Acute Complications after Total Pancreatectomy/Islet Autotransplantation (TPIAT)

1. Intraabdominal Bleeding

After TPIAT, patients are at risk for intraabdominal bleeding for several reasons. First, the pancreatectomy is often performed in a reoperative field, possibly with dense adhesions and/or altered anatomy. Also, the indication for surgery is pancreatitis, and thus these patients will typically have dense inflammation and chronic scar in the surgical bed. During this surgery a major named vessel, the gastroduodenal artery, is divided. Shortly after surgery, these patients are given 70U/kg of heparin with their islet transplant. During the transplant, they have increased portal venous pressure and may also have some transient hepatic dysfunction from the transplant (which is a portal vein embolization), both of which increase risk for bleeding. With all of these factors in mind, the physician caring for these patients should be on alert for signs of bleeding.

Any tachycardia, hypotension, increased fluid requirement, increased bloody drain output, or increased abdominal distension should be evaluated with a check of the patient’s serum hemoglobin. In all patients in the postoperative period, serial checks of the serum hemoglobin should be performed every 6 hours until it is stable.

If there is due concern that the patient is bleeding, the patient should be resuscitated and any coagulopathy should be corrected. The patient’s primary surgeon should be notified for an action plan. Depending on the timing of the bleed and the condition of the patient, reoperation versus angiographic intervention may be warranted. In general, a CT scan is not in the usual algorithm for the evaluation of postoperative bleeding.

2. Portal Vein Thrombosis

Islet autotransplantation occurs through a catheter placed into the portal vein. (At our institution this is done through a percutaneous, transhepatic route). The islets have on their surface tissue thromboplastin which is thrombogenic. To minimize the risk of PVT during the transplant, portal venous pressures are monitored during the infusion and are not allowed to go above 30 mmHg. In addition, the patients are given 70U/kg of heparin (contained within the bag of islets).

Clinical indicators of portal vein thrombosis are signs of hepatic dysfunction, including elevated liver biochemistries, elevated INR, or thrombocytopenia. A mild, transient bump in these numbers is expected in all cases postoperatively, but a significant bump (greater than 3x normal) or persistence beyond 24 hours should raise suspicion of this complication.
PVT can be best evaluated with a RUQ duplex ultrasound exam. When ordering this you MUST indicate you want a doppler study of the hepatic vessels. Otherwise they will not do it and you will not get the information you need.

If you suspect PVT, the primary surgeon should be notified for an action plan. Depending on the clinical scenario, anticoagulation is typically the treatment for PVT.

3. **Systemic Inflammatory Response Syndrome**

Systemic inflammatory response syndrome may occur in patients after TPIAT. The isolated islets have tissue thromboplastin on their surface, which can precipitate activation of the inflammatory cascade.

SIRS should be suspected in patients with tachycardia, hypotension, hypoxia, oliguria, acidosis or increased fluid requirements postoperatively.

SIRS is a diagnosis of exclusion. All other causes of the above signs and symptoms should be sought and excluded (ie bleeding, sepsis, PE, etc).

The treatment for SIRS is supportive.

4. **Hepatic Artery Pseudoaneurysm**

Hepatic artery pseudoaneurysm (HAP) can occur after TPIAT. At our institution, the islet infusion typically occurs in the interventional radiology suite via the percutaneous, transhepatic route. This involves several passes with a needle through the liver to access the biliary tree. This access needle potentially could injure a branch of the hepatic artery. This could cause acute bleeding, or more often, a pseudoaneurysm. The risk of a pseudoaneurysm is the potential for rupture/acute bleeding.

HAP is most often discovered incidentally during a CT scan done for another purpose. It is an enhancing intrahepatic lesion seen on contrasted CT. Alternatively, HAP may present with acute intraabdominal bleeding. If a HAP is discovered or suspected, an angiogram should be performed promptly to embolize it.

5. **Biliary Anastomotic Leak**

After TPIAT, patients may leak from the choledochojejunostomy. A leak is suggested by bilious output in the operative drain or, in the patient without a drain, fever, leukocytosis, and/or hyperbilirubinemia. Management should be discussed with the primary surgeon, as the
approach to a leak depends on the condition of the patient and the magnitude of the leak. Some leaks will heal with drainage alone; other leaks require stent via PTC.

6. **Biliary Anastomotic Stenosis**

Patients may develop an anastomotic stricture of the choledochojejunostomy. Typically a stricture presents with elevated liver enzymes, particularly alkaline phosphatase and bilirubin. If this is suspected, the primary surgeon should be consulted for management. Magnetic resonance cholangiopancreatography is usually the best test to evaluate the biliary anastomosis. Stenosis is typically managed with a percutaneous transhepatic cholangiogram with balloon cholangioplasty and stenting.

7. **Gastrointestinal bleeding**

GI bleeding can occur after TPIAT. The most common cause is gastritis and therefore a proton pump inhibitor should be initiated. For persistent bleeding or bleeding requiring a transfusion, further investigation is warranted, typically with upper endoscopy. Anastomotic bleeding from the gastrojejunostomy should be evaluated.

8. **Delayed gastric emptying**

Delayed gastric emptying is reported to occur in 15 to 25% of cases after pancreatic head resection. Similarly, this complication can occur in patients after total pancreatectomy. For this reason, careful consideration should occur on the timing of nasogastric tube removal and diet initiation and advancement.