Acute pancreatitis: a pictorial review

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Authors: P. Tarcu, M. D. COMSA; Cluj-Napoca/RO
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Learning objectives

To review the diagnostic and staging criteria for acute pancreatitis according to the 2012 revised Atlanta classification of acute pancreatitis in adults.

To exemplify through a series of cases the imaging timing and features of acute pancreatitis.
Background

Acute pancreatitis is defined as an acute inflammatory process of the pancreas with variable involvement of other local tissues and remote organ systems. It is one of the most common causes of hospital admission for gastrointestinal disorders.

It has a variety of causes (gallstones and alcohol in the vast majority) and can range in severity from mild to severe and life threatening. Despite the fact that mild acute pancreatitis (the most common form) has a very low mortality rate, organ failure presence and infected necrosis can bring this rate up to 20%-30% [1].

The revised Atlanta classification system is an international consensus which provides clear definitions when assessing acute pancreatitis type, phase, severity and complications, using easily identifiable clinical and radiological criteria [2].

Diagnosis

According to this classification, the diagnosis of acute pancreatitis requires at least two of the following three features [2]:

- abdominal pain consistent with acute pancreatitis;
- serum lipase and/or amylase activity at least 3 times greater than the upper limit of normal;
- characteristic findings on contrast-enhanced CT (CECT) or MRI, or less commonly on transabdominal US.

Course and severity of the disease

The onset of acute pancreatitis is considered to be the 1st day of pain.

Two distinct clinical phases of evolution, the early phase and the late phase, are reflected by the two peaks in mortality: one very early after onset (usually within the first week) and another after 2-6 weeks from onset [3].

The two phases are:

- the early phase (1st week) - severity is based on clinical/functional parameters, because treatment is determined primarily by the presence and duration of organ failure secondary to the host's systemic inflammatory response (SIRS)
During the early phase (1st week after the onset), the pathological process in and around the pancreas evolves from the initial state of inflammation and variable degrees of ischemia and/or edema to either resolution or to irreversible necrosis and liquefaction, and/or development of fluid collections in and around the pancreas. Systemic inflammatory response syndrome (SIRS) development can lead to organ failure, the presence and duration of which determines the severity and the need for treatment. Over the course of the 1st week, organ failure either resolves or becomes more severe. The modified Marshall scoring system is used to assess organ failure. There is not always a direct correlation between clinical severity with or without organ failure and extent of morphological characteristics in and around the pancreas [5, 6].

During the late phase (after the 1st week) the pathological process either resolves (interstitial edematous pancreatitis - IEP) or tends to stabilise or progress (necrotising pancreatitis). The progression is characterised by extension and/or infection of the necrosis and persistent multiorgan failure. Infected necrosis can lead to bacteraemia and sepsis. The main determinant of severity and the need for treatment continues to be persistent organ failure, but local complications may have direct implications in choosing the type of treatment. Thus, the late phase requires both clinical and morphological, image-based criteria [2].

This classification defines three degrees of severity [7, 8]:

- **mild** - absence of organ failure and of local or systemic complications
- **moderately severe** - presence of transient (<48h) organ failure and/or local or systemic complications
- **severe** - persistent organ failure (>48h)

**Imaging**

Imaging is required to confirm the diagnostic if suggestive abdominal pain is present but the amylase/lipase level is under the threshold value (e.g. in case of delayed presentation). Imaging is also useful early in the disease course when the cause of the disease is unclear, to look for causative factors such as choledocholithiasis and pancreatic cancer [4].

Imaging is fundamental in staging acute pancreatitis, assessing complications, guiding catheter placement for drainage, and monitoring treatment response through follow-up studies.
Because of the wide availability and high accuracy, contrast-enhanced CT plays the leading role in acute pancreatitis imaging, while MRI is usually reserved for a better characterisation of collections and for choledocholithiasis not seen in CT. MRI should also be used in patients in whom contrast-enhanced CT is contraindicated (e.g. due to pregnancy or iodinated intravenous contrast agents allergy). Transabdominal US can evaluate the presence of gallbladder stones [9, 10].

The ideal time for assessing local complications with CT is 72 hours after onset and the examination should be repeated whenever the clinical picture drastically changes.

Sometimes, in the early phase of the disease, the heterogeneous enhancement of the parenchyma can impede the differentiation between IEP and ill-defined necrosis. CECT performed 5-7 days later allows for a definitive characterisation.

The radiologist report should include the following: the presence/absence of pancreatic necrosis, characterisation of fluid collection in and around the pancreas, other findings such as ascites, gallstones, biliary dilatation, venous thrombosis (splenic, portal, mesenteric etc.), varices, pseudoaneurysms, pleural effusions and inflammatory involvement of the gastrointestinal tract, liver, spleen, kidneys or ureters [2].

There are two types of acute pancreatitis:

- interstitial edematous pancreatitis (IEP)
- necrotising pancreatitis

Four types of pancreatic and peripancreatic collections are described:

- acute peripancreatic fluid collection (APFC)
- pancreatic pseudocyst
- acute necrotic collection (ANC)
- walled-off necrosis
Findings and procedure details

Image findings

**Interstitial edematous pancreatitis (IEP)** - the most common form of acute pancreatitis

- diffuse pancreatic parenchyma enlargement (occasionally localised) due to inflammation
- homogeneous enhancement by intravenous contrast agent (i.e. no areas of pancreatic necrosis)
- mild peripancreatic fat stranding or haziness
- with or without peripancreatic fluid collections (with no solid component)

![Image](image.png)

**Fig. 1:** IEP in a 77-year-old man with gallstone-related pancreatitis. Axial contrast-enhanced CT image shows wispy peripancreatic inflammation (yellow arrows) with normal pancreatic enhancement and no collections.

**References:** Department of Radiology, Regional Institute of Gastroenterology and Hepatology, Cluj-Napoca/Romania 2016
Fig. 2: Same patient as in Fig. 1. Coronal image shows the choledocholithiasis (red arrow) and peripancreatic haziness (yellow arrow).

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Fig. 3: IEP in a 30-year-old woman. Axial contrast-enhanced CT image shows enlargement with normal pancreas enhancement (blue arrow) and minimal peripancreatic fluid collection (red star).

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Fig. 5: IEP in a 41-year-old man. Axial contrast-enhanced CT image shows normal enhancement of the pancreas with focal enlargement (blue arrow) and homogeneous fluid-attenuation collections adjacent to the pancreas, anterior to the left pararenal fascia and in the left paracolic gutter space (red stars), findings that are consistent with APFCs.

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**Necrotising pancreatitis**

There are three forms of necrotising pancreatitis, depending on location: pancreatic parenchymal necrosis alone, peripancreatic necrosis alone and pancreatic parenchymal necrosis with peripancreatic necrosis (in 75% of patients with acute necrotising pancreatitis) [11].
Pancreatic parenchymal necrosis is defined as a lack of enhancement by intravenous contrast agent (the extent should be reported as <30% or >30%)
Peripancreatic necrosis is defined as heterogeneous areas of enhancement that contain non-liquefied components, commonly located in the retroperitoneum or lesser sac.

**Fig. 6**: Necrotising pancreatitis in a 37-year-old woman. Coronal contrast-enhanced CT image shows an area of nonenhancement in the neck of the pancreas (red star), normal enhancement of the head and body (yellow stars) and peripancreatic heterogeneous collection (blue arrows), a finding consistent with ANC.

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Fig. 7: Necrotising pancreatitis in a 79-year-old woman. Axial CECT image shows multiple areas of abnormal pancreatic parenchymal enhancement (red stars) and a collection in the left anterior pararenal space (blue arrow).

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Fig. 8: Same patient as in Fig. 7. Multiple areas of abnormal pancreatic enhancement (red stars), collections distributed in the entire abdomen (blue arrows) and the presence of a gallstone (green arrow).

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Fig. 9: Same patient as in Fig. 7. Image acquired after 2 weeks shows the progression of the parenchymal necrosis (red star) and a heterogeneous collection containing both fluid and non-fluid densities (blue arrow), a finding that is consistent with ANC.

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**Fig. 10**: Same patient as in Fig. 7. Coronal image acquired after 2 weeks shows the progression of the parenchymal necrosis (red star) and heterogeneous collections containing both fluid and non-fluid densities (blue arrows), a finding that is consistent with ANCs.

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**Pancreatic and Peripancreatic Collections**

The morphological description of these local complications provides an accurate diagnosis. It includes location (pancreatic, peripancreatic, other), content nature (fluid, non-liquefied material, gas) and wall thickness, if any (thin or thick) [2].

The wall usually develops after 4 weeks, thus collections seen within the first 4 weeks after onset are categorised as acute collections.
All of these collections can be sterile or infected, but collections that contain non-liquefied material are much more likely to become infected. The distinction is important because treatment and prognosis are different [12].

IEP can be associated with acute peripancreatic fluid collections (APFCs) and pancreatic pseudocysts.

**APFC**

- peripancreatic homogeneous collection with fluid-attenuation density (without non-liquefied components) arising within the first 4 weeks after onset
- confined by normal peripancreatic fascial planes and without intrapancreatic extension
- without a definable encapsulating wall

*A fluid collection within the pancreatic parenchyma should not be labelled as APFC but as ANC.*
**Fig. 5**: IEP in a 41-year-old man. Axial contrast-enhanced CT image shows normal enhancement of the pancreas with focal enlargement (blue arrow) and homogeneous fluid-attenuation collections adjacent to the pancreas, anterior to the left pararenal fascia and in the left paracolic gutter space (red stars), findings that are consistent with APFCs.

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Fig. 11: IEP in a 26-year-old man. Axial contrast-enhanced CT image shows homogeneous pancreatic enhancement and several collections with fluid-attenuation densities (blue arrows), marks of APFCs.

References: Department of Radiology, Regional Institute of Gastroenterology and Hepatology, Cluj-Napoca/Romania 2016
**Fig. 12:** Same patient as in Fig. 11. Coronal contrast-enhanced CT image shows collections with fluid-attenuation densities (blue arrows) that are confined by the normal anatomical fascial planes. These findings are consistent with APFCs.

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**Pseudocyst**

- well-circumscribed collection seen >4 weeks after onset
- well-defined inflammatory wall
- homogeneous fluid density content
**Fig. 13**: Pseudocyst in a 50-year-old man with recurring pancreatitis. The fluid collection is homogeneous and presents a well-defined wall (blue star).

**References**: Department of Radiology, Regional Institute of Gastroenterology and Hepatology, Cluj-Napoca/Romania 2016
Fig. 14: Pseudocyst (blue star) in an 87-year-old woman with recurring pancreatitis. 

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Necrotising pancreatitis is associated with acute necrotic collections (ANCs) and walled-off necrosis (WON).

**ANC**

- heterogeneous collection containing both liquid and non-liquid attenuation densities (some appear homogeneous in the early phase) arising within the first 4 weeks after onset
- involving the pancreatic parenchyma and/or the peripancreatic tissues
- without a definable encapsulating wall
**Fig. 6:** Necrotising pancreatitis in a 37-year-old woman. Coronal contrast-enhanced CT image shows an area of nonenhancement in the neck of the pancreas (red star), normal enhancement of the head and body (yellow stars) and peripancreatic heterogeneous collection (blue arrows), a finding consistent with ANC.

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WON

- well-circumscribed collection seen >4 weeks after onset
- well-defined inflammatory wall
- heterogeneous liquid and non-liquid attenuation densities content
- involving the pancreatic parenchyma and/or the peripancreatic tissues
Fig. 16: WON in a 65-year-old woman with necrotising pancreatitis. Axial CECT image shows an encapsulated collection with mixed fluid and non-fluid attenuation densities (red star).

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Fig. 17: WON in an 80-year-old man with pancreatic parenchyma necrosis greater than 30% (enhancing remainder highlighted by blue arrow) and several heterogeneous collections containing non-liquefied debris (red stars).

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The presence of gas within a collection indicates the presence of infection.
Fig. 18: Infected WON in a 63-year-old man. Coronal image shows the infected WON replacing the pancreatic parenchyma (blue arrows).

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Fig. 19: Axial image of the same patient as in Fig. 18. Lung window highlights the gas bubbles (red arrows), markers of gas-producing bacteria.

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Fig. 20: Complications in a 64-year-old man with necrotising pancreatitis. Axial CECT image shows a peripancreatic and pancreatic heterogeneous collection with fluid and non-fluid (fat spots) attenuation densities (blue arrows) and an enhancing remainder of pancreatic parenchyma (yellow arrow).

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Fig. 21: Same patient as in Fig. 20, after 2 weeks. ANC infection (gas bubbles - red arrows) and percutaneous drainage catheters (green arrows).

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Splenic artery pseudoaneurysm is another possible complication (Fig. 22 on page 52).
Fig. 22: Same patient as in Fig. 20, after another 5 weeks. Coronal CECT image shows a ruptured splenic pseudoaneurysm (orange arrow).

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Conclusion

Acute pancreatitis is an evolving, dynamic condition and its severity may change during the course of the disease.

The revised Atlanta classification acts as a common language for radiologists, gastroenterologists, surgeons and pathologists.

Standardised radiological reporting is helpful for a better communication between clinicians, treatment planning and precise comparison of results among different departments and institutions.
References


