Acute Pancreatitis - Spectrum of imaging findings based on the Revised Atlanta Classification

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Learning objectives

- Review acute pancreatitis imaging features, focusing on the differentiation between interstitial oedematous pancreatitis and necrotizing pancreatitis, and between the various pancreatic and peripancreatic fluid collections.
- Review the regional complications.
Background

Acute pancreatitis (AP) is an inflammatory process of the pancreas that classically presents with abdominal pain and elevated serum pancreatic enzymes. It is a common cause of hospital admission with an annual incidence of 13-45 cases per 100000 persons [1]. Most cases are mild and resolve within the first week with adequate treatment. However, some patients progress to organ failure and/or local complications, in which cases the mortality can range to 30% [1].

Imaging plays an important role in the management of patients with AP, especially in severe AP and the use of a standardized nomenclature is crucial. The Atlanta Classification of 1992 [2] provided a set of terms describing the various features of AP. However, current knowledge of pathophysiology, new advances in imaging and treatment options required an update. This was accomplished in 2012, with the publishing of the Revised Atlanta Classification [3]. New concepts were added, while some confusing terms were abandoned and a special focus were given to the various morphologic aspects of AP as seen at imaging.
Findings and procedure details

Diagnosis

The diagnosis of acute pancreatitis relies on the presence of two or more of the following criteria:

- typical abdominal pain,
- elevated serum lipase (three or more times the upper limit of normal) and
- characteristic imaging findings.

More often, imaging is not necessary to establish the diagnosis. When needed, usually in patients with prolonged symptoms and non-diagnostic serum lipase levels or in the sedated patient, contrast-enhanced computed tomography (CECT) is the preferred imaging method. Other indications to perform CECT early in the course of the disease are to confirm severe pancreatitis based on clinical predictors and no improvement or clinical deterioration with the initial treatment.

The protocols vary among institutions, but contrast medium (typically 100-150mL, at a rate of 3-5mL/sec) is necessary for necrosis detection. Images should be acquired at pancreatic phase (late arterial phase) and/or portal phase. Usually a single-phase scan is sufficient and permits the dose of radiation to the patient to be reduced. In patients whom contrast medium is contraindicated, magnetic resonance imaging should be considered. Upper abdominal ultrasonography is indicated at admission to look for a causative agent, namely choledocholithiasis.

Phases and classification based on severity of acute pancreatitis

Two phases are described in the revised Atlanta Classification: the early phase within the first week and the late phase that could last weeks to months. This distinction is not rigid so both phases should be viewed as a continuum. Noteworthy, the onset of AP is defined as the first day of abdominal pain.

In the early phase predominates a systemic inflammatory response and the management of the patient is guided by clinical and laboratory indicators. Imaging has a limited role in this phase, and is usually not required.

The late phase is defined as persistent organ failure and development of local complications. CECT is warranted in this phase, because identification and characterization of these local complications can impact on the treatment.

Three degrees of severity are defined in the Revised Atlanta Classification.
Mild acute pancreatitis doesn’t involve organ failure or local or systemic complications. It usually resolves within the first week and imaging is not necessary.

Moderately severe acute pancreatitis courses with transient organ failure (<48 hours) or local or systemic complications.

Severe acute pancreatitis manifests as organ failure lasting >48 hours.

Classification of acute pancreatitis base on morphology

The Revised Atlanta Classification divides AP in two types, based on the absence or presence of necrosis: interstitial oedematous pancreatitis and necrotizing pancreatitis. CECT is needed to make this distinction.

Interstitial oedematous pancreatitis

This is the most common type of acute pancreatitis. On CECT, there’s enlargement of the pancreas, either diffuse or focal with peripancreatic fat stranding. The pancreatic parenchyma enhances homogeneously and only pure fluid collections are seen.

Necrotizing pancreatitis

Necrosis can involve the pancreatic parenchyma, the peripancreatic fat or both. The most common scenario is combined pancreatic and peripancreatic necrosis. On CECT, the pancreatic necrosis is seen as areas of non-enhanced parenchyma, usually associated with heterogeneous fluid collections containing non-liquefied debris. Peripancreatic necrosis is more difficult to diagnosis, but should be suspected when heterogeneous collections are seen. In the first few days, CECT can underestimate the degree of necrosis, because of perfusion alterations caused by inflammation. CT Severity Index should only be assessed after 72 hour of the onset of AP.

Pancreatic and peripancreatic collections

Regarding the fluid collections seen in acute pancreatitis, they are divided and named according to their content and time of onset (before or after 4 weeks).

Collections that contain only pure fluid appear in the setting of oedematous pancreatitis and are termed acute pancreatic fluid collections (APFC) or pseudocyst, if developed in the first 4 weeks or after, respectively. In necrotizing pancreatitis, the collections contain, besides fluid, necrotic tissue and are termed acute necrotic collections (ANC) and walled-off necrosis (WON) in the first 4 weeks and after 4 weeks, respectively. Any of this fluid collections can become infected.
Acute pancreatic fluid collections Fig. 1 on page 9 Fig. 2 on page 8

These are peripancreatic collections that contain only pure fluid seen within the first 4 weeks of oedematous pancreatitis. On CECT, they are homogeneous and, as they lack a capsule, tend to conform to the retroperitoneal spaces. APFC usually resolve spontaneously within the first 4 weeks.

Pseudocyst Fig. 3 on page 8

Pancreatic pseudocyst results from encapsulation of an APFC that didn't resolve after 4 weeks. As such, they are seen in the setting of oedematous pancreatitis. An exception, is a pseudocyst resulting from a disconnected duct in a patient with previous necrosectomy. On CECT, pseudocysts are seen as rounded or ovoid homogeneous fluid collection contained by a well-defined capsule. Pseudocysts are anecogenic on ultrasound and hyperintense on MR T2-weighted images. These imaging modalities can be useful if doubt persists after CECT.

Acute necrotic collection Fig. 6 on page 10 Fig. 9 on page 13

This type of fluid collection presents in the first 4 weeks of necrotizing pancreatitis, and can be peripancreatic or, contrarily to APFC, pancreatic. On CECT, besides fluid, ANC contain non-liquefied debris (fat or soft-tissue components), are heterogeneous and loculated, but with no capsule. In the early stages (usually the first week), they can appear homogeneous, and be difficult to differentiate from APFC. Repeating the examination in the second week allows the diagnosis of ANC.

Walled-off necrosis Fig. 10 on page 11 Fig. 11 on page 11

After 4 weeks, ANC may organize and develop a capsule resulting in a WON. They can be pancreatic or extrapancreatic, including distant locations, and as with ANC, they demonstrate non-liquefied debris on CECT.

Infection

Any fluid collection described above can become infected, although this complication is more common in ANC and WON. On CECT, there are no reliable signs of infection, although the presence of gas within the collection should alert for this scenario. In doubtful cases, aspiration and culture of the fluid may be necessary.

Local complications

Besides infection, other local complications include biliary obstruction, pancreatic duct strictures or disconnection, and vascular complications. The most common vascular complication is vein thrombosis Fig. 4 on page 13 Fig. 12 on page 12, usually involving the splenic vein. Others include pseudoaneurysm formation and haemorrhage.
Fig. 2: Contrast-enhanced CT scan of the same patient of fig. 1 performed 16 days after the onset of epigastric pain shows the pancreatic collections starting to organize (arrow). They are still called acute pancreatic fluid collections since they are seen within 4 weeks of disease onset and contain no debris.

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Fig. 3: Contrast-enhanced CT scan of the performed 7 weeks after the onset of edematous pancreatitis shows an organized collection of pure fluid adjacent to the pancreatic tail (arrow). This type of collection are named pseudocysts.

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**Fig. 1:** Contrast-enhanced CT performed 4 days after the onset of epigastric pain shows pancreatic enlargement with homogeneous parenchymal enhancement. The surrounding collections contain pure fluid and are named acute pancreatic fluid collections. Fatty liver infiltration is also seen.

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**Fig. 6:** Non-enhanced CT scan (A) performed during the 2nd week of disease shows a fluid attenuation area containing fat within it (arrows), adjacent to the pancreatic body. This represents an acute necrotic collection. In patients who have contraindications to
the administration of iodinated contrast, the finding of fat within a collection makes the diagnosis of necrotizing pancreatitis. Contrast-enhanced CT scan (B) of the same patient, demonstrate the collection to a better extent. The necrosis was mainly peripancreatic, with no parenchymal areas of non-enhancement.

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**Fig. 10:** Contrast-enhanced CT scan performed after 4 weeks of disease onset. The collection shows a thick capsule and containing necrotic debris - walled-off necrosis (arrows).

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**Fig. 11:** Contrast-enhanced CT scan (A and B) shows fluid collections surrounding the pancreas in a patient with a previous history of necrotizing pancreatitis. The collections are predominant of fluid but a few areas of fat density can be seen within the collection anterior to the pancreatic body (arrow), making the diagnosis of walled-off necrosis.
Upper abdominal ultrasound (C) can identify necrotic debris in a predominantly liquid collection and in this case shows a heterogeneous collection.

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**Fig. 12:** Contrast-enhanced CT scan shows portal vein thrombosis associated with necrotizing pancreatitis.

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**Fig. 5:** Contrast-enhanced CT scan shows a large portion of pancreatic parenchyma that doesn't enhance, making the diagnosis of necrotizing pancreatitis. Only a small portion of the head (A) and tail (B) of the pancreas remain unaffected.

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**Fig. 4:** Contrast-enhanced CT scan shows enlargement of the pancreatic head and peripancreatic fat stranding (A and C) while the remaining pancreas appears normal (B). No areas of necrosis are seen. This is a case of focal interstitial edematous pancreatitis. A thrombus is also identifiable in the superior mesenteric vein (arrow in C).

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Fig. 9: Contrast-enhanced CT scan shows numerous fluid collections in the setting of acute pancreatitis, some of them containing necrotic debris. These are termed acute necrotic collections since they are identified during the first 4 weeks of disease. Of note is a ANC in a hepatic subcapsular location.

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**Fig. 7:** Contrast-enhanced CT scan (A and B) only shows enhancement of the tail of the pancreas. The remaining parenchyma is necrotic. In B, note the main biliary duct that courses without any viable pancreatic tissue surrounding it.

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**Fig. 8:** Contrast-enhanced CT scan (A and B) shows extensive necrosis of the pancreas, only a few areas of preserved parenchyma remain in the pancreatic head (arrow in B).

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Conclusion

The Revised Atlanta Classification established a common nomenclature regarding AP. Distinction are made between interstitial oedematous and necrotizing pancreatitis and among the various fluid collections seen. Familiarity with this classification and with its imaging counterparts is of greatest importance to convey accurate information to the referring clinicians and thus assist in the management of the patient.
References