Imaging of acute pancreatitis and its complications. Part 2: Complications of acute pancreatitis

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Abstract The Atlanta classification of acute pancreatitis was introduced in 1992 and divides patients into mild and severe groups based on clinical and biochemical criteria. Recently, the terminology and classification scheme proposed at the initial Atlanta Symposium have been reviewed and a new consensus statement has been proposed by the Acute Pancreatitis Classification Working Group. Major changes include subdividing acute fluid collections into "acute peripancreatic fluid collection" and "acute post-necrotic pancreatic/peripancreatic fluid collection (acute necrotic collection)" based on the presence of necrotic debris. Delayed fluid collections have been similarly subdivided into "pseudocyst" and "walled of pancreatic necrosis". Appropriate use of the new terms describing the fluid collections is important for management decision-making in patients with acute pancreatitis. The purpose of this review article is to present an overview of complications of the acute pancreatitis with emphasis on their prognostic significance and impact on clinical management and to clarify confusing terminology for pancreatic fluid collections.

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Acute pancreatitis is an acute inflammatory disease of the pancreas that may also involve peripancreatic tissues and even remote organs. Patients with acute pancreatitis may present with a mild, self-limiting disease without complications or severe disease, which results in local or systemic complications with significant morbidity and mortality. Different clinical or radiological scoring systems to predict severity and outcome in acute pancreatitis have been developed since the early 1980s [1–5]. Validation and comparison of the different scoring systems are complicated by confusing and incompatible use of terminology and definitions of severity, complications, and outcome of the disease. In 1992, the Atlanta Symposium developed a consensus statement that specifically defined both severe acute pancreatitis and its complications [1]. As morphologic abnormalities of the pancreas were used in this classification, it recognized the important role of computed tomography (CT) in describing the disease severity. The authors defined severe acute pancreatitis as “acute pancreatitis with organ failure and/or local complications, such as abscess, pseudocyst, or necrosis”. Recently, the terminology and classification scheme proposed at the initial Atlanta Symposium have been reviewed, and a new consensus statement has been proposed [6]. Major changes include subdividing acute fluid collections into “acute peripancreatic fluid collection (APFC)” and “acute post-necrotic pancreatic/peripancreatic fluid collection (PNPFC)” based on the presence of necrotic debris. Delayed fluid collections have been similarly subdivided into “pseudocyst” and “walled of pancreatic necrosis (WOPN)”. The terms, such as pancreatic abscess, and hemorrhagic pancreatitis have been abandoned. Appropriate use of the new terms describing these fluid collections is important for management decision-making in patients with acute pancreatitis.

Currently, contrast enhanced CT has a crucial role in evaluating the extent and evolution of the acute pancreatitis and its complications [7]. Magnetic resonance imaging (MRI) is especially useful for imaging of patients with iodine allergies or renal insufficiency, characterizing collections and assessment of an abnormal or disconnected pancreatic duct. A T2-weighted image is more sensitive than CT in the assessment of internal contents of fluid collections and therefore in the evaluation of their drainability [8,9]. Ultrasound may be helpful when there is concern whether a pseudocyst or a WOPN is the correct diagnosis, especially if MRI is not readily available.

The purpose of this review article is to present an overview of complications of the acute pancreatitis with emphasis on their prognostic significance and impact on clinical management and to clarify confusing terminology for fluid collections.

Pancreatic necrosis

Pancreatic parenchymal necrosis, developing as a result of the thrombosis of the pancreatic microcirculation, is defined as diffuse or focal areas of non-viable pancreatic parenchyma that typically are associated with peripancreatic fat necrosis. In general, it emerges 24–48 hours after the onset of acute attack and it is usually well established with contrast enhanced CT or MRI performed 48–72 hours after the onset of acute attack [7,10,11]. The revised Atlanta classification system distinguishes three forms of acute necrotizing pancreatitis, depending on location: pancreatic parenchymal necrosis alone, peripancreatic fat tissue necrosis alone and peripancreatic parenchymal necrosis with peripancreatic fat tissue necrosis [6]. Pancreatic parenchymal necrosis alone can be seen in fewer than 5% patients and usually involves the body or the tail of the pancreas. In the first week, contrast enhanced CT demonstrates necrosis as a more homogeneous non-enhancing area of variable attenuation and, later in the course of the disease, as a more heterogenous area [12]. This is the result of a process in which the non-viable and necrotic tissues (pancreatic parenchyma and peripancreatic fat tissue) slowly begin to liquefy. The extent of pancreatic parenchymal necrosis is divided into three categories: less than 30%, 30–50% and greater than 50% of the gland involved. Approximately 20% of patients, with only peripancreatic fat tissue necrosis without pancreatic gland necrosis may occur [13]. Its presence is diagnosed when heterogeneous areas of non-enhancement are visualized that contain non-liquified components. Because CT cannot reliably diagnose retroperitoneal fat necrosis, it has been suggested that all heterogeneous peripancreatic collections should be considered as areas of fat tissue necrosis unless proven otherwise [7]. Patients with peripancreatic necrosis alone have a better prognosis than the patients with pancreatic parenchymal necrosis but have a higher morbidity rate than patients with interstitial edematous pancreatitis only [14]. Pancreatic parenchymal necrosis with peripancreatic fat tissue necrosis is the most common type and can be seen 75–80% of patients with acute necrotizing pancreatitis [13] (Fig. 1).

The head and tail of the pancreas are protected, while the neck and/or body of the pancreas are completely necrosed, existing almost always with the disrupted continuity of pancreatic duct (disconnected pancreatic duct syndrome) (Fig. 2). The diagnosis of the disconnection of the main pancreatic duct requires the visualization of a necrotic region of at least 2 cm in size, viable tissue proximal to the necrosis, and extravasation at pancreatography [15]. Since the pancreatic fluid secreted by the caudal part of the pancreas cannot be drained by the pancreatic duct, this situation leads to complications, such as persistent fluid collection, fistula, ascites or pleural effusion.

Infection of the pancreatic necrosis results from secondary bacterial contamination of the necrotic pancreatic and peripancreatic tissues, especially with Gram (−) enteric basilli. The incidence of infection increases in the cases with prolonged stay at the hospital (about 60% above 3 weeks) [16]. Suspect of infected pancreatic necrosis will arise if the cases with necrosis findings found on CT scans also have the clinical picture of sepsis. This is almost always a poor prognostic factor and infected pancreatic necrosis is accounting for about 80% of the deaths from acute pancreatitis [17]. It does not have any specific findings on CT, except for the air bubbles seen in the necrotic pancreatic tissue. The diagnosis can be confirmed with fine-needle aspiration biopsy accompanied by US or CT. Aggressive surgical approaches, such as necrosectomy and
debridement may be needed to treat this clinical condition [18,19].

**Fluid collections associated with acute pancreatitis**

Acute pancreatitis may be accompanied by pancreatic parenchymal or peripancreatic fluid collections. The revised Atlanta classification subdivides acute fluid collections in the first 4 weeks into APFC and PNPFC based on the presence of necrotic debris and delayed fluid collections into pseudocyst and WOPN [10]. Interstitial edematous pancreatitis can be associated with APFC and, over time, with pancreatic pseudocysts. Acute necrotizing pancreatitis can be associated with PNPFC and, over time, with WOPN. All of these collections can be sterile or infected.

**Acute peripancreatic fluid collections**

APFC is a collection of enzyme-rich pancreatic juice predominantly collected adjacent to the pancreas. This collection develops within the first 48 hours in 30–50% of patients with acute pancreatitis [20,21]. They are resulted from pancreatic and peripancreatic inflammation or by rupture of one or more small peripheral pancreatic side duct branches. APFC is most frequently collected in the lesser sac, but may be seen in the anterior pararenal space (most commonly left), transverse mesocolon, mesenteric root and gastro-hepatic, gastrosplenic and gastrocolic ligaments [20–22] (Figs. 3 and 4). Most of the APFCs remain sterile and disappear spontaneously within 2–4 weeks in 50% of the patients. Intervention at this stage is to be avoided, since drainage or aspiration of fluid could introduce infection [12]. Only the rare infected APFC necessitates drainage. When APFCs

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**Figure 1.** Pancreatic necrosis in a 68-year-old woman with gallstones. Contrast enhanced CT image at the portal venous phase (a), obtained 3 days after the onset of acute attack, show full width necrosis (N) of the pancreatic neck, body and proximal tail. Parenchyma of the distal tail is seen to enhance normally. Contrast enhanced CT image (b), obtained 4 weeks after the onset of acute attack, show a walled of pancreatic necrosis (WOPN) containing necrotic fatty tissue. Intraabdominal fluid collection is also seen (P: pancreas).

**Figure 2.** Disconnection of the pancreatic duct in a 49-year-old man with prior episodes of alcoholic pancreatitis. Contrast enhanced CT image, obtained 3 weeks after the onset of acute attack, reveals necrosis (arrow) of the entire width of the proximal body of the pancreas (P), which communicates with a large fluid collection (F). Disconnected pancreatic duct was proved at surgery.

**Figure 3.** Interstitial edematous pancreatitis in a 48-year-old woman with gallstones. Contrast enhanced CT image obtained at admission, reveals heterogeneous enhancement of the pancreatic parenchyma due to edema and acute peripancreatic fluid collection (F) predominantly collected in the left anterior pararenal space (P: pancreas).
do not resolve, they evolve into pseudocysts after at least 4 weeks [21].

In the first week of acute pancreatitis, differentiation between APFC and acute necrotic collections may be difficult, since both fluid collections may appear as areas of non-enhancement. If non-enhancing components of variable attenuation are seen in these collections, the diagnosis of peripancreatic necrosis with non-liquified components (hemorrhage, fat, and/or necrotic fat) is suggested [12]. In these cases, the process should be diagnosed as acute necrotizing pancreatitis with peripancreatic necrosis alone, not interstitial edematous pancreatitis [14]. After 1 week from onset, these fluid collections become clearly heterogeneous, and necrosis can be diagnosed on contrast enhanced CT images.

Pancreatic pseudocysts

Pancreatic pseudocyst is defined as a fluid collection of pancreatic juice enclosed by a non-epithelialised wall of fibrous or granulation tissue [1]. Pseudocysts are defined as a collection 4 weeks from the onset of interstitial edematous pancreatitis and they occur in approximately 10–20% of cases. Pseudocysts are most frequently developed in the lesser sac, although they may be seen anywhere from the mediastinum to the pelvis [23–25] (Figs. 5 and 6). On contrast enhanced CT, pseudocysts are usually seen as a thin-walled (1–2 mm), round- or oval-shaped cystic lesion with a density < 20 HU [21,22,26]. Their walls may be thick and irregular and develop calcification over the time [22]. Enhancement may be observed in the walls on contrast enhanced CT or MRI [26]. Pseudocysts contain no non-liquefied components within the fluid collection [6]. Pancreatic pseudocysts have been reported to communicate with the pancreatic duct in between 25–58% of cases [22]. Demonstration of the presence or absence of communication with the pancreatic duct is important because it can help determine management.

The differentiation of pancreatic pseudocysts from cystic tumors of the pancreas can be difficult, a point emphasized in a series by Warshaw et al. who reported that 30% of cystic tumors of the pancreas were initially considered pancreatic pseudocysts [27]. Clinical history is important in making this distinction. If the patient does not have a history of acute pancreatitis or imaging features suggest septations, or wall nodularity, the possibility of cystic neoplasm of the pancreas should be considered [27]. Periphereal solid nodules or intraluminal enhancement are not seen in pseudocysts [22,27,28].

Approximately 50% of the pseudocysts are asymptomatic and resolve spontaneously over the time [22]. Occasionally, spontaneous drainage into the adjacent stomach or transverse colon may develop [29]. Only half of the
non-spontaneously resolved pseudocysts cause clinical symptoms or complications, such as pain, secondary infection, hemorrhage related to the erosion of adjacent vessels, systemic inflammatory response syndrome due to the rupture into the peritoneal cavity and bile duct obstruction or gastric outlet obstruction due to the mass effect [30]. Pseudocysts should be treated with percutaneous or endoscopic drainage or surgically; if they are symptomatic, their size is over 5 cm or gradually increasing, and if they persist longer than 6 weeks [22,30–34].

According to the new definition, pancreatic pseudocysts should be described as non-infected or infected. Infected (suppurative) pseudocyst is the new name for what had been described in the Atlanta Symposium as a pancreatic abscess [1,6]. An infected pseudocyst is a well-circumscribed, pus containing, encapsulated fluid collection near the pancreas [1,27]. On contrast enhanced CT images, the wall of the infected pseudocyst is thicker and more irregular than that of a sterile pseudocyst [29]. Air bubbles or air-fluid level may be seen within the pseudocyst in 20% of the patients [21] (Fig. 7). In these cases, infected pancreatic necrosis and retroperitoneal enteric fistula should be considered in the differential diagnosis [21,28,29].

Post-necrotic pancreatic/peripancreatic fluid collections (PNPFC) (acute necrotic collections)

PNPFC develops as a result of the pancreatic glandular and/or peripancreatic fatty tissue necrosis to become liquefied over the time. This collection contains liquefied, necrotic fatty tissue and pancreatic and extrapancreatic solid necrotic debris [6] (Fig. 8). Its content is solid or liquid depending on the time elapsed since the onset of the disease. Within the first weeks of onset of the acute necrotizing pancreatitis, any collection in the pancreas that replaces pancreatic parenchyma should be considered a PNPFC and not a pseudocyst. PNPFCs are often connected to the main pancreatic duct since they show association with the disrupted integrity of the pancreatic duct [35]. Collection content may be sterile or infected.

![Figure 7](image-url) An infected pseudocyst (Ps) in a 31-year-old woman with gallstones. Contrast enhanced CT image, obtained 8 weeks after the onset of acute attack, reveals an encapsulated, rounded fluid collection (Ps) with septations and a thick and irregular wall increased contrast enhancement. Note air bubbles within the pseudocyst (P: pancreas).

Walled off pancreatic necrosis

Similar to the development of a pseudocyst from APFC over the time, WOPN evolves from the PNPFC, and results in a non-epithelialised thick wall developed between the necrosis and the adjacent viable tissue after 4 weeks or longer [6]. WOPN replaces the formerly used terms of "organized pancreatic necrosis", "pseudocyst associated with necrosis", "central cavitary necrosis" and "necroma" [36]. Similarly, PNPFC, WOPN may involve the pancreatic parenchymal tissue and/or peripancreatic tissue. Any apparent fluid collection that occupies or replaces portions of the pancreatic parenchyma should be called a WOPN after 4 weeks from the onset of acute necrotizing pancreatitis. WOPN is an irregular, partially liquefied collection, which may contain solid necrotic debris and may expand to the peripancreatic space [37] (Figs 1b, 8b, 9). Solid components in these

![Figure 8](image-url) Necrotizing acute pancreatitis in a 45-year-old man with gallstones. Contrast enhanced CT images (a) obtained 3 weeks after the onset of acute attack reveal a post-necrotic pancreatic/peripancreatic fluid collection (PNPFC) contained solid necrotic debris. A follow-up contrast enhanced CT images (b), obtained 6 weeks after the onset of acute attack, show an encapsulated walled off pancreatic necrosis (WOPN), evolving from PNPFC, contained pancreatic/extrapancreatic solid necrotic debris. Note the hyperdense stones in the gallbladder (long arrow) and common bile duct (short arrow) (P: pancreas).
collections are identified better on US and T2-weighted MR images than on CT [9] (Fig. 10). Collection content may be sterile or infected (Fig. 11). In the absence of gas within the collection, diagnosis of infection can be obtained only by performing fine-needle aspiration of the collection with a positive Gram stain and culture for bacteria or fungal organisms [14].

In the past, evolving necrotic collections, now termed WOPN, have been mistaken for pseudocysts. This error results in inadequate drainage planning and ultimately might lead to treatment failure. The existence or non-existence of necrosis to on the CT performed in the beginning of acute pancreatitis, and clinical course enables to differentiate the WOPN from a pseudocyst, although sometimes this may be difficult [37]. Differentiation of these two clinical conditions is of importance since each of them has a different treatment. Ideal treatment of WOPN is controversial and most centers prefer the treatment with operative necrosectomy in the infected or symptomatic cases. However, laparoscopic, percutaneous and endoscopic transgastric or transduodenal approaches are used recently with increasing frequency in the treatment of this clinical situation. While multiple with large-bore catheters and aggressive irrigation are required to discharge the solid components in the cavity for endoscopic treatment of WOPN, simple drainage with a single catheter is mostly sufficient for the pseudocysts and abscesses [38]. Findings that are in favour of WOPN include having a larger size, extension to the paracolic or retrocolic space, an irregular wall definition, the presence of solid or fat attenuation debris, presence of pancreatic parenchymal deformity and discontinuity, and absence of dilatation of the main pancreatic duct [37]. In WOPN, the main pancreatic duct does not dilate due to the leakage of pancreatic juice into the peripancreatic fluid via often associated disruption of the main pancreatic duct or its side branches. Whereas, dilatation of the main pancreatic duct caused by the compression of the pancreatic parenchyma by pseudocyst or due to proximal ductal stricture is a finding in favour of pseudocyst [37]. The infected pseudocysts tend to have a thick irregular wall with thick or multiple septations similar to WOPN, but tend to be small and often have main pancreatic duct dilation.

**Vascular complications**

Vascular complications occur in 25% of patients with acute pancreatitis [39–41]. The most common complications are thrombosis of the portal venous system, hemorrhage related to the erosion in arteries of the upper gastrointestinal system and pseudoaneurysm development [40,41].
Splenic vein thrombosis is the most common (10–40%) complication of acute pancreatitis and results from the inflammatory intimal injury or the external compression by fluid collections [38,39] (Fig. 12). This may result in portal hypertension, variceal development and splenic infarction in long term [39]. Although rare, thrombosis of portal vein or superior mesenteric vein may also be seen [42].

Spontaneous arterial hemorrhage in acute pancreatitis is a rare, but crucial complication. Erosion of pancreatic or peripancreatic arteries by the proteolytic enzymes may result in a free hemorrhage or pseudoaneurysm development [43–45] (Fig. 13). The most commonly affected arteries are the splenic artery (40%), gastroduodenal artery (30%) and the pancreaticoduodenal artery (20%) [43–45]. The bleeding may be into the gastrointestinal tract as well as into the peritoneal cavity. Pseudoaneurysms may rupture into the peritoneal cavity, retroperitoneum, pseudocyst, and rarely into the pancreatic duct [43]. The latter refers to “hemosuccus pancreatitis” and the bleeding from the ampulla vateri is seen on endoscopic examination [46,47]. If the wall of pseudocyst involves a visceral artery, this condition refers to “pseudoaneurysmatic pseudocyst” [48]. Adding arterial phase to CT or MRI protocol will allow optimal evaluation of the active bleeding and pseudoaneurysms.

**Figure 12.** Walled off pancreatic necrosis (WOPN) and venous thrombosis in a 61-year-old man with alcoholic acute pancreatitis. Contrast enhanced CT image at the portal venous phase, obtained 8 weeks after the onset of acute attack, shows a multiloculated WOPN with an enhanced irregular thick wall. Note the filling defects in the main portal vein consistent with thrombus (arrow).

**Figure 13.** Intracystic hemorrhage in 41-year-old man with an idiopathic recurrent acute pancreatitis. Contrast enhanced CT images at the arterial (a) and portal venous (b) phases reveal active hemorrhage into the pseudocyst (Ps) from the common hepatic artery (black arrow). High attenuation fluid (F) consistent with free intraabdominal hemorrhage is also seen. Note a filling defect in the splenic vein due to a thrombus (white arrow).
Conclusion

Acute pancreatitis is associated with a wide variety of complications affecting the gland and the surrounding structures. Contrast enhanced CT is the primary imaging modality for initially identifying local complications. MRI or ultrasound examination can be useful to evaluate the content of fluid collections. The use of a new standardized terminology is important to facilitate communication between radiologists, gastroenterologists, and surgeons.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References


