Pancreatic surgery Complications

Pancreatic Fistula

Pancreatic fistula is one of the most common and feared complications from partial pancreatectomy and occurs generally in 10% to 25% of cases.

The most common clinical presentation is the appearance of murky fluid in a drain left next to the pancreaticoenteric anastomosis or cut edge of the pancreas.

Alternatively, the patient manifests an intraabdominal fluid collection of pancreatic juice or abscess following the operation.

- **ETIOLOGY**

Pancreatic fistula often results from the disruption of the pancreaticoenteric anastomosis either from ischemia, erosion by pancreatic enzymes, or other technical issues.

Pancreatic fistula can also develop from the cut edge of the pancreas if the pancreatic duct has not been sufficiently ligated with suture or controlled with staples.

- **Risk factors**

The texture of the pancreas appears to be the most important risk factor of pancreatic fistula after pancreatectomy. A soft pancreas does not hold suture or staples well, and postoperatively the local inflammatory process can disrupt these. Soft glands are often found in patients with neuroendocrine tumors, ampullary tumors, and cystic lesions.

In patients with chronic pancreatitis, the nature of this disease process leads to inflammation and fibrosis of the pancreas, making the gland firm, and the occurrence of pancreatic fistula is relatively low .

Pancreatic cancer is associated with a fibrotic pancreas and this is responsible for a lower PF rate in patients with this disease.

Other risk factors include the size of the pancreatic duct diameter and intraoperative blood loss

- **CLINICAL PRESENTATION**

- Change in the character of the effluent found in a surgically placed drain.(cloudy or completely clear)

- Prolonged ileus

- An elevated white blood cell count with a fever

- Tachycardia

- Renal dysfunction

- Mental confusion

TABLE 76-1: DEFINITIONS OF PANCREATIC FISTULA

1. Output >10 mL/d of amylase-rich fluid (>3× serum amylase) postoperative (postop) day 5 or for >5 days
2. Output >10 mL/d of amylase-rich fluid after postop day 8 or for >8 days
3. Output between 25 mL/d and 100 mL/d of amylase-rich fluid after postop day 8 or for >8 days
4. Output >50 mL/d of amylase-rich fluid after postop day 11 or for >11 days

Four final definitions summarizing the current pancreatic fistula concept according to the literature. Reproduced with permission from Bassi C, Dervenis C, Butturini G, et al. Postoperative pancreatic fistula: an international study group (ISGPF) definition, *Surgery* 2005 Jul;138(1):8-13.

TABLE 76-2: GRADES OF PANCREATIC FISTULA FROM ISGPF

Main Parameters for POPF Grading

Grade	Biochemical Leak	B	C
Clinical conditions	Well	Often well	Ill appearing
Specific treatment	No	Yes/no	Yes
US/CT (if obtained)	Negative	Negative/ positive	Positive
Persistent drainage (after 3 weeks)	No	Yes	Yes
Reoperation	No	No	Yes
Death related to POPF	No	No	Possibly yes
Signs of infections	No	Yes	Yes
Sepsis	No	No	Yes
Organ failure	No	No	Yes/no

Reproduced with permission from Bassi C, Dervenis C, Butturini G, et al. Postoperative pancreatic fistula: an international study group (ISGPF) definition, *Surgery* 2005 Jul;138(1):8-13.

TREATMENT

The treatment of pancreatic fistula is mostly conservative, and fortunately, most pancreatic fistulae (70%-82%) will resolve within weeks with conservative management. This is true for both pancreaticoduodenectomy and distal pancreatectomy.

With biochemical leak pancreatic fistula, which is the most common form of pancreatic fistula, the patients can still feed orally.

Total parenteral nutrition (TPN) or somatostatin analog such as octreotide is not required and this class of fistula rarely delays hospital discharge.

In contrast, grade B pancreatic fistula may requires : strict NPO and TPN. Octreotide may be indicated if the volume is significant.

If the patient has fever or leukocytosis, antibiotics are also needed.

Hospital discharged is likely to be delayed as these patients may need interventional drainage of fluid collections or angiographic embolization for hemorrhage.

Grade C pancreatic fistula requires major changes of the standard clinical pathway.

The patient often requires NPO, TPN, intravenous antibiotics, and somatostatin analog and care in an intensive care unit.

CT scan may show peripancreatic fluid collection.

Hospital stays are often lengthened.

If the patient continues to deteriorate clinically, reoperation may be required to repair or revise the pancreaticoenteric anastomosis.

In extreme conditions, completion pancreatectomy may be necessary.

Delayed Gastric Emptying

DGE is characterized by oral intolerance, inability to remove the nasogastric tube, and/or the necessity of reinserting the nasogastric tube several days after the operation.

In most reports, the rate of DGE ranges from 19% to 57%.

ETIOLOGY/RISK FACTORS

The mechanism of DGE is largely unknown.

It has been postulated that the resection of duodenum can trigger DGE, and this is supported by the fact that there is less DGE with duodenum-preserving pancreatic head resection.In addition, distal pancreatectomy that does not involve duodenal resection rarely causes DGE.

Decreased motilin level has also been suggested to trigger DGE, given that the prokinetic drug erythromycin, which is a motilin agonist, can reduce the incidence of DGE.

Pylorus-preserving pancreaticoduodenectomy is one of the most common variations of the classic pancreaticoduodenectomy, and some reports have claimed that it is associated with higher incidence of DGE, while others have shown the opposite. The etiology of this may be that pylorus-preserving pancreaticoduodenectomy can cause devascularization or denervation of the pylorus with subsequent pylorospasm.

In contrast to pancreatic fistula, for which imaging is often not required, diagnosis of DGE frequently requires imaging. The most commonly used study is fluoroscopic upper gastrointestinal series. CT scan can also be used to visualize distended stomach and also rule out stenosis in the gastric outlet, which may require reoperation or endoscopic management.

TREATMENT

1. Replacement of NGT
2. Prokinetic medications
3. Radiological or even operative intervention
4. TPN

Biliary Leak

Biliary leak is a fairly uncommon complication following pancreaticoduodenectomy.

Nevertheless, biliary leak increases postoperative morbidity and mortality.

DIAGNOSIS

Biliary leak usually does not require special diagnostic workup. It is often evidenced by bile in the fluid collected by the surgical drain left at the time of operation. If it presents as abdominal abscess, CT scan can often be sufficient.

TREATMENT

Although postoperative biliary leak is associated with increased morbidity and mortality, it usually can be managed conservatively without percutaneous drainage or reoperation. For some patients with high-volume output, diversion with a transhepatic catheter may be helpful.

Pancreatic Insufficiency

Pancreatic exocrine insufficiency is fairly common with chronic pancreatitis or pancreatic cancer even prior to pancreatectomy.

In a recent systematic review, Tseng et al. (2016) found that 20% to 63% of patients presented with preoperative pancreatic exocrine insufficiency. This rate increased to 67% to 80% 6 months after pancreatectomy.

The extent of pancreatic resection directly predicts the probability of developing pancreatic exocrine insufficiency imposing a significant negative impact on the quality of life.

Pancreatic exocrine insufficiency is often diagnosed with a combination of clinical presentation and pancreatic exocrine function tests with fecal fat content and fecal elastase determination. Patients may present with malnutrition.

Steatorrhea is often a late symptom of pancreatic exocrine insufficiency.

Currently the mainstay therapy is pancreatic enzyme supplementation or replacement.

Pancrelipase, often in the delayed-release form (Creon), is often used.

A recent study by Whitcomb (2016) has shown that pancrelipase significantly increases fat and nitrogen absorption.

Proton-pump inhibitors (PPIs) are also often used to suppress gastric pH, given the decrease pancreatic bicarbonate secretion. However, no significant difference in fat absorption has been found in the PPI group and the placebo group .

New-Onset Diabetes

Since pancreatectomy removes pancreatic endocrine components, it is not uncommon for patients to develop new-onset insulin-dependent diabetes.

If the pancreatectomy is for chronic pancreatitis, the pancreas is likely already compromised before the surgery. It is therefore not surprising that chronic pancreatitis patients have an even higher risk of insulin-dependent diabetes than other pancreatic surgical patients.

A large systematic review has shown that the risk of new-onset diabetes after distal pancreatectomy in patients with chronic pancreatitis is 39%, and the risk of development of diabetes after resection of pancreatic tumor is only 14%.

The amount of resection is also positively correlated with the risk of diabetes .

In patients with new-onset diabetes after pancreatectomy, glucose control is more difficult due to severe fluctuations in glucose levels associated with exogenous insulin and deficiency of pancreatic polypeptide.

Mortality from hypoglycemia has been reported in patients with pancreaticoduodenectomy, and it was thought to result from a combination of exogenous insulin and lack of glucagon .

Patients with distal pancreatectomy are at particularly high risk for hypoglycemia, because the glucagon-producing alpha cells are located mainly in the pancreatic body and tail.

Adrenal surgery Complications

The intraoperative risks of adrenal surgery are due largely the close proximity to large vascular structures and other retroperitoneal organs.

Consequently, minimally invasive adrenalectomy poses the same anatomic risks as open adrenalectomy: major vascular injury (IVC, splenic vessels, renal vessels) and injury to the spleen, liver, and colon.

Although rare, transection of the porta hepatis, hepatic artery, ureter, and renal artery has been reported.

The dissection of the adrenal gland is in close proximity to the posterior aspect of the diaphragm, making ipsilateral pneumothorax a potential complication necessitating a tube thoracostomy in some.

Further, pneumoperitoneum can impair venous return, which can be particularly dangerous in the setting of catecholamine surges during resection of pheochromocytoma.

This risk can be minimized with pre and intraoperative hydration. The spleen and liver are also at risk for injury during laparoscopic adrenalectomy; these organs can sustain trocar injuries, capsular tears from grasping or retraction, or vascular injury.

The most life-threatening complication of adrenalectomy is a vascular injury.

Releasing insufflation and hyperventilating the patient can relieve intraoperative hypercarbia.

Subcutaneous emphysema and subcostal nerve dysfunction can be observed after RP adrenalectomy, and both are transient in nature.

Hypoglycemia is a well-recognized complication in adrenalectomies performed for pheochromocytoma.

Splenic Surgery Complications

Complications Overall, the complication rate of elective laparoscopic splenectomy is 10% to 15%, with a mortality rate of less than 1%.

Splenectomy for hematologic malignancy and splenomegaly has a higher complication rate, with reports of a 9% to 18% mortality rate for patients with myeloproliferative disorders.

In observational studies, the rate of complications was lower for patients having laparoscopic compared to open splenectomy, including for splenomegaly.

Patients must be closely monitored for early postoperative bleeding, particularly those with thrombocytopenia or myeloproliferative disorders.

Patients with massive splenomegaly and underlying coagulopathy should be in a monitored setting in the immediate postoperative period.

It is an error to ascribe bleeding to hematologic abnormalities, and although these should be corrected, it is generally safer to reexplore patients early and to evacuate a hematoma to reduce the incidence of subphrenic abscess.

Injury to the tail of the pancreas with a symptomatic complication can occur in up to 10% of cases.

Development of a pancreatic collection will require drainage that remains in place until the fistula closes.

Pulmonary complications such as atelectasis, effusion, and pneumonia occur more frequently following open splenectomy.

One intraoperative complication that may occur during laparoscopic splenectomy but is rarely seen with open splenectomy is diaphragmatic perforation, usually related to thermal injury during mobilization of the superior pole.

In unusual cases, the platelet count may rise to very high levels. In cases where the platelet count rises to >1000 × 10 9 /L, a drug that inhibits platelet aggregation, such as acetylsalicylic acid, can be used.

Thrombosis of the splenic vein, with extension into the portal vein and superior mesenteric vein, is a potentially lethal complication of splenectomy if it results in bowel ischemia or portal hypertension.

In a review of splenic-portal vein thrombosis (SPVT), the overall incidence of symptomatic thrombosis was 3.3% and similar between the open and laparoscopic approach.

Patients with myeloproliferative disorders, lymphoproliferative disorders, and hereditary hemolytic anemias (particularly HS and thalassemia) are at particularly high risk, whereas patients with ITP are at lower risk.

Interestingly, splenectomy for trauma is not associated with a significant risk for SPVT.

Clinically, patients with splenomegaly are at higher risk than patients with smaller spleens.

In one study, the incidence of SPVT on screening postoperative Doppler ultrasound was 78% for patients with spleen size >20 cm, 31% for spleens between 15 and 20 cm, and 13% for spleens ≤15 cm.

Although abnormalities in prothrombotic screening tests are very common, they do not predict SPVT.

The high incidence of asymptomatic SPVT has generated a debate about whether patients should have surveillance imaging (ultrasound or CT) after laparoscopic splenectomy, since untreated thrombosis could lead to significant morbidity and mortality.

The median time from splenectomy to asymptomatic SPVT is 6 days, whereas the median interval to symptomatic SPVT is about 8 to 12 days.

Prompt treatment of asymptomatic PSVT leads to resolution in 90% of cases,but it is also known that isolated thrombosis in the splenic vein may resolve without anticoagulation.

Since symptoms of SPVT can be subtle, abdominal imaging should be performed for any patient with any deviation from the expected postoperative course, including fever, abdominal pain, diarrhea, anorexia, or nausea.

Perioperative anticoagulation prophylaxis is recommended, and European Association for Endoscopic Surgery guidelines further recommend prophylactic anticoagulation for 4 weeks after surgery.

Although a single small randomized trial of extending postoperative prophylaxis failed to show a benefit,this is strongly considered for patients at particularly high risk (eg, myelodysplastic disorders with splenomegaly) once hemostasis is assured.