Chapter

Pancreatic Pseudocyst

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Abstract

Pancreatic pseudocysts frequently occur in the context of acute or chronic pancreatitis and seldom appear as a post-surgical outcome or trauma. Complicated pancreatic pseudocysts represent extremely rare entities but still life-threatening situations, including infection, hemorrhage, rupture, pseudoaneurysms, pancreatic fistulas, obstructions, and splenic complications. Premature diagnosis, based on transabdominal ultrasonography or computed tomography, is crucial for the early therapeutic approach. Conservative treatment, surgical and endoscopic intervention consist the therapeutic options. Thus, management of the complicated pseudocysts demands a multidisciplinary team eligible to cope with complications that might even occur due to the intervention. Pancreatic pseudocysts represent a challenge for clinical doctors.

Keywords: pancreatic pseudocyst, differential diagnosis, complications, multidisciplinary approach, treatment

1. Introduction

Pseudocyst of the pancreas is a localized fluid collection that is rich in amylase and other pancreatic enzymes and is enclosed by a wall of non-epithelialized fibrous tissue. Pancreatic pseudocysts (PPCs) frequently occur in the context of acute or chronic pancreatitis and seldom appear as a postsurgical outcome or trauma. PPCs are less commonly related to acute pancreatitis compared to chronic pancreatitis, due to progressive ductal obstruction while the most common causative factor is alcohol consumption [1, 2]. Computed Tomography (CT) is the diagnostic modality of choice, as it considered to be superior to Ultrasound (US), providing more detailed information regarding the surrounding anatomy. It can demonstrate additional pathology, including pancreatic duct dilatation and calcifications, common bile duct dilatation, and extension of the pseudocyst outside the lesser sac. Complicated PPCs are extremely rare entities but still life-threatening situations, which affect the adjacent tissues of the pancreatic parenchyma. They can lead to infection, hemorrhage, rupture, pseudoaneurysms, pancreatic fistulas, obstructions, and splenic complications. Although they are well described, there is no consensus regarding the "gold-standard" therapy. Therapeutic approaches include conservative treatment(as a majority of cases have been resolved spontaneously), surgical and endoscopic intervention.

2. Historic review and classification for acute pancreatitis

Atlanta classification was the first classification for acute pancreatitis and was originally stated in 1992; giving the opportunity to the universal surgical community to have a common aspect regarding its definition [3]. However, soon this terminology proved to be inadequate and confusing and became outdated. In addition, definition of the pancreatic fluid collections was not well-established and there was huge variety among the surgeons [4]. Better comprehension of the etiology and the pathophysiology of the acute pancreatitis has led to revision of the Atlanta Classification for the acute pancreatitis, two decades later, correcting the aforementioned deficiencies. This revised classification differentiates the acute pancreatitis into two phases: early and late onset as well as the severity as mild, moderate, and severe [5].

Regarding the pancreatic and the peripancreatic fluid collections, terms such as "acute pseudocyst" and "abscess" were misleading and therefore discouraged. Instead, there was a clear distinction between collections that are consisted of sole fluid and those with debris (solid components due to necrosis). Another important factor affecting the categorization of the fluid collections is the presence of infection and certainly the duration of existence (**Figure 1**) [5]. In the Atlanta classification, PPC was described as a well-defined extra-pancreatic fluid collection with minimal solids, which lasts more than 4 weeks as the pancreatitis recedes.

3. Etiology

The appearance of PPC parallels that of pancreatitis and the etiology is strictly associated with the causes of pancreatitis. Typically, the PPCs form as a result of pancreatic duct disruption with subsequent fluid leakage or by the maturation of peripancreatic necrosis. Ninety percent of them occur in the context of pancreatitis, while only 10% are caused by trauma (surgery, gunshots, and blunt abdominal trauma) [6]. Regarding the acute pancreatitis, PPCs formation (approximately 15%) is infrequent

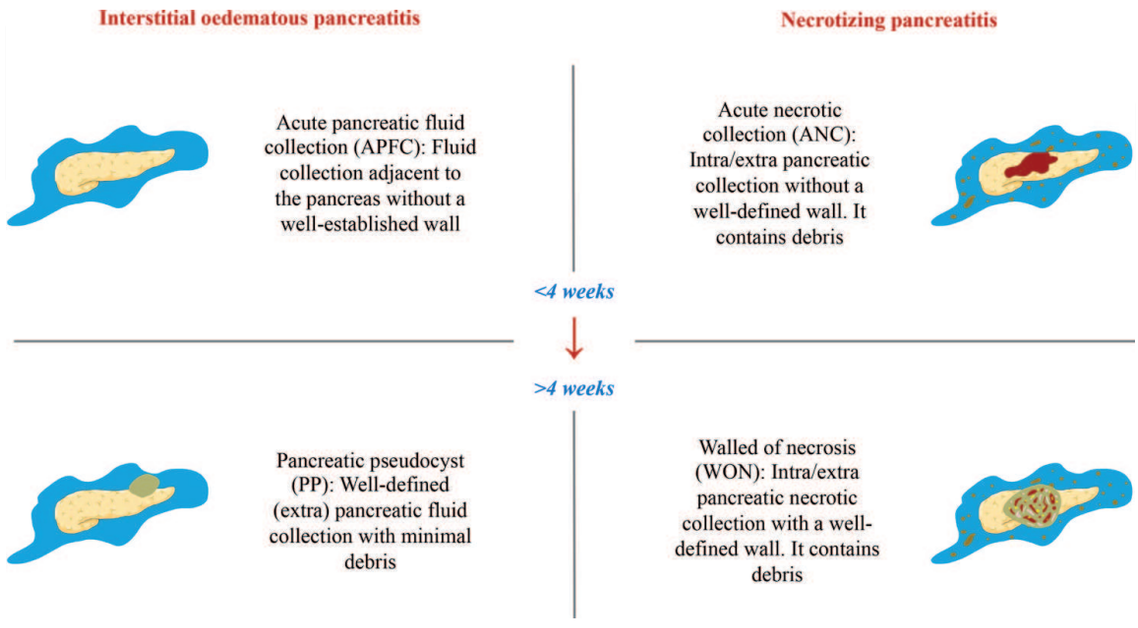

Figure 1.
Classification of pancreatic fluid collection.

in comparison with the chronic pancreatitis. Alcohol-associated pancreatitis appears to be the major causative factor in countries where alcohol consumption is high [7].

4. Pathogenesis

Several different procedures participate in the pathogenesis of the PPCs. In the cases that pseudocysts arise as a complication of severe acute pancreatitis, there is extravasation of pancreatic secretions due to disruption of the pancreatic duct. The gland necrosis leads to local fluid collection, which persists for more than 4 weeks as the inflammation recedes. Such pseudocysts usually contain enzymatic fluid and necrotic debris [8]. Concerning the pathogenesis of pseudocysts in chronic pancreatitis patients, at least two mechanisms may be involved. The cyst may develop as a consequence of progressive ductal obstruction by a protein plug, calculus, or localized fibrosis. In addition, a potentially acute exacerbation of the underlying disease can cause the cystic formation. Two-thirds of the patients with pseudocysts appear to have a connection between the pseudocyst and the pancreatic duct, while the rest do not have this exact finding and the cystic formation is caused due to the inflammatory reaction [9].

5. Classification system

Although PPC as a term is well-established, there is no classification system widely accepted. The first classification system was proposed by D'Egidio and Schein based on the underlying etiology of pancreatitis (acute or chronic), the pancreatic ductal anatomy and the presence of communication between the cyst and the pancreatic duct [10]. Using this classification system, the cyst may be divided into three distinct types:

- Type I, or acute "post-necrotic" pseudocysts, occur after an episode of acute pancreatitis and are associated with normal duct anatomy and rarely communicate with the pancreatic duct.

- Type II, also post-necrotic pseudocysts, occur after an episode of acute-on-chronic pancreatitis (the pancreatic duct is diseased, but not structured, and there is often a duct-pseudocyst communication).

- Type III, defined as "retention" pseudocysts, occur in chronic pancreatitis and are uniformly associated with duct stricture and pseudocyst-duct communication.

The latest classification system was proposed by Pan G. et al. based on the anatomical location and clinical manifestation of the pseudocysts, along with the relationship between the cyst and the pancreatic duct (**Table 1**) [11]. His aim was the selection of the optimal therapeutic approach for each stage.

6. Pancreatic pseudocysts differential diagnosis

Although PPCs are the most frequent cystic lesions, there are other malignant cystic lesions that can mimic the clinical manifestations of the PPCs. Malignant

Type	Description of pancreatic pseudocyst
I	<5 cm and without complications, symptom, and neoplasia
II	Suspected cystic neoplasia
III	The Location of pancreatic pseudocyst is uncinate
IIIa	Pseudocyst communication with the pancreatic duct
IIIb	Without communication between pseudocyst and pancreatic duct
IV	Location of pancreatic pseudocyst is head, neck and body
IVa	Exist communication between pseudocyst and pancreatic duct
IVb	Distance from the cyst to the gastrointestinal wall is <1 cm
IVc	Neither IVa nor IVb
V	Location of pancreatic pseudocyst is tail
Va	Splenic vein involvement or upper gastrointestinal bleeding
Vb	Distance from the cyst to the gastrointestinal wall is <1 cm, without splenic vein involvement or upper gastrointestinal bleeding

Table 1.
Pan G. et al. classification of pancreatic pseudocysts.

cystic lesions account for 10–15% of the pancreatic cysts [12]. It is well established, that imaging modalities alone can be misleading in diagnosing cystic malignancies due to the imaging similarities [13]. In general terms, the risk of potential malignancy in incidentally detected cysts is low [14]. The most common cystic malignancy is Branch Duct Intraductal Papillary Mucinous Neoplasm (BD-IPMN) [15]. Predictive factors for malignancy are the size of the cyst (>3 cm), dilated pancreatic duct, and the solid component associated with the cyst. Multiple cysts and cyst enlargement over time are not correlated with the appearance of neoplasm [14].

The distinction is important in order to provide the optimal therapy for the patient. The differential diagnosis should include serous cystic tumors, mucinous cystic neoplasms, solid pseudopapillary neoplasms, and the recently known Intraductal papillary mucosa neoplasm (IPMNs). In the context of absence of history of pancreatitis, the physicians should suspect malignancy and further diagnostic modalities such as image-guided aspiration/biopsy should be performed. Magnetic resonance cholangiopancreatography (MRCP) can visualize possible communication between the main pancreatic duct and a cystic lesion noninvasively. In addition, endoscopic ultrasound can provide further structure information in greater detail and facilitate aspiration or biopsy of smaller lesions [16].

7. Clinical manifestations

Patients with acute pancreatitis who are not treated within seven days or those whose symptoms reappear after a transient improvement period should be suspected of pancreatic pseudocysts. The clinical manifestations are strictly associated with the local mass effect. The symptoms and the signs are summarized in **Table 2**.

Frequency	Sign and symptom
Most frequent	Abdominal pain and early satiety
Uncomon	Fever, palpable mass, weight loss/anorexia (due to gastric duodenal compression), feeding intolerance
Rare	Jaundice (due to bile duct compression)

Table 2.
Signs and symptoms of pancreatic pseudocysts.

8. Radiological examinations

The golden standard radiological measure of the PPCs is the CT. It can visualize the size of the cyst, its shape as well as any possible association with the adjacent tissues. Also, bearing in mind that PPCs are a progressive disease, CT can facilitate the follow-up.

Regarding the US, it is a side-bed, inexpensive, and noninvasive radiological modality. Also, with its ability to measure blood flow, it is suitable to differentiate pseudoaneurysms or ruptures inside the PPC. Finally, US can serve as an imaging guide for further diagnostic and interventional methods. Despite these advantages, the most crucial problem is the visibility and the exposure of the pancreas and the peri-pancreatic region due to the bowel gas and the patient's weight. In addition, it is operator-dependent with a sensitivity in pancreatic fluid collections of approximately 75–93% [17].

Last but not least, magnetic resonance imaging (MRI) can also provide similar data for the PPCs. Its main advantage is the capacity for easier differentiation of the solid debris [18]. MRI also proved to be superior to CT in the prediction of a potential drainable peri-pancreatic fluid collection [19]. However, MRI is far more expensive than CT and its availability is limited at several institutions.

9. Pancreatic pseudocysts complications

Generally, peri-pancreatic fluid collections are sterile and most of the cases are resolved without any invasive intervention. Potentially, untreated pancreatic pseudocysts can cause life threatening complications including Infection, rupture, pancreatic fistulas and ascites, vascular complications (Pseudoaneurysm formation, Hemosuccus Pancreaticus, Splenic or Portal vein thrombosis), and splenic complications and local mass effect (Gastrointestinal, Urinary obstruction or biliary complications).

9.1 Infection

As aforementioned, peri-pancreatic fluid collections are sterile. Infected pancreatic pseudocysts occur in up to 10% of cases, usually spontaneously or after iatrogenic intervention (diagnostic or therapeutic manipulation) [20]. The most common species of pathogens that are frequently found in PPCs originated from the enteric flora and include E. coli, Klebsiella pneumoniae, Enterococcus spp., and, Enterobacter spp., less frequent are Pseudomonas aeruginosa, Streptococcus spp., Staphylococcus spp., and Bacteroides [21]. The route of the bacteria leading to infection in pancreatic

pseudocyst is still unclear. Several mechanisms have been proposed, such as infection from the biliary tree or duodenum, translocation of bacteria from the gut, and hematogenous or lymphatic spread from other sites.

Since clinical manifestations may vary, infection should be suspected in any.

patient with fever or suggestive signs or symptoms of sepsis. An infected pancreatic pseudocyst is accompanied by fever, shivering, and elevated white blood cell count. The presence of bubble gas sign on CT is a crucial finding for infection and the physician should be suspected. Nevertheless, US-guided aspiration (EUS-FNA) and sending the fluid for gram stains and cultures will provide the definitive diagnosis.

In addition, the results would provide information for the appropriate antibiotic treatment. If the acute infection is confirmed, then drainage should be performed by endoscopic, percutaneous, or surgical procedures.

9.2 Rupture

Rupture of the pancreatic pseudocyst can lead to a favorable outcome or a potentially life-threatening situation. Rupture to the adjacent gastrointestinal tract will lead to vomiting, diarrhea, melena, hematemesis, or hematochezia. However, rupture into the peritoneal cavity can cause severe peritonitis or hemorrhagic shock and pancreatic ascites. Its clinical manifestation includes severe abdominal pain, fever, food intolerance, tachycardia, and hypotension. Intraperitoneal hemorrhage from ruptured pancreatic pseudocyst is associated with an extremely high mortality rate (35.3%) [22]. The exact mechanism of rupture remains unknown. Possibly, erosion or disruption due to either severe inflammation or the activated lytic enzymes in the pseudocyst, in a superficial vessel may have weakened the pseudocyst wall, subsequently resulting in the spontaneous rupture of the pseudocyst [23]. The content of the pseudocyst (amylase, lipase, and other proteolytic enzymes) can cause erosion of the nearby viscera, thrombosis of the adjacent vessels, or further complications [24].

Traditionally, the optimal therapeutic choice is the internal drainage either through cysteogastrostomy or Roux-en-Y cysteojejunostomy [25]. Extensive local inflammation or incapability of identifying the cyst walls can lead to the failure of creation of the anastomosis. In these cases, external drainage and lavage of the peritoneal cavity can be achieved with safety [9]. Recently, another option, which was reported, is the endoscopic ultrasound-guided drainage and endoscopic ultrasound-guided gastrocystostomy with a fully covered self-expandable metallic stent [26]. However, the authors highlighted that can be useful in local fluid collection due to the ruptured pseudocyst.

Regarding the ruptured pseudocysts in nearby viscera, the literature recommends conservative treatment unless there is active bleeding, or the patient is febrile. The most common site seems to be the stomach, but there is not enough data to support this. Beside the conservative treatment, the authors recommend endoscopic intervention (potential clipping of bleeding vessels, stenting) as first choice of treatment and surgical intervention when endoscopic management is impossible (gastrectomy) [23, 27–29].

9.3 Pancreatic fistula and ascites

A big majority of patients with acute pancreatitis will develop pseudocysts, while only a small percentage of them will develop fistula or ascites as pseudocyst complications. There is no data regarding the mechanism for the creation of the fistula.

Fistulas are divided into two categories: internal which include fistulas associated with the adjacent viscera to the pseudocyst; andnd external, mainly due to iatrogenic manipulations. Connection from the pseudocyst to the stomach, colon, small intestine, bronchi, biliary tract, and esophagus have been described. Early recognition of this rare entity is crucial. CT, MRI, and MRCP have a principal role. In addition, fistulography has been proven trustworthy for a definitive diagnosis [30]. Like the aforementioned complications, a stepwise approach is the key starting from conservative treatment to endoscopic or surgical interventions.

The external pancreatic pseudocyst fistulas can mostly occur as a complication of the percutaneous drainage. On suspicion, any aspired fluid must be checked for amylase levels ensuring the diagnosis. Also, another option is to inject a contrast media through the drain or fistula to assess for a pancreatogram, which confirms the diagnosis. Initial treatment is considered to be conservative as in the majority of the cases, fistulas are resolved without any intervention [31]. Although external fistulas are iatrogenic complications, there are a few cases that have been reported with spontaneous pancreatocutaneous fistula [32, 33]. In both cases, pseudocyst occurred retroperitoneally with swelling at the left lumbar and left flank region accordingly. In the first case, conservative treatment was chosen while the second one underwent surgical drainage. Both cases had favorable outcomes.

Ascites are another complication of the pancreatic pseudocyst. In most of the cases (about 80%), ascites appears due to leakage of the pseudocyst in patients with chronic pancreatitis [34, 35]. Patients with pancreatic ascites usually refer to mild abdominal pain, decreased appetite, sense of satiety, and weight loss. One very important leading point is the medical history of patient, which must include chronic pancreatitis or a recent episode of acute pancreatitis. The diagnosis is set by drainage and the ascitic fluid has high amylase concentration (over 1000 IU/L) and protein concentration over 3 g/dl, which differentiates it from cirrhosis, tuberculosis, or malignancy [36]. Imaging modalities that could lead to diagnosis is the endoscopic retrograde cholangiopancreatography (ERCP) which is the "gold standard" to confirm the site of leakage; while in cases where ERCP is contraindicated, MRCP can define the anatomy of pancreatic duct and its abnormalities [37, 38]. Treatment of this entity concerns mainly the therapy of the pancreatic pseudocyst. Conservative treatment, drainage either internal (cystogastrostomy, cystojejunostomy, or cystoduodenostomy) or external and distal pancreatectomy when the leak is in the pancreatic tail are possible options [37].

9.4 Vascular complications

Patients suffering from pancreatic pseudocysts can potentially develop vascular complications, such as pseudoaneurysm formation within the cyst, splenic and portal vein complications.

Formation of pseudoaneurysm inside the pancreatic pseudocyst is a rare pathology and life-threatening situation with high mortality rates. The exact mechanisms are still under investigation, but three possible mechanisms have been proposed. Firstly, inflammation in conjunction with pancreatic enzymes could lead to erosion of pancreatic or peripancreatic artery and consequently the formation of pseudoaneurysm; communication of a pancreatic pseudocyst with a vessel; and lastly a pseudocyst eroding the bowel wall with bleeding [39, 40]. The symptoms are nonspecific, and even on suspicion the patient must undergo a thorough examination to avoid any rupture resulting in severe bleeding. Contrast-enhanced CT or angiography if the

patient is stable is used for recognition of the vessel. In addition, angiography can be used for immediate angio-embolization after tracking the bleeding site. Endovascular interventions should be the first-line treatment [41]. In case of unsuccessful endovascular intervention, a surgical treatment should be performed. The general idea is drainage of the pancreatic pseudocyst and arterial ligation of the vessel that causes the pseudoaneurysm. Splenic artery is the most frequent vessel involved [42].

A pseudocyst can also be the cause of portal vein or splenic vein thrombosis. Pathophysiologically, local inflammation and complement system activation can contribute to thrombosis. In addition, pseudocyst can compress the portal or splenic vein leading to obstruction and consequently to portal hypertension. Treatment includes management of the pancreatic pseudocyst and its cause, e.g. lithrotripsy if choledocholithiasis exist, and management of the thrombosis. Anti-coagulation therapy, thrombolytic agents (urokinase), endovascular intervention (transjugular intrahepatic portosystemic shunt) as well as surgery have been described [43–45].

Last but not least, communication between the pseudoaneurysm and the pancreatic duct can result in severe bleeding to gastrointestinal tract through the ampulla of Vater. This life-threatening situation is called hemosuccus pancreaticus also known as wirsungorrhagia and pseudohemobilia. The most frequent clinical manifestation includes melena, hematochezia or hematemesis, symptomatic anemia, abdominal pain, nausea, and vomiting [46]. The "gold standard" diagnostic as well as therapeutic modality is the angiography identifying the causative vessel and applying the proper interventional method (stent placement and metallic coil embolization). In patients whose endoscopic intervention failed, or in those that are unstable, surgery is still an option without experiencing unwanted complications [47].

9.5 Splenic complications

Splenic rupture in acute and chronic pancreatitis accounts for 9% of the atraumatic splenic ruptures [48]. Especially, if a pancreatic pseudocyst occurs at the tail of the pancreas, the pancreatic enzymes and the inflammation can erode the splenic parenchyma secondary to hematoma. The main etiological factor is excessive alcohol consumption, while the majority of patients are referring to abdominal pain, nausea, vomiting, and lumbar pain [49]. Early recognition of this complication with CT and/or angiography is important for the immediate therapeutic approach, which is consisted of conservative management, percutaneous drainage, splenic artery embolization (hematoma exists without rupture), and splenectomy (when a rupture occurs) [50–52].

Other splenic complications, such as splenic artery pseudoaneurysm and splenic vein thrombosis are described in the "vascular complications" session.

9.6 Local mass effect

There have been reported cases in which the pancreatic pseudocyst caused compression to the adjacent viscera due to its huge size. Additionally, a big pancreatic pseudocyst can increase the intra-abdominal pressure leading to orthopnea, dyspnea, abdominal pain, and distention. Depending on the region of the cyst, the common bile duct and the poral vein or the splenic vein could be obstructed resulting in obstructive jaundice and portal hypertension (see session "vascular complications") accordingly [53, 54]. Endoscopic approach reducing the size of the cyst combined with stenting is the ideal treatment for this situation.

10. Conclusions

PPC is a frequent complication of acute or chronic pancreatitis. Maturation of the pseudocyst needs at least 2–6 weeks. In this short period of time, the majority of them are resolved without any invasive treatment. Patients with persistent symptoms should be examined thoroughly. Early recognition of the complication of the pancreatic pseudocyst is mandatory. An abdominal CT scan is the initial radiological modality. Multidisciplinary and stepwise approaches to evaluating the data properly will lead to favorable outcomes for the patient. The physicians should be suspicious of these aforementioned rare complications, which can potentially be fatal.

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