal spontaneously and merely requires placement of a percutaneous catheter under CT or US guidance. Otherwise, treatment usually involves reducing the pressure on the proximal duct by placing a drain in the common duct through the ampulla via ERCP or through the liver via PTC. Whether it is a short or a nasobiliary stent does not appear to influence the outcome. These stents are left in place for six to eight weeks. Surgical intervention is rarely necessary. Cystic duct leaks can be managed with or without sphincterotomy, but sphincterotomy alone without stent placement can result in prolonged bile leakage and delayed healing^[26].

Persistent collections from other sources may require open drainage techniques^[27]. Patients with signs of peritonitis, sepsis, or any other clinical suspicion of biloma should have parenteral antibiotics and percutaneous drainage of any fluid collections. If signs and symptoms of cholangitis are present an urgent cholangiogram with biliary drainage is needed. If major bile duct injury is detected, repair should be undertaken after control of the bile leakage and treatment of sepsis. Surgery can be postponed for four to six weeks, provided that adequate drainage has been obtained. Surgical repair is bilio-enteric anastomosis, mainly Roux-en-Y hepaticojejunostomy, since primary anastomosis and repair of common duct injuries over a T-tube is associated with a high failure rate.

Biliary strictures

Patients with bile duct strictures, as opposed to bile leaks, usually

present within 30 days of their initial operation. One-quarter of these patients present with painless obstructive jaundice alone. Up to half present with fever and sepsis, while a few present with an external bile leak. Patients with total occlusion by clipping of the common bile duct show early obstructive jaundice frequently followed after one or two weeks by biliary leakage and biliary peritonitis due to the increasing intraductal pressure and subsequent leakage at the site of the clips. Some patients with presumed ischemic or thermal injury may not present for several months or years after the initial surgery, when cholangitis or jaundice secondary to the stricture prevails. If a sectorial duct has been ligated, it may cause asymptomatic atrophy of the liver sector but also fever and liver abscess if bile becomes infected.

Postoperative biliary strictures are the most common indication for reoperation following biliary tract surgery. Eighty per cent of benign biliary strictures are the result of a bile duct injury during laparoscopic cholecystectomy. Less frequent benign postoperative biliary strictures occur after bile duct reconstruction, tumour resection or liver transplantation. Inappropriate treatment may result in biliary cirrhosis or cholangitis. In a long-term review of 130 patients with postoperative biliary strictures, mortality (17.7%) was related to the presence of liver parenchymal disease with portal hypertension in all cases^[28].

US can discover a bile duct dilatation or changes in the hepatic parenchyma. This dilatation of intrahepatic and extrahepatic bile ducts provides indirect evidence of a bile duct stricture, but US is not very accurate for determining the etiology and level of obstruction. Its sensitivity also depends on the degree of obstruction, which is high (more than 90%) with a serum bilirubin level of more than 10 mg/dL but only 47% with bilirubin levels of less than 10 mg/dL. CT scan may detect a biloma, a hepatic abscess formation, and an atrophy or hypertrophy of the hemi-liver. It can be as sensitive as US to detect biliary obstruction but can identify the site with greater accuracy than US. A HIDA scan may help determine the clearance of bile across strictures and surgical anastomosis but is useless for detecting biliary dilatation or the site of bile duct obstruction, as stated above.

MRCP is very useful to establish the features of the obstruction, but the definitive diagnosis is made by cholangiography. It is required to establish the ductal stricture, identify its level and recognize the nature of the injury. ERCP is only useful in cases of bile duct continuity. If the stricture is too tight to go across with ERCP, PTC may be performed for proximal biliary decompression. Indications for PTC in bile duct strictures are the presence of biliary-enteric anastomosis, the presence of complex hilar strictures, or an unsuccessful ERCP. It allows biliary drainage, dilation of strictures, extraction of stones if present, or placement of a stent across a malignant stricture.

The diagnostic yield of EUS combined with FNA is good, especially in distal bile duct strictures, and may surpasses ERCP with brushings when a suspicion of cancer arises^[29]. PET may be also of value in this setting.

Angiography could be considered since an injury to the hepatic artery is present in 7%-32% of the patients with bile duct injury after cholecystectomy^[2,30], which results in slow hepatic infarction in about 10% of the cases^[31]. Patients with Bismuth level IV and V injuries or patients with failed primary biliary-enteric repairs should have a preoperative angiography^[32]. Injuries involving the portal vein or common or proper hepatic arteries are much less common, but have more serious effects including rapid infarction of the liver. Repair of the artery is rarely possible and the overall benefit unclear, although the presence of a right hepatic artery disruption does not preclude the

bile duct repair^[30].

The management depends on the degree of injury, the presence of induced complications and the operative risk of the patient. The biloma must be drained and the infection (abscess or cholangitis) controlled, as outlined above. A definitive therapy with surgical reconstruction or stenting must be planned after cholangiography, although an immediate repair of the bile duct can yield good results if properly performed. Previous cholangitis, incomplete cholangiography or early primary repair (within 3 weeks of the injury) can lead to stricture recurrence.

PTC is useful when there is intrahepatic ductal disease or ERCP is not possible, and to dilate anastomotic strictures or assist in the intraoperative identification of the proximal bile duct. It is more successful in cases of bilioenteric anastomotic stenosis (up to 92%) than in those of ischemic strictures (61%)^[33]. Moreover, multiple sessions of dilation are often required to achieve long-term success rates. Endoscopic dilation has similar efficacy treating extrahepatic stenosis causing clinical symptoms. This procedure also requires repeated sessions and achieves better results with nonischemic strictures. The more common complications include hemobilia, bile leak, pancreatitis, and cholangitis.

Stent placement is associated with complications such as stent occlusion and subsequent cholangitis. This, along with strictures as the time from the initial dilation increases, limits the role of endoscopic therapy. Attempts to place multiple plastic stents across the stricture to permit greater stricture dilation over a longer period of time, preventing restenosis when the stents are removed, seem to yield good results^[29]. Open-mesh metal stents are associated with several flaws, including occlusion, stone formation, and lack of permanency. The use of covered metal stents needs further evaluation. Self-expanding stents made of bioabsorbable material, which can also be impregnated with antimicrobial and antineoplastic agents are promising but remains investigational at the present time^[34,35]. Endoscopic access to the dilated and/or infected proximal bile duct through puncture, dilatation and stenting from the distal duct or stomach, guided by EUS, is a helpful maneuver. Endoscopic and radiologic techniques for dilatation and stenting are gradually coming close results of those of the surgical techniques. They are a good treatment option for patients unfit for surgery and for palliative treatment in patients with unresectable or recurrent biliary tract tumours.

Successful surgical repair of biliary strictures requires the use of proximal bile duct with minimal inflammation, the creation of a tension-free anastomosis with a Roux-en-Y jejunal limb, and a direct mucosa-to-mucosa anastomosis. Although an end-to-end bile duct anastomosis was previously encouraged, this repair is associated with a 40% to 50% long-term failure rate^[36]. Direct biliary-enteric bypass is the gold standard procedure for the long-term treatment of biliary strictures^[37-40]. In some cases, liver resection^[41] or transplantation may be required.

Patients with strictures and liver dysfunction should have an evaluation of portal hypertension. Endoscopic examination may identify the presence of esophageal varices and a liver biopsy can reveal the presence of biliary fibrosis. The presence of esophageal varices or ascites is ominous and should preclude repair^[2].

Not all late bile duct injuries require intervention. Some patients may remain entirely asymptomatic, the injury being diagnosed by an accidental abdominal US or blood test showing abnormal liver function tests. If the injury has caused atrophy of part of the liver without resulting in sepsis or cholangitis, the patient may merely be observed.

Unretrieved stones in the peritoneal cavity

During mobilization of the gallbladder from the liver and again during extraction, perforation of the gallbladder is common, occurring in 10% to 40% of laparoscopic cholecystectomies, with the possibility of stone spillage and loss. The rate of stone loss is unknown. The risk of perforation is increased in acute cholecystitis, previous cholecystectomy and gallbladder hydrops. The laparoscopic approach makes retrieval of dropped stones difficult. Even though a number of reports have suggested that unretrieved stones can cause abscess, inflammation, fibrosis, adhesions, cutaneous sinuses, small bowel obstruction, and perforation into the intestine, uterus, and even the thorax, or generalized sepsis, for the majority of the patients this is of no consequence because the overall risk of serious complication is low (0.6%-2.9%)^[21,42].

As many stones should be retrieved as possible, without converting to an open procedure, and the abdomen should be irrigated copiously prior to completing the procedure. If an intra-abdominal abscess develops, percutaneous drainage and prolonged antibiotic administration will usually be successful. Percutaneous intubation of the abscess cavity and stone retrieval has even been performed in some cases. Repeat drainage and antibiotics can treat a single recurrence, but a third recurrence should lead to surgical intervention^[26].

Choledocholithiasis following biliary surgery

After cholecystectomy around 1% to 2% of patients will have retained stones in the common bile duct requiring further intervention, usually with nonoperative methods, to avoid biliary stasis and cholangitis. Diagnosis is established by US but there is no assurance of the reliability of negative findings. In these cases MRCP is the imaging modality of choice. EUS with the probe located in the duodenal bulb can show common duct stones as small as 2 mm^[43].

ERCP with sphincterotomy is the preferred initial procedure for the extraction of recurrent or retained bile duct stones^[44-46], reporting a success rate of more than 85%. Peroral cholangioscopy can visualize choledocholithiasis in 90% of cases and the scope permits passage of an electrohydraulic lithotripsy probe or holmium laser for therapeutic purposes although experience is still limited^[32]. If this methods fails or is technically impossible because of previous biliary enterostomy, percutaneous methods may be attempted. If these methods fail, operative re-exploration is necessary to clear the common duct of stones and ensure free distal ductal drainage. Since simple common bile duct exploration has a high failure rate, a transduodenal sphincteroplasty must be performed when there is no proximal biliary stricture and the common bile duct is less than 1.5 cm in diameter^[47]. In cases of stone formation secondary to stricture or inflammation of the bile ducts, choledochoduodenostomy, Roux-en-Y choledochojejunostomy or, preferably, Roux-en-Y hepatojejunostomy must be constructed.

Postoperative pancreatitis

Postoperative pancreatitis arise in 1% to 3% of patients who have operations in the vicinity of the pancreas though it may appear after renal, parathyroid or cardiac surgery, among others. It has a higher complication rate than pancreatitis associated with other etiologies. The mechanism is unclear. It is related to a delayed gastric emptying after duodenopancreatectomy^[48]. Biliary surgery particularly tends to precipitate this complication. Pancreatitis may occur after the passage of common duct stones originating from the gallbladder or after direct instrumentation with probes, catheters, choledoscopes, and other instruments used for common bile duct exploration. Its incidence after laparoscopic cholecystectomy is 1%-4%. Acute

pancreatitis may occur even after pancreatic biopsy (1%)^[49].

The diagnosis is difficult to confirm. CT scan and an elevated amylasemia, blood leukocyte count and serum C-reactive protein concentration may suggest its existence. MRI is a viable alternative to CT. Treatment is usually conservative, but bleeding or infection may ensue requiring surgery, with necrosectomy or a completion total pancreatectomy in some cases.

Port site metastasis after laparoscopic cholecystectomy

Metastatic spread of gallbladder carcinoma is an unexpected and unusual complication of laparoscopic cholecystectomy. Ideally, gallbladder carcinoma would always be diagnosed preoperatively and at an early stage of invasion, and an open cholecystectomy with an appropriate negative margin and lymphadenectomy would then be performed without violation of the gallbladder wall. In reality, however, gallbladder carcinoma is diagnosed preoperatively in only one-third of cases.

Metastasis may develop rapidly at the extraction port, appearing as umbilical incisional hernias. Although the extraction port is more often the site of recurrence, other ports are certainly not immune. The rate of port site metastases in one prospective study was 14% of patients with carcinoma who had undergone laparoscopic cholecystectomy, representing 0.05% of all^[42]. When gallbladder carcinoma is diagnosed following laparoscopic cholecystectomy, wide excision of all port sites is recommended, although it has not been shown that this will increase survival or disease-free interval.

Biliary complications following liver transplantation

Biliary complications occur frequently (4% to 29%) after liver transplantation (LT) and its incidence may be even higher after living donor liver transplantation (LDLT) reaching up to 65%^[50]. In fact, biliary tract complications are the most common postoperative complications following LDLT. They occur in 7.5% of the donors and are more frequent after right lobe graft than after left lobe graft^[51]. Overall complications rate in the right lobe adult living donor is 29%, with a few donors needing liver transplantation due to hepatic failure donation. Aborted donor hepatectomy is a major complication estimated to occur in 1% to 5% of cases^[52]. The diagnosis of biliary anastomotic stenosis can be established using US, CT scan and MRI, as well as biliary excretion scintigraphy for doubtful cases.

Most of the cases (up to 95% in some series) resolve with conservative treatment^[53]. Patients have endoscopic retrograde cholangiography (ERC) first. If it failed to handle the problem, PTC is done. To treat biliary leaks, nasobiliary drainage is placed for several weeks. If a biliary stricture persists after the leakage resolved, an endoprosthesis is inserted. If the stricture persists after several months, the stenosis is dilated and a larger endoprosthesis is inserted. Surgery is needed for the unsolved leakages and strictures^[51,54]. Intrahepatic biliary strictures are much more difficult to treat, and up to 63% of these patients may require liver resection or retransplantation^[55].

Posttransplantation biliary stones are usually managed with endoscopic or operative techniques similar to those for retained stones. Despite the lack of randomized controlled trials, endoscopy has become the first line therapy for post-liver transplant anastomotic strictures and distal post-operative strictures^[29].

COMPLICATIONS IN PANCREATIC SURGERY

Surgical diseases of the pancreas are often more difficult to treat than those of other abdominal viscera. The difficult location of

the pancreas gland, with its intimate association with other vital structures that surround it, add to the complexity of pancreatic surgery. Resection of the pancreas is considered a major operative procedure and, although mortality rates over the last two decades are less than 5%, morbidity rates remain high (30%-60%)^[56-60]. Whilst the majority of perioperative complications are not life-threatening, with less than 10% requiring reoperation^[50], they can, however, amount to increased lengths of stay and costs, readmissions for care and, for cancer patients, delays in adjuvant therapy^[62,63].

The most common causes of postoperative morbidity after pancreatoduodenectomy are delayed gastric emptying (8%-45%), pancreatic fistula/leak (3%-30%), hemorrhage (2%-16%), intra-abdominal abscess (1%-14%), wound infection (5%-10%), other infections (3%-5%) and biliary complications (3%-9%)^[64]. Other study with 283 patients found that 16.6% of the patients were readmitted for surgical complications not related to tumour recurrence, mainly for abscesses, fistula and intestinal obstruction^[65]. The readmission rate during a 10-year period at Massachusetts General Hospital was 9.6% for dehydration, failure to thrive, wound infection, intra-abdominal abscesses and gastrointestinal bleeding^[60]. Fourteen per cent of the readmissions were due to surgery-related complications in other study^[66].

Ultrasound examination may be used to detect peritoneal fluid in the early post-operative period as well as lesion recurrence in long-term follow-up. Radiological gastrointestinal studies has a major role in evaluation of intestinal functionality. CT is the most effective imaging technique after pancreatic surgery since it may demonstrate early and late surgical complications. MRI may be used as alternative imaging modality to CT, when renal insufficiency or contrast sensitivity prevents the use of iodinated intravenous contrast material or when the biliary tree study is primarily requested^[67]. Occlusion or disruption of the pancreatic duct and pseudocysts may be readily visualized with MRCP^[68].

Perioperative octreotide for decreasing the incidence of postoperative complications is recommended for routine use in patients undergoing pancreatic resection for cancer, but not for other reasons^[69].

Delayed Gastric Emptying

Delayed gastric emptying is a frequent and significant postoperative problem following pancreatoduodenectomy. In most series, delayed gastric emptying, defined as the need for postoperative nasogastric decompression for more than 10 days postoperatively, has a reported incidence ranging from 20% to 40%. Patients without any complications have a delayed gastric emptying rate of less than 1%, but this increases to 28% and 43% in the presence of moderate and severe postoperative complications. Although not life-threatening, delayed gastric emptying results in a significant prolongation of hospital stay and contributes to increased hospitalization costs. The etiology is uncertain, but possible causes include decreased motilin levels, removal of the duodenal pacemaker, and disruption of gastroduodenal neural connections. Erythromycin, a motilin agonist, has been found to improve gastric emptying of both solids and liquids when administered intravenously during the postoperative period.

The treatment of delayed gastric emptying is nonoperative. After ruling out a mechanical obstruction or an intraabdominal, the treatment include bowel rest, gastric decompression, enteral feeding via a jejunostomy tube or a nasojejunal feeding tube, and/or parenteral nutrition. Somatostatin has been shown to suppress plasma motilin level and worsen gastric emptying in a few small studies^[64]. Metoclopramide and erythromycin are commonly used in

the treatment of delayed gastric emptying. Erythromycin, a motilin agonist, has been found to reduce the incidence of this complication by 37-75% versus controls in randomized prospective trials^[70]. Very few cases require reoperation^[71].

Pancreatic fistulas

Pancreatic fistulas, usually resulting from an anastomotic leak, are an important cause of morbidity and mortality following surgery in which a pancreas remnant is left. This serious complication also occurs with operations involving the stomach, spleen, colon, adrenal gland or kidney. There is still no consensus on a uniform definition of pancreatic fistula^[72], but is usually admitted as such a persistent drainage of 50 mL or more per day of amylase-rich fluid (more than 5000 units) on or after the tenth postoperative day 56 or, alternatively, any volume of more than three times the serum amylase appearing on or after the third day^[73]. The incidence of pancreatic anastomotic leak after pancreatic resection varies from 5% to 25% in most series, regardless of the type of anastomosis and of the use of perioperative octreotide^[74]. Its incidence appears to be higher in laparoscopic surgery than in the reported open series^[75]. Pancreatic fistula rate in chronic pancreatitis is 5%, whilst that for the same procedure in pancreatic, ampullary or bile duct cancer is 12% to 33%^[56]. Following necrosectomy the fistula rate is even higher (3%-72%)^[76]. After pancreatic surgery for trauma, the incidence of pancreatic leak is 60% compared to 11% for patients who had elective distal pancreatectomy. Fortunately, most of these fistulae heal with external drainage and seem to have fewer propensities to cause further complications.

Most leaks may run a benign course, requiring just maintenance of intraoperatively placed drains. A clinical leak occurs when the drainage of amylase-rich drainage fluid is associated with fever, leukocytosis, sepsis or the need for percutaneous drainage of an amylase-rich fluid collection or confirmation of pancreatic anastomosis breakdown through fistulogram. CT shows a collection in 20% of these cases. Fistulous tract is best evaluated by fistulogram or ERCP. The associated mortality of pancreatic leaks has markedly declined over the past two decades, now ranging between 0% and 5%.

Pancreatic fistulas can usually be managed conservatively if there is no evidence of abdominal sepsis. Isolated fluid collections should be drained, percutaneously if possible, and they usually heal spontaneously if adequately drained. The management includes bowel rest, intravenous antibiotics, nasogastric suction and, frequently, total parenteral nutrition (TPN). In 70% to 90% of the cases, the fistula heals with conservative management^[56,77]. Octreotide decreases its volume but does not accelerate its closure nor improve survival^[78].

A fistula caused by an anatomic problem will not close with a pharmacological approach. A pancreatic duct obstruction proximal to the fistula must be released either through endoscopy or operative means. Endoscopy drainage is a safe and effective therapy for patients who do not respond to conservative measures. The goal of endotherapy is to reduce the pressure in the pancreatic duct and bypass any existing ductal disruption. Biliary and/or pancreatic sphincterotomy may be done, followed by placement of a nasopancreatic drain. Ductal obliteration with surgical glues may be an option to preserve endocrine function^[77].

When endoscopy is not feasible or the pancreatic ductal disruption can not be bypassed, and pancreatic secretions produced in a portion of the pancreas do not flow into the gastrointestinal tract (fistulas following debridement of pancreatic necrosis, complicating pancreatic or juxtapancreatic surgery, after external drainage of pseudocysts, associated with pancreatic ascites and pancreato-

pleural fistulas), operative intervention is indicated^[79]. If the fluid from the fistula becomes bloody after initially being clear, a second operation should be strongly considered because this finding suggests inflammation and erosion of surrounding vessels. Fistulas located in the body or tail of the pancreas may be treated by distal pancreatectomy. Otherwise, a resection of the fistulous tract and drainage into a Roux-en-Y loop of jejunum may be all right. In septic patients without a radiologically localized cause, a laparotomy should be done, although in a number of cases a source can not be identified. A completion pancreatectomy should be performed in this setting since this may be the only mean of controlling the infection.

Gastrointestinal fistulas

Extravasated pancreatic secretions may directly result in necrosis of adjacent segments of the gastrointestinal tract. The development of fistulas may also be iatrogenic, due to trauma to the surface of organs of the gastrointestinal tract, either secondary to debridement, repeated packing or pressure necrosis from an adjacent drain. Fistulas are defined by a contrast study showing direct communication with the underlying organ.

Gastrointestinal fistulas, when they occur, can be difficult to manage, with the likelihood of spontaneous closure dependent on the fistula site. Repair of duodenal fistulas may be managed by Roux-en-Y duodenojejunostomy or, if necessary, by a pyloric exclusion procedure. Enteric fistulas are usually corrected by segmental resection. Colonic fistulas may occasionally be treated with segmental resection, but they frequently require proximal diversion.

Enteric fistulae occur in 0.4-7.4% of cases after pancreatoduodenectomy and usually indicate a leakage of the gastro-jejunostomy, but are much more frequent after laparostomies or relaparotomies as treatment of infected pancreatic necrosis (41%-75%)^[76]. Conservative management including the maintenance of drains, percutaneous drainage and total parenteral or enteral nutrition are usually sufficient to heal these fistulae. The benefit of somatostatin or octreotide is controversial, although currently available information seems to suggest a considerable benefit of somatostatin when administered in association with standard conservative treatment^[80]. A reoperation is necessary in case of persistent sepsis.

Delayed hemorrhage

Postoperative haemorrhage occurs in 2-15% of the patients following pancreatic resection. Hemorrhage within the first 24 hours is generally caused by a nonsecured vessel, which needs immediate hemostasis through a relaparotomy, or from an anastomosis suture line and is usually managed conservatively. Selective angiography should be done if bleeding persists.

Delayed or secondary haemorrhage, usually around two weeks following surgery, is often caused by an anastomotic leak and secondary erosion of the retroperitoneal vasculature or as a complication of necrosectomy. It carries a mortality of 15-58%. In the event of portal venous bleeding, embolisation may not be possible and surgical intervention is usually necessary. Arterial hemorrhage, due to pseudoaneurysm rupture, can be from the right and common hepatic artery, splenic artery, superior mesenteric artery or the gastroduodenal artery stump. Splenic artery and common hepatic artery pseudoaneurysms are treated with surgery or coil embolization. Celiac artery stenosis is usually not treated unless there are attributable symptoms. Because of the rich collaterals between the celiac and superior mesenteric arteries, isolated vessel stenosis is usually asymptomatic. Proper hepatic artery bleeding requires other

measures.

Investigations include contrast-enhanced CT, endoscopy and selective angiography with selective embolisation if a bleeding point can be identified, with a success rate of 63-79%. If the bleeding vessel can not be occluded, reoperation is mandatory with procedures depending on the site of the damaged artery^[81-83].

Pancreatic and chylous ascites

Pancreatic ascites is an uncommon condition caused by the disruption of the main pancreatic duct draining to the peritoneal cavity. It produces peritoneal fluid that is rich in amylase and leads to massive ascites.

The benefit of octreotide is controversial in these cases. Endoscopic sphincterotomy or a transpapillary stent may be useful at reducing the pressure within the duct or the pseudocyst. Surgery is indicated if ascites is not resolved after 3 or 4 weeks of conservative treatment, which occurs in around 50% of the patients. ERCP is very helpful in the surgical treatment, given its ability to delineate a point of leakage. A Roux-en-Y anastomosis with the site of ductal disruption or a distal pancreatectomy if the lesion is in the body or tail of the pancreas are the procedures of choice.

Postoperative chylous ascites is not a frequent complication (3.4%) that may result in serious nutritional and immunological impairment due to loss of proteins and lymphocytes. Apart from the improvement of nutritional condition, octreotide is effective in the treatment of chylous ascites^[84].

Pancreatic pseudocyst

A pancreatic pseudocyst is a localized collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue, arising as a consequence of acute or chronic pancreatitis, neoplastic obstruction of the pancreatic duct^[85,86], pancreatic trauma or pancreatic surgery. Pseudocyst related complications include infection, hemorrhage, rupture, recurrence and obstruction of the gastrointestinal tract.

While cystic fluid collections in and around the pancreas may be visualized via ultrasound, it is not the study of choice to establish a diagnosis of recurrence. CT scan is the imaging criterion standard for pancreatic pseudocysts with a sensitivity of 90-100%. MRI is useful in detecting a solid component to the cyst and in differentiating between organized necrosis and a pseudocyst. ERCP is not necessary in diagnosing pseudocysts; however, as endoscopic US (EUS), is useful in planning therapy^[87].

Primary pancreatic pseudocyst may be observed, treated with percutaneous drainage, with endoscopic drainage or with either open or laparoscopic internal drainage, which is the standard treatment. Open surgery is the recommended treatment for patients with recurrent pseudocysts. These different procedures show recurrence rates between 6% and 18%^[74,88].

Recurrent intraabdominal abscess

Recurrent intraabdominal abscess is reported to develop in 13% to 26% of patients following necrosectomy for infected pancreatic necrosis after acute pancreatitis. The incidence of repeated abscesses is higher in patients with fewer reoperative necrosectomies, highlighting the importance of complete removal of all infected necrotic tissue. CT scan can distinguish between sterile and infected necrosis, the presence of gas and the potential for drainage^[89]. Many of these patients can be successfully managed by CT-guided percutaneous drainage, with only a few patients requiring reoperative drainage. The development of these intraabdominal abscesses does not appear to have an effect on survival.

Persistent pain

After surgical treatment of chronic pain from pancreatitis through decompression techniques or pancreatic resection, pain can develop or continue in 10% to 40% of the patients. Investigation to exclude other potential sources of pain, including peptic ulcer disease and biliary stricture are mandatory. If other processes are excluded, an ERCP or MRCP should be performed to examine the patency of the pancreaticojejunal anastomosis and to ensure that there are no undrained segments of the pancreatic duct. If the anastomosis is occluded or if residual undrained segments are identified, redrainage can provide satisfactory pain relief. Completion pancreatectomy is the alternative.

Dumping syndrome

It is a rare complication after duodenopancreatectomy and is even rarer after the pylorus preserving technique^[90,91]. Dumping is the effect of an altered gastric reservoir function and an abnormal postoperative gastric motor function. Dumping syndrome can be separated into early (30-60 min) and late (1-3) forms, depending on the occurrence of symptoms in relation to the time elapsed after a meal. The severity of the symptoms is proportional to the rate of gastric emptying, but the pathogenesis is not clear. A postprandial increase in blood flow to the superior mesenteric artery and in gut hormones (enteroglucagon, peptide YY, pancreatic polypeptide, vasoactive intestinal polypeptide, glucagonlike peptide-1 and neurotensin) seems to be pivotal factors in the early symptoms while hyperinsulinemic (reactive) hypoglycemia is involved in the late form.

The expression of the symptoms is highly variable. Early systemic symptoms are desire to lie down, palpitations, fatigue, faintness, syncope, diaphoresis, headache and flushing. Early abdominal symptoms include epigastric fullness, diarrhea, nausea, abdominal cramps and borborygmi. Late symptoms are perspiration, shakiness, difficulty to concentrate, decreased consciousness and hunger. Signs and symptoms can be elicited with the glucose challenge test (50 g oral glucose). A positive result from a hydrogen breath test after ingestion of glucose is also 100% sensitive. A gastric emptying study may be helpful to document rapid gastric emptying while an endoscopy or a barium study can be helpful in discerning the anatomy.

Dietary prohibitions and instructions are very important in the management of dumping syndrome. Octreotide is very helpful in controlling the symptoms but its long-term efficacy is much less favorable. Acarbose has been shown to help in patients with late dumping. Exceptionally, a 10-cm antiperistaltic jejunal loop can be interposed.

Endocrine Insufficiency

Diabetes may be a complication if a major portion of the gland is removed. In an otherwise normal pancreas, as much as 80% of the pancreas may be removed without the development of diabetes; in chronic pancreatitis, resection of as little as 50% of the gland may cause diabetes. In cases of distal pancreatectomy, the reported incidence of new onset, insulin-dependent diabetes mellitus was approximately 8%; in patients with chronic pancreatitis the risk is reported to range from 12% to 46%. In the setting of chronic pancreatitis there is no difference in the incidence of diabetes between operated patients and non-operated patients in the long term, which suggests that, with regard to endocrine status, progression of disease has a greater impact than the surgical intervention.

Pancreatogenic diabetes is characterized by the absence of the major glucoregulatory hormones insulin and glucagon, instability, and frequent hypoglycemia. However, though patients experienced alterations in lifestyle, most of them are able to resume a reasonable functional status and level of activity, with intermittent hypoglycemia being the most frequent complication.

Exocrine Insufficiency

Loss of exocrine function is a common problem (around 40%) among patients with chronic pancreatitis. Surgical procedures for chronic pancreatitis, especially resection, invariably result in some degree of exocrine insufficiency. Pancreaticoduodenectomy is associated with a 10% to 20% risk of symptomatic exocrine insufficiency^[49].

Symptoms of exocrine insufficiency are gas bloating, postprandial cramping, and foul, loose stools. Exocrine insufficiency is not considered a serious complication of pancreatic surgery and can usually be easily treated by oral pancreatic enzyme supplementation, given orally and titrated to effect.

Pancreas rejection

Rejection of the transplanted graft may occur at any time from within seconds to years after transplantation. Acute rejection usually occurs from approximately 1 week to 3 months after transplantation and is the only type of rejection that can be treated. Chronic rejection can occur at any time after approximately 3 months after transplantation and leads to insidious, progressive loss of graft function. Incidence of rejection are higher after solitary pancreas transplant than after simultaneous kidney and liver transplant.

Acute rejection can be diagnosed with pancreas biopsy or with CD2 or CD3 counts if biopsy is difficult to obtain.

In pancreas transplant alone or in pancreas transplant after kidney transplant the lack of sensitive indices for rejection makes the diagnosis of acute rejection difficult. Signs and symptoms include low-grade fever, unexplained leukocytosis, and a swollen, painful graft. Alterations in C-peptide and insulin levels are not sensitive enough. Fasting blood glucose levels and increases in serum amylase, lipase, and glucose levels may be useful, but are usually not apparent until late. Serum amylase may double or triple with rejection; however, an elevated serum amylase level may also indicate pancreatitis. In transplantations with bladder drainage, a decline in the urinary amylase level precedes hyperglycemia, making urine amylase a useful marker for acute rejection. However, urine amylase levels may be elevated with diuretic therapy. The posttransplant protocol pancreas biopsy is the most sensitive and specific method for diagnosing acute rejection of the pancreas allograft. When acute rejection is suspected, an ultrasound-guided cystoscopic transduodenal or percutaneous biopsy is done.

In simultaneous pancreas and kidney transplant recipients, increased serum creatinine and blood urea nitrogen are surrogate markers for pancreas rejection, because the kidney and pancreas reject simultaneously and a decline in kidney function precedes a decline in pancreas function by a few days.

Acute rejection requires hospitalization for aggressive immunosuppressive therapy. Dosages of current immunosuppression medications are adjusted, and pulsed corticosteroid therapy and/or antilymphocyte agents are administered.

The diagnosis of chronic rejection is challenging. Chronic rejection of the pancreas allograft is manifested by hyperglycemia and low C-peptide levels, and eventually results in graft loss. Chronic rejection is confirmed by biopsy. The rate of graft loss after 5 years from chronic rejection is 55%.

Portal-splenic-mesenteric vein thrombosis

Thrombosis of the portal venous system can be a complication of necrotizing pancreatitis and of surgery of the pancreas, biliary tract and spleen.

The presentation of these patients may be with symptomatic ascites, gastrointestinal bleeding, venous infarction or evidence of hypersplenism.

The diagnosis can be made by contrast-enhanced CT scan or duplex ultrasound. Contrast-enhanced CT scan is able to reconstruct the vascular anatomy to assist in planning for any proposed surgical intervention. There is some controversy on whether any acute intervention should be undertaken on the diagnosis of this condition. Although a number of interventional radiological techniques are possible, they need to be performed with a low morbidity in patients who already are severely unwell. Because recanalisation can occur with anticoagulation, formal anticoagulation is recommended. In the long term, meso-caval shunts offer good relief from debilitating symptoms with minimal long term morbidity^[76].

Splenic Infarction and abscess

Infarction of the spleen can follow pancreatic resection or pancreatic necrosectomy and can occur either as a result of vessel trauma or from postoperative pancreatitis^[92]. It occurs more commonly after a spleen-preserving procedure but can also occur after a conventional spleen-sparing distal pancreatectomy. Ischemia can lead to abscess formation or severe postoperative pain. In some situations, the spleen undergoes hypertrophy in an attempt to compensate for inadequate perfusion. This requires close follow-up and analgesic.

This complication often follows the spleen-preserving procedures (ligating vessels distal to the pancreas). Symptoms are abdominal pain and fever. A technetium-99 m sulfur colloid spleen scan will show an abnormal uptake, and a CT scan will indicate sufficient or insufficient blood flow. Depending on each case, either drainage or splenectomy can be performed.

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