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Key Role of Radiology in Imaging and Managing Acute Pancreatitis and its Complications

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Abstract: ***Learning Objectives: 1. To diagnose a case of acute pancreatitis, identify and classify its complications by imaging studies based on revised Atlanta classification and differentiate it from other causes of acute abdomen. 2. To intervene in select cases of peripancreatic collections to drain them through interventional procedure. Background: Pancreatitis is an inflammatory process in which pancreatic enzymes auto digest the gland. Imaging modalities plays a primary role in management of acute pancreatitis. CT confirms the diagnosis and detects the severity of disease. Laboratory tests (serum amylase and lipase) are obtained to support the clinical impression, to help define the aetiology, and to look for complications. Recognizing the patients with severe acute pancreatitis and its complications is critical for achieving optimal outcomes. Imaging Findings: CECT abdomen and pelvis helped to differentiate between acute interstitial oedematous versus necrotizing pancreatitis. It also showed complications like acute pancreatic/ peripancreatic fluid collection, pseudocyst & walled off necrosis & differentiated between these collections based on revised Atlanta classification. Conclusion: Acute pancreatitis is associated with a wide variety of complications affecting the pancreatic gland, pancreatic duct, peripancreatic planes and surrounding vasculature. The role of the radiologist in imaging of acute pancreatitis and its complications is to select the most appropriate imaging techniques, timing of study and to diagnose local complications that will guide management decisions and reduce morbidity & mortality.***

Keywords: Acute pancreatitis (AP), Computed tomography (CT), Interstitial oedematous pancreatitis, Necrotizing Pancreatitis.

1. Definition

Acute pancreatitis (AP), an inflammatory disorder of the pancreas, refers to the autodigestion of the pancreas, in which pancreatic enzymes injure pancreatic tissue and lead to dysfunction of the gland, as well as remote organs and systems. The epidemiology of diseases often changes with time—for pancreatitis, this aspect is certainly true. The reasons for such changes are many: population growth and migration, change in patterns of alcohol consumption and tobacco smoking, rising rates of obesity and recognition of metabolic causes of pancreatitis, and increasing use and improving quality of imaging modalities [1–3].

Epidemiology Incidence

The global incidence of AP is 34 cases per 100,000 general population per year [95% confidence interval (CI) 23–49], with no statistically significant difference between men and women [4]. The disease affects people predominantly between 60 and 75 years old [5].

Clinical presentation

AP is an inflammatory condition of the pancreas that can cause local injury, systemic inflammatory response syndrome, and organ failure [11]. According to the revised Atlanta classification, accurate diagnosis of AP requires at least two of the following three diagnostic features [12]:

1) Abdominal pain consistent with AP.
2) Serum lipase or amylase levels that are at least 3 times the upper limit of the normal range, and
3) Findings of AP on cross-sectional imaging (computed tomography—CT)

If abdominal pain suggests strongly that AP is present, but the serum amylase and/or lipase activity is less than three times the upper limit of normal, as may be the case with delayed presentation, imaging will be required to confirm the diagnosis [13, 14]. If the diagnosis of AP is established by abdominal pain and increase in the serum pancreatic enzyme activities, a CT is not usually required for diagnosis in the emergency room or on admission to the hospital.

Phases of AP

AP is divided into early and late phases.

- The early phase—it is the first week after onset—which is characterized by activation of cytokine cascade with resultant systemic inflammatory response syndrome (SIRS). If SIRS persists there is an increased risk of developing organ failure, that can be—transient—if it resolves within 48 h or—persistent—if it persists for>48 h [16–18].
- The late phase, starting in the 2nd week and can lasts for weeks to months, occurs only in patients with moderately severe or severe pancreatitis, as defined by persistent organ failure and by local complications [12] and it is characterized by the presence of local complications, systemic manifestations and/or by transient or persistent organ failure.

Grading of AP

According to the revised Atlanta classification, the severity of AP identifies three classes:

- Mild AP, with no organ failure, and no local or systemic complications. Patients generally do not require pancreatic imaging, and mortality is very rare [19].
- Moderately severe AP is characterized by the presence of transient organ failure or local or systemic complications in the absence of persistent organ failure.
- Severe AP, characterized by persistent organ failure, that may be single or multiple organ failure; these patients can have one or more local complications, and have an increased risk of death [16–18].

To correlate complications and mortality clinical scoring system in 2004 Mortele et al, introduced the MCTSI, which includes as prognostic indicators the pancreatic inflammation, the pancreatic necrosis and extra pancreatic complications (Table 1) [20].

Prognostic Indicators	Points
Pancreatic inflammation	
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in pancreatic fat	2
Pancreatic or peripancreatic fluid collection or peri pancreatic fat necrosis	4
Pancreatic necrosis	
None	0
≤ 30%	2
≥ 30%	4
Extra pancreatic complications	
One or more of pleural effusion, ascites, vascular complications, or gastrointestinal tract involvement	2

Interstitial edematous pancreatitis (IEP) versus Necrotizing Pancreatitis (NP)

AP can be subdivided into two types, according to the pathologic changes: IEP and NP [12].

IEP is more common and represents non necrotizing inflammation of the pancreas. On CECT, the pancreatic parenchyma shows relatively homogeneous enhancement, but there are not unenhanced (necrotic) areas. The peripancreatic fat usually shows some inflammatory changes of haziness or mild stranding; there may also be some peripancreatic fluid [12] (Fig. 1); the clinical symptoms usually resolve within the 1st week. 5 -10%, of patients with acute pancreatitis develop a necrotizing pancreatitis (Fig. 2).

There are three subtypes of necrotizing pancreatitis, based on the anatomic area of necrotic involvement:

- Pancreatic only
- Peripancreatic only
- Combined pancreatic and peripancreatic [15].

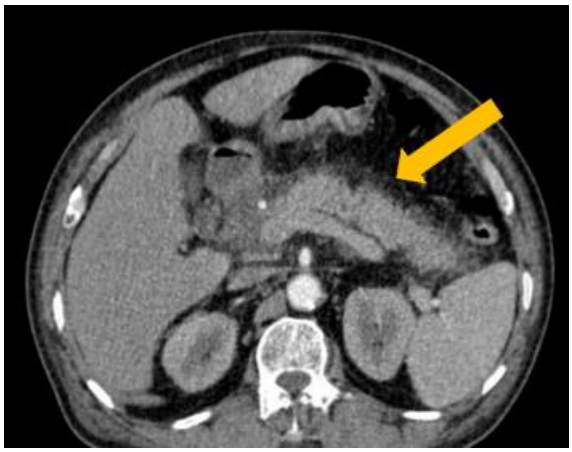


Figure 1: Acute interstitial pancreatitis in 17-year-old man, after alcohol abuse: axial IV contrast-enhanced CT scan shows mild diffuse enlargement of the whole pancreatic gland with poorly defined contours (arrow).

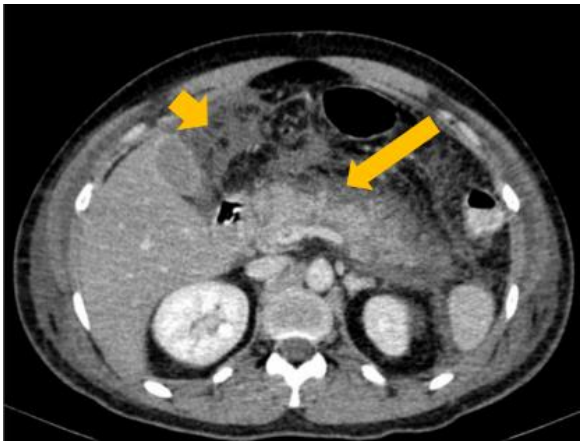


Figure 2: Necrotizing Pancreatitis in a 39 years-old man, with acute abdominal pain and sepsis.

Axial contrast-enhanced CT scans show enlarged pancreas with poorly defined contours and decreased enhancement of the pancreatic parenchyma (arrow), surrounded by heterogeneous fluid collection and acute necrotic collection (small arrow)

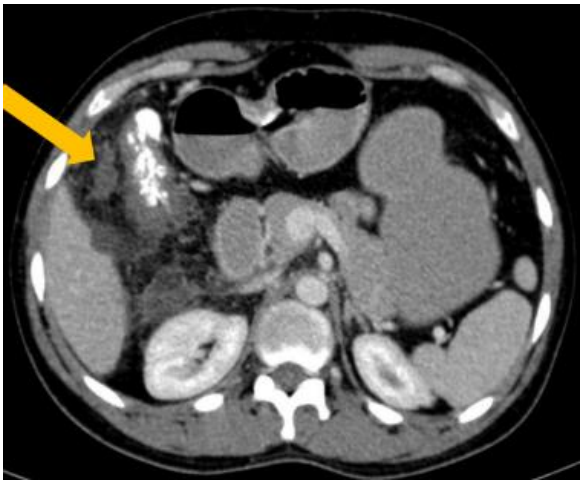


Figure 3: Extra pancreatic fat necrosis- Image shows extra pancreatic necrosis in a patient with severe organ failure at follow-up.

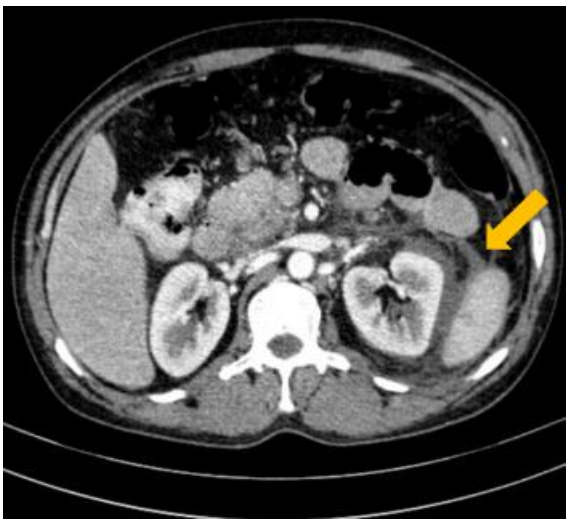


Figure 4: Acute peripancreatic fluid collection in a 59-year-old woman. Contrast-enhanced CT image performed 3 days after onset of acute attack, shows acute peripancreatic fluid collection.

Necrotizing pancreatitis manifests as necrosis involving both the pancreas and peripancreatic tissues in 75% of the cases (Fig. 3), while less commonly—in the 20%—it manifests as necrosis of only peripancreatic tissue, and in 5% as necrosis of the pancreatic parenchyma alone [12]. The combined subtype demonstrates non-enhancing pancreatic parenchyma, as well as non-enhancing heterogeneous peripancreatic collections, and typically accumulating in the lesser sac and anterior pararenal space. Peripancreatic necrosis alone occurs in 20% of cases, with normal enhancement of the pancreas, while in the peripancreatic tissues there is necrosis, with collections. Pancreatic necrosis alone is the least common subtype, occurring in 5% of cases [15]. An early CECT may underestimate the extent of pancreatic and peripancreatic necrosis because the impairment of pancreatic perfusion and signs of peripancreatic necrosis evolve over 7 days, therefore a CT examination should not be performed before 72 hrs, from the onset of symptoms, in order to grade the severity of the disease [12, 19, 21].

CECT is usually performed employing a protocol depending on the clinical question; at our institution the protocol includes an unenhanced scan, followed by arterial phase and portal venous phase. Iodinated intravenous contrast is essential to evaluate of pancreatic necrosis, as well as evaluate for vascular complications such as pseudoaneurysm or splenic thrombosis.

The revised Atlanta classification distinguishes different kinds of collections according to if they are purely fluid collections or containing necrotic debris in addition to fluid and considering the time course (≤4 weeks or >4 weeks from the onset of the pain). In patients with IEP in the first 4 weeks acute peripancreatic fluid collections (APFCs) can occur, that are fluid collections in the peripancreatic region, with no well-defined walls and no internal solid components. Most APFCs remain sterile and usually resolve spontaneously without intervention; on CECT they appear as homogeneous collections, with low attenuation, frequently seen in the lesser sac and in the anterior pararenal space (Fig.4). If an APFCs has not resolved after 4 weeks, it becomes more organized and develops a capsule that manifests as an enhancing wall at CECT, containing only fluid, no necrosis. At this point the collection refers to as a pseudocyst, a well-circumscribed peripancreatic fluid collection, surrounded by a well-defined enhancing capsule (fibrous or granulation tissue (Fig 5). They generally resolve spontaneously, while the 50% of persistent pseudocysts will cause clinical symptoms or complications, which can include secondary infection, pain, haemorrhage secondary to erosion into adjacent vessels, decompression or rupture, or local mass effect .

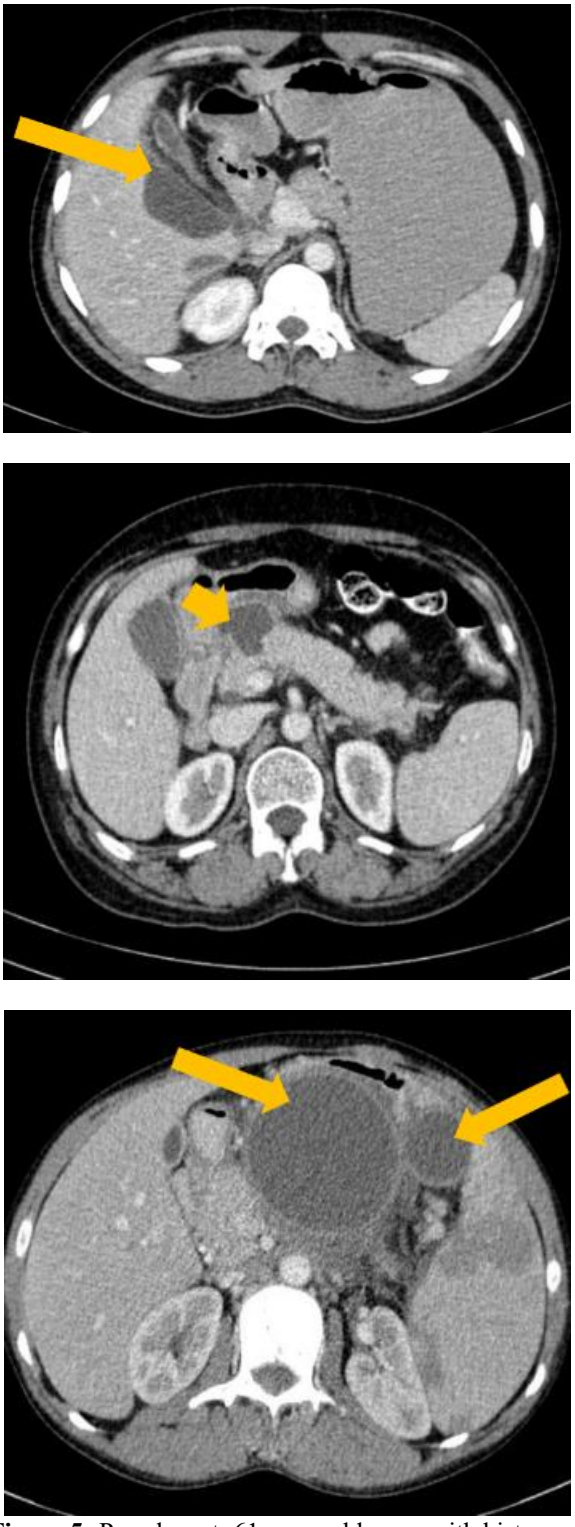


Figure 5: Pseudocyst: 61 years old man, with history of alcohol abuse, hospitalized for necrotizing pancreatitis. CECT scan after 4 weeks from the onset, shows a dissecting pseudocyst of liver (Long arrow). B) Other patients with evolving intrapancreatic pseudocyst (small arrow) and double pseudocyst (Arrow) (C)

Acute necrotic collection (ANCs) present within the first 4 weeks of necrotising pancreatitis and are poorly organized necrotic collections. On CECT, ANCs are heterogeneous in appearance and have no definable wall enclosing the collection (Fig.6); however, even if the collection is homogenous, it is considered ANC when associated with known pancreatic parenchymal necrosis. After 4 weeks of

necrotising pancreatitis ANCs become WON (walled of necrosis) (Fig.7). It resembles a pseudocyst, but it can be differentiated on CECT by the presence of internal solid components. (Table 2).

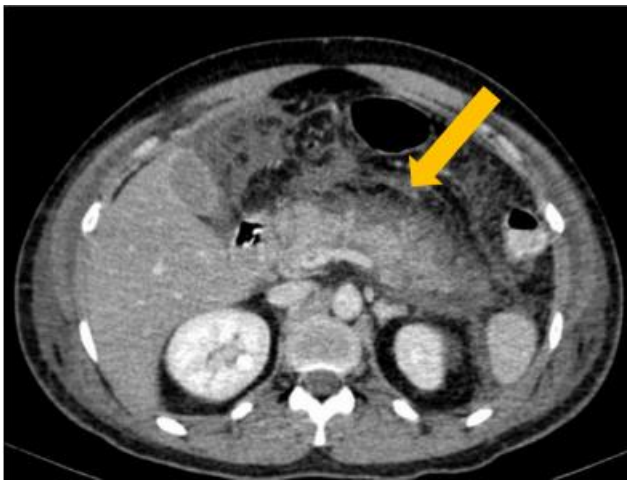


Figure 6: Acute necrotic collection in a 37-year-old man known with chronic alcohol abuse.

Contrast-enhanced CT image at the portal venous phase shows focal non-enhancing area of necrosis (arrow) in the peri pancreatic region.

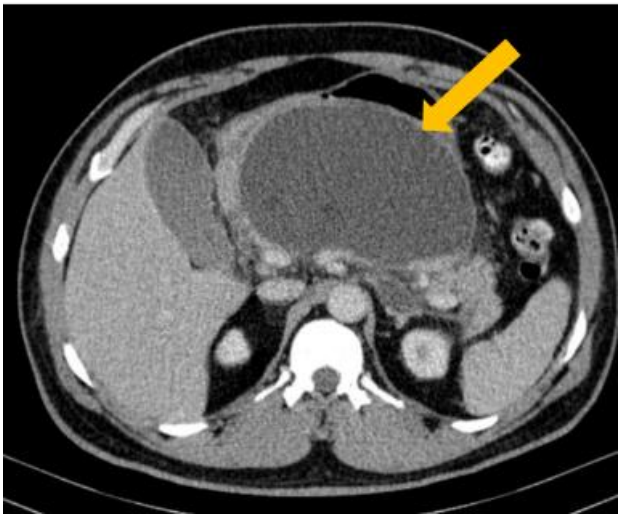


Figure 7: WON evolution. Acute necrotizing pancreatitis in a 61-yearsold man. Four weeks after the onset axial CECT scan shows the presence of an encapsulated collection (WON) in the peripancreatic fat near the body and the tail of the gland (arrow)

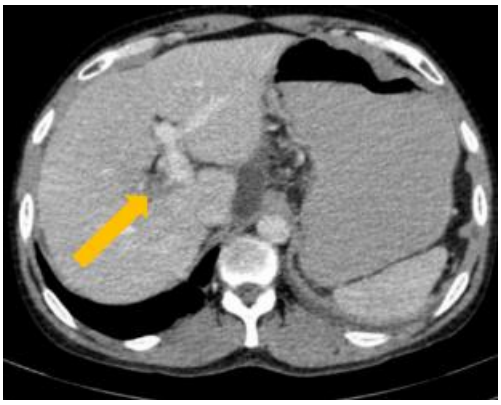
Table 2: Pancreatic and peri peripancreatic collections in necrotizing pancreatitis

Collection	Time after onset of pain (week)	Location	CECT features
ANC	≤ 4	Intra and / or extra pancreatic	Heterogeneous and non-liquid density of varying degrees in different locations No definable wall encapsulating the collection
WON	≥ 4	Intra and / or extra pancreatic	Encapsulated collection of pancreatic and/or peripancreatic necrosis Heterogeneous Well defined wall

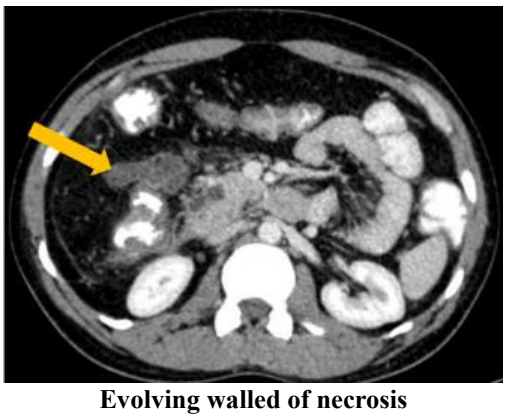
Complications

Any collection can be sterile or infected, although infection occurs more frequently in necrotic collections. Infection should be clinically suspected since the only imaging finding of an infected collection is the presence of gas within the collection. The gas often appears as multiple small bubbles scattered throughout the collection owing to the complex nature of necrotic collections [12].

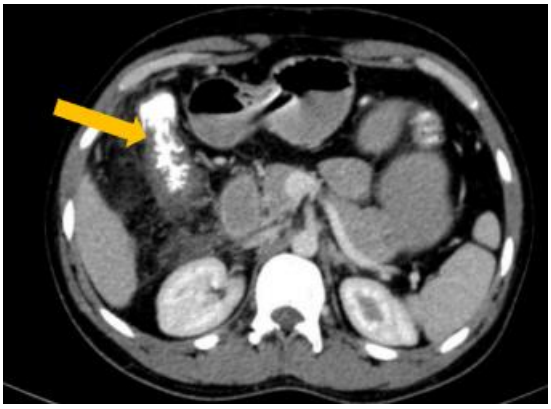
Apart from collections, other complications may occur, such as vascular complications. Splenic vein thrombosis represents the most common vascular complication in patients with AP. The release of pancreatic enzymes in AP results in erosion of local vasculature which may lead pseudoaneurysm malformation as well as spontaneous haemorrhage; the most common source of bleeding is the splenic artery, portal vein, and other peripancreatic vessels [17].



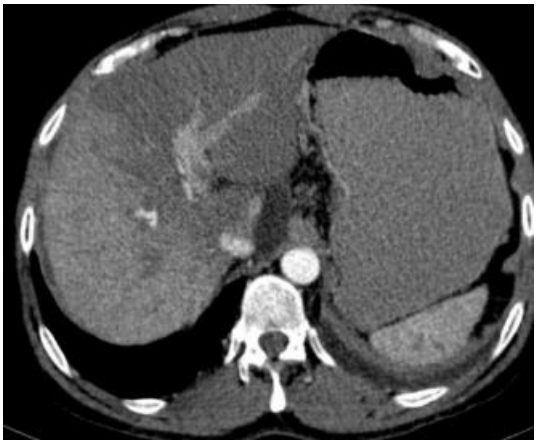
Occluded right branch of portal vein



Evolving walled of necrosis



Colonic wall thickening



Differential enhancement of liver

2. Conclusion

Acute pancreatitis is associated with a wide variety of complications affecting the pancreatic gland, pancreatic duct, peripancreatic planes and surrounding vasculature. The role of the radiologist in imaging of acute pancreatitis and its complications is to select the most appropriate imaging techniques, timing of study and diagnose local complications that will guide management decisions and reduce morbidity & mortality.

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