Pancreaticoportal fistula - A rare complication of pancreatitis

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Learning objectives

• To describe and identify ‘pancreaticoportal fistula’ as a complication of pancreatitis.

• To describe relevant imaging findings in pancreaticoportal fistula. Imaging modalities reviewed in this presentation include CT, MRI/MRCP, and ERCP.

• Discuss management of pancreaticoportal fistula and long-term outcome correlated with imaging.

Background

Introduction:

Pancreaticoportal fistula is an uncommon complication of pancreatitis. There are a few case reports of this entity published in the literature. Due to lack of knowledge of this condition, radiologists may inaccurately diagnose acute thrombosis of the portal venous system rather than a fistulous communication of the pancreatic duct and/or pseudocyst with the portal venous system. Improper diagnosis may lead to unnecessary high-risk anticoagulant therapy in the setting of pancreatitis.

Proposed Pathogenesis:

It has been established that pancreatitis can cause internal fistulas. These fistulas have been reported between the pancreas and the stomach, colon, small bowel, biliary tree, peritoneal space, and pleural space (1,2). The exact mechanism of pancreaticoportal fistula formation is unknown. One theory suggests that activated pancreatic enzymes leak from an injured duct (either main or side branch) and cause regional inflammation with acute portal vein thrombus. Over time, these activated pancreatic juices erode the wall of the adjacent portal vein. Subsequently, the acute intravascular portal vein thrombus is lysed and becomes replaced by the draining pancreatic juices. This process results in a direct communication between the pancreatic duct (main or side branch) and the portal venous system, either directly or via a pseudocyst, creating the pancreaticoportal fistula (1). Most case reports of pancreaticoportal fistula in the literature are noted in the setting of chronic, long-standing pancreatitis, with alcohol being the most common cause (1, 3-8).
Clinical Presentation:

Acute pancreatitis most commonly presents as abdominal pain with elevated serum enzymes. There are many associated complications of this condition, including peripancreatic effusions or ascites, pseudocysts, hemorrhage, portal venous thrombosis, retroperitoneal necrosis (including the pancreas and/or surrounding fat), and sepsis. The most common clinical presentation of pancreaticoportal fistula described in case reports is abdominal pain (1). Abdominal pain was the commonest symptom in all of our patients. This is not unexpected, as this condition presents in the setting of pancreatitis. In our experience, the diagnosis of pancreaticoportal fistula was serendipitously discovered during routine imaging for patients with pancreatitis.

Management:

As there are only a few case reports in the literature, no unified clinical management guidelines for pancreaticoportal fistula are available. The majority of patients are managed on a case-by-case basis, either conservatively or surgically. It is important to recognize this condition in order to avoid unnecessary treatment with anticoagulation for inaccurately diagnosed portal venous thrombosis.

Imaging findings OR Procedure details

Imaging Findings:

Hallmark imaging findings in pancreaticoportal fistula include:

1. Changes of acute or chronic pancreatitis.

2. Diffuse fluid-like appearance of the expanded portal-venous system.
   - CT - Diffuse fluid-like low attenuation of the portal veins. The density is much lower than expected for bland acute thrombus.
   - MR - Diffuse fluid-like T2 hyperintense signal of the portal veins. T2 hyperintensity is much brighter than expected for acute thrombus.

3. Direct communication of the pancreatic duct and/or a pseudocyst with the portal-venous system.
• Migration of a calculus from the pancreatic duct into the portal venous system can also be seen.

The diagnosis of pancreaticoportal fistula on any imaging modality (CT, MRI/MCRP, or ERCP) must demonstrate these findings, independently or in conjunction with one another.

Example Cases:

**Patient 1** (Figures 1-6) is a 61 year old male with chronic alcohol abuse and recurrent pancreatitis. His initial presentation was with abdominal pain. Imaging work-up revealed communication of the pancreatic duct with the portal venous system. This was visualized on both CT and MRI, and confirmed with ERCP. Percutaneous drainage of the pseudocyst was performed and the patient abstained from further alcohol use. Migration of a pancreatic ductal calculus into the superior mesenteric vein was seen on long term follow up imaging. Follow-up images also demonstrate cavernous transformation of the portal venous system.
**Fig. 1:** Patient 1. Contrast enhanced axial CT images from 4/2008 demonstrate changes of chronic pancreatitis with parenchymal calcifications in the pancreatic head (yellow arrow), prominence of the pancreatic duct (red arrow), and a calculus within the pancreatic duct (blue arrow). Note patency of the superior mesenteric vein (green arrow).

**References:** Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US

**Fig. 2:** Patient 1. Contrast enhanced axial CT images from 9/2009 demonstrate new fluid density within the portal venous system (red arrow) extending to the splenic vein and superior mesenteric vein (white arrow). Note the communication between the pancreatic head pseudocyst containing the calculus (green arrow) with the splenic vein (yellow arrow).

**References:** Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US
**Fig. 3:** Patient 1. T2 weighted axial MR image from 9/2009 shows the pancreatic pseudocyst communicating with the splenic vein (red arrow).

**References:** Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US
Fig. 4: Patient 1. Single ERCP image from 3/2010 illustrates changes of chronic pancreatitis, with beaded dilatation of the pancreatic duct (red arrow), and opacification of the portal venous system (yellow arrow denotes opacification of the splenic vein). Note also the pigtail catheter draining the large pancreatic pseudocyst.

References: Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US
Fig. 5: Patient 1. Contrast enhanced axial cine CT images from 4/2010 showing progression of pancreatitis with increasing free fluid and a large epigastric pseudocyst. There is increased pancreatic ductal dilatation communicating with the pancreatic head psueducyst. This pseudocyst also communicates with the portal, splenic, and superior mesenteric veins. Note that the calculus previously seen in the pancreatic head pseudocyst has migrated to the superior mesenteric vein, proving a fistulous communication.

References: Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US

Patient 2 (Figures 7-10) is a 55 year old female who presented initially to the emergency department with abdominal pain. Her initial imaging showed changes of acute pancreatitis.
(Fig. 7). On follow up imaging, the patient developed a pancreatic pseudocyst which communicated with the portal venous system (Fig. 8-10).

**Fig. 7:** Patient 2. Contrast enhanced axial CT image from 12/2007 demonstrates acute pancreatitis with peripancreatic inflammatory changes.

**References:** Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US
Fig. 8: Patient 2. Contrast enhanced axial CT images from 12/2010 demonstrate a prominent pancreatic pseudocyst with fluid density within the portal venous system (green arrow). There is a communication between the pseudocyst and the portal vein (red arrow) as well as the pseudocyst and the splenic vein (yellow arrow).

References: Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US
Fig. 9: Patient 2. Single coronal T2 weighted MRCP image from 12/2010 again illustrates the pancreatic pseudocyst and its communications with the portal vein (red arrow) and the portal venous confluence/ splenic vein (yellow arrow).

References: Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/ US
**Fig. 10**: Patient 2. Contrast enhanced axial CT images from 2/2011 showing similar findings of the pancreatic pseudocyst in communication with the portal and splenic veins with hypodense contents within the portal and splenic veins

**References**: Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US

**Patient 3** (Figures 11-12) is a 25 year old female with a history of polysubstance and alcohol abuse who initially presented with abdominal pain. Preliminary CT images showed changes of acute pancreatitis (Fig. 11 A & B) with development of a pseudocyst on follow up scans (Fig. 11 C & D). A fistulous communication between pancreatic duct and portal venous system was seen on a subsequent CT scan (Fig. 12).
**Fig. 11:** Patient 3. Contrast enhanced CT images in a single patient with acute pancreatitis. A) Axial and B) coronal CT images from 12/2010 demonstrate changes of acute pancreatitis with peripancreatic inflammatory stranding. C) Axial and D) coronal CT images from 10/2011 show persistent acute pancreatitis changes with pseudocyst formation (red arrow). The pancreatic duct is also dilated (yellow arrow).

**References:** Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US
**Fig. 12**: Patient 3. Contrast enhanced axial and coronal CT images from 5/2012 demonstrate the interval development of multiple pseudocysts (red arrows). There is new hypodensity within the expanded portal venous system (white arrow), which is contiguous with one of the pseudocysts (yellow arrow).

**References**: Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US

**Patient 4** (Figures 13-14) is a 52 year old male who presented with abdomen pain to the emergency department in 2005. At that time, his CT findings were consistent with acute pancreatitis (Fig. 13 A). He had several recurrent episodes of acute pancreatitis, and CT in 2008 showed a cystic area in the pancreatic head, likely representing a pseudocyst (Fig. 13 B). A follow up CT study in 2012 showed changes of chronic pancreatitis (dilated pancreatic duct and calcifications) and a growing cystic lesion in the pancreatic head (Fig. 13 C & D). Communication of this cystic lesion with splenic vein as well as hypodense material in the portal venous system was noted, suggesting pancreaticoportal fistula (Fig. 14). Chronic changes of cavernous transformation of portal vein are also present (Fig. 14).
Fig. 13: Patient 4. Serial contrast enhanced axial CT images demonstrate pancreatic and peripancreatic changes in a single patient over the course of time. A) Single image from 8/2005 shows findings of acute pancreatitis with peripancreatic inflammatory changes. B) Single image from 10/2008 illustrates a pancreatic head pseudocyst (red arrow). C-D) Images from 5/2012 show an enlarging pancreatic head pseudocyst and other changes of chronic pancreatitis, including pancreatic ductal dilatation and parenchymal calcifications.

References: Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US
**Fig. 14:** Patient 4. Contrast enhanced axial and coronal CT images from 11/2012 demonstrate pancreatic pseudocyst communicating with the portal venous system (red arrow). There is hypodense content within the portal venous system (white arrow). The pseudocyst also communicates with the pancreatic duct (yellow arrow). There is cavernous transformation of the portal vein (green arrow). Incidentally noted are bilateral renal solid and cystic lesions.

**References:** Radiology, Medical College of Wisconsin, Froedtert Hospital - Milwaukee/US

**Images for this section:**
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Fig. 5: Patient 1. Contrast enhanced axial cine CT images from 4/2010 showing progression of pancreatitis with increasing free fluid and a large epigastric pseudocyst. There is increased pancreatic ductal dilatation communicating with the pancreatic head pseudocyst. This pseudocyst also communicates with the portal, splenic, and superior mesenteric veins. Note that the calculus previously seen in the pancreatic head pseudocyst has migrated to the superior mesenteric vein, proving a fistulous communication.
Fig. 6: Patient 1. Contrast enhanced axial CT images from 10/2011 demonstrate the long-term sequelae cavernous transformation (red arrow) and the persistent calculus within the superior mesenteric vein (yellow arrow).

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Fig. 8: Patient 2. Contrast enhanced axial CT images from 12/2010 demonstrate a prominent pancreatic pseudocyst with fluid density within the portal venous system (green arrow). There is a communication between the pseudocyst and the portal vein (red arrow) as well as the pseudocyst and the splenic vein (yellow arrow).
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**Fig. 10:** Patient 2. Contrast enhanced axial CT images from 2/2011 showing similar findings of the pancreatic pseudocyst in communication with the portal and splenic veins with hypodense contents within the portal and splenic veins.
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Conclusion

Our exhibit raises awareness to the condition of 'pancreaticoportal fistula' in the setting of pancreatitis. We review the critical imaging findings of pancreaticoportal fistula. Diagnosis of this condition is important in order to avoid unnecessary anticoagulant therapy for inaccurately diagnosed portal venous thrombosis.

References


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