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SPECIAL SECTION: PANCREATITIS

CT imaging, classification, and complications of acute pancreatitis

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Abstract
Acute pancreatitis is an increasingly common condition and can result in significant morbidity and mortality. Contrast enhanced computed tomography (CECT) is the primary initial imaging modality in the characterization of acute pancreatitis. In this article, we provide sample CECT technical acquisition parameters for pancreatic imaging. We also review the classification systems for acute pancreatitis and give examples of common and uncommon complications of acute pancreatitis.

Keywords Pancreatitis · Complication · CECT · Guideline · Classification

Introduction to acute pancreatitis

Acute pancreatitis is an increasingly common condition with an incidence of 20–80 per 100,000, ranging widely by country. For example, while in the USA, the incidence of acute pancreatitis is estimated at 30–40 per 100,000, in Japan, the incidence was 49.4 per 100,000 in 2011 [1, 2]. Clinical presentation varies from transient abdominal discomfort to systemic inflammatory response syndrome and death may occur in up to 5% of cases [3, 4]. Patients with acute pancreatitis result in over 275,000 hospital admissions annually in the USA at a cost of over $2.6 billion (USD) in a study from 2009 [5].

Historically, approximately 80% of adult cases were considered secondary to alcohol use or obstructing gallstones, with other etiologies including drug reaction, pancreatic neoplasm, and hypertriglyceridemia comprising the majority of the remaining 20% of cases [3]. Newer data suggests that rates of idiopathic causes of acute pancreatitis are increasing and are now accounting for up to 20% of moderately severe to severe acute pancreatitis in the USA [2].

Acute pancreatitis is generally stratified into mild, moderately severe, and severe acute pancreatitis, further discussed below. Mild acute pancreatitis is self-limiting, with very low mortality and morbidity, and can often be diagnosed clinically/biochemically without imaging. Moderately severe acute pancreatitis, however, presents with transient (< 48 h) organ failure and/or local or systemic complications. Though moderately severe pancreatitis results in high morbidity compared to the mild version, its mortality is considered low at up to 2% [2]. Organ failure (frequently established using the modified Marshall scoring system) lasting greater than 48 h is classified as severe acute pancreatitis (Table 1). Mortality in the setting of severe acute pancreatitis is up to 50% [1, 3, 4, 6, 7].

Atlanta classification for acute pancreatitis

The Atlanta classification for acute pancreatitis was initially developed in 1992 and provided common terms for acute pancreatitis and related complications [1]. Advances in imaging and pathophysiology understanding necessitated a subsequent revision, the Revised Atlanta Classification (RAC) in 2012 [6]. Per the RAC, diagnosis of acute pancreatitis requires two of the following three features:

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Table 1 Modified Marshall criteria for organ failure

Organ system	Score				
	0	1	2	3	4
Respiratory (PaO2/FiO2)	> 400	301–400	201–300	101–200	≤ 101
Renal					
Serum creatinine (μmol/L)	≤ 134	134–169	170–310	311–439	> 439
Serum creatinine (mg/dL)	< 1.4	1.4–1.8	1.9–3.6	3.6–4.9	> 4.9
Cardiovascular (sBP, mmHg)[a]	> 90	< 90, fluid responsive	< 90, not fluid responsive	< 90, pH < 7.3	< 90, pH < 7.2

[a]Systolic blood pressure (sBP) measurements are determined in patients off ionotropic support

1. acute onset of abdominal pain that is persistent, severe, and epigastric often radiating to the back
2. serum lipase at least triple than of the upper limit of normal
3. characteristic findings of acute pancreatitis on imaging.

Types of acute pancreatitis

Interstitial edematous pancreatitis (Fig. 1)

The majority of the patients with acute pancreatitis will have diffuse or localized enlargement of the pancreas. On CECT, there is generally homogenous enhancement of the pancreas with mild stranding of the peripancreatic tissue. Clinical symptoms of interstitial edematous pancreatitis usually resolve within a week [6] (Fig. 1).

Necrotising pancreatitis (Fig. 2)

When necrosis develops in the setting of acute pancreatitis, which occurs in 5–10% of cases, this is deemed necrotising pancreatitis. Necrosis most commonly affects the pancreas and the peripancreatic tissue although one may occur without the other. The extent of necrosis becomes more clearly demarcated on CECT as an area of non-enhancing pancreatic parenchyma after one week of disease presence [6] (Figs. 2, 3).

Severity of acute pancreatitis

The RAC stratifies acute pancreatitis into mild, moderately severe, and severe disease (Table 2). The majority of patients will have mild pancreatitis which, by definition, does not present with organ failure or local complications. Symptoms of mild acute pancreatitis and its laboratory findings

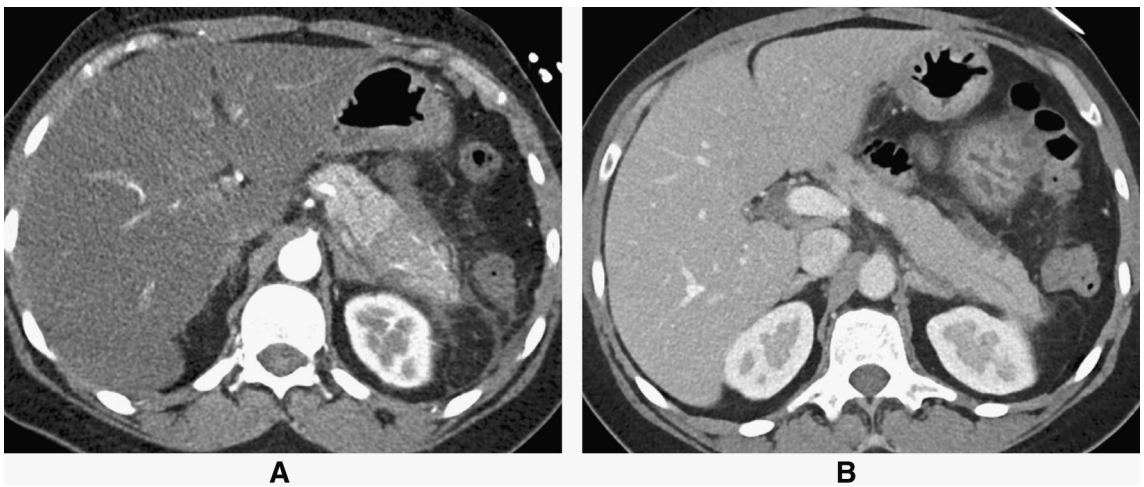

Fig. 1 Acute pancreatitis with early heterogeneous enhancement. 49-yo-F with chest pain and epigastric pain with 48 h duration. **a** Initial chest CT for PE protocol shows acute pancreatitis. Heterogeneous enhancement in tail of the pancreas raised possibility of pancreatic necrosis. **b** Follow-up contrast enhanced venous phase CT in 2 weeks shows improvement of heterogeneous enhancement, with no evidence of pancreatic necrosis; the heterogenous enhancement was secondary to interstitial edema

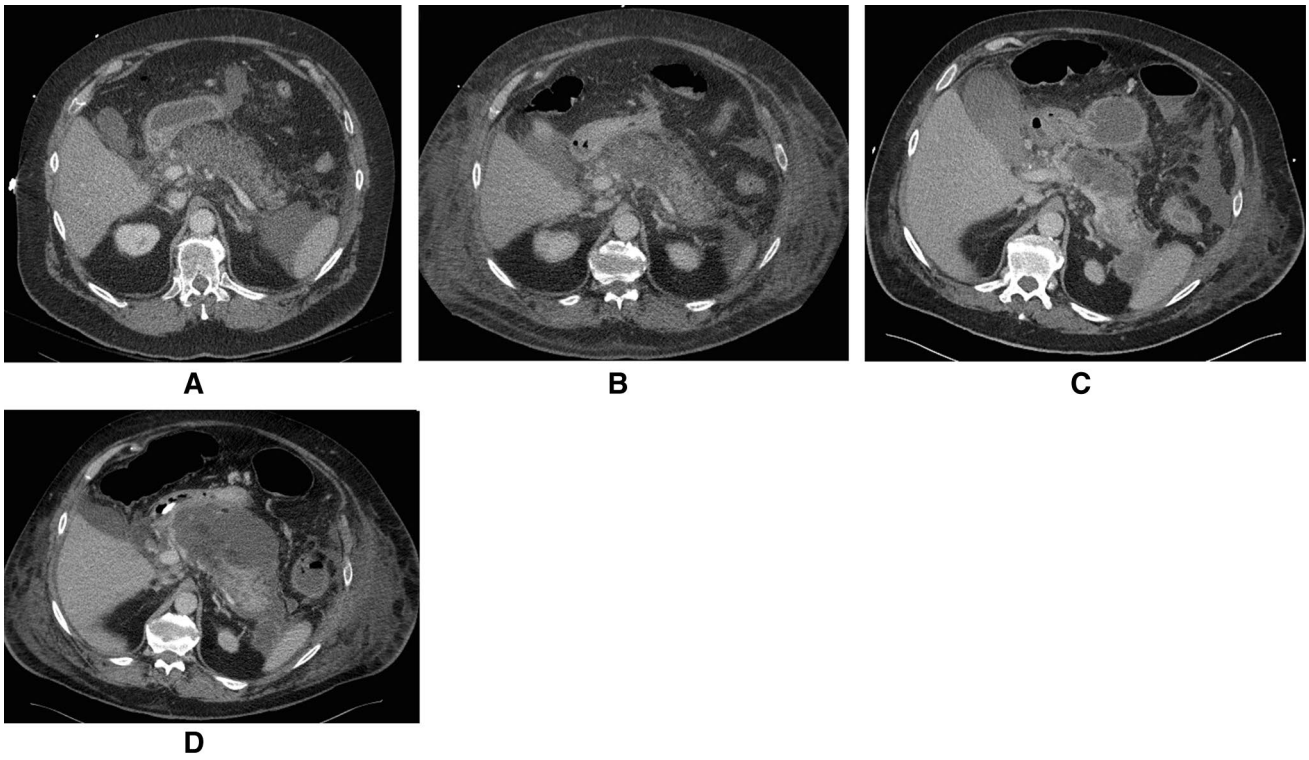

Fig. 2 Evolving necrotizing pancreatitis with acute necrotic collections developing into walled off necrosis. 75-yo-M with severe mid-epigastric pain for 72 h and markedly elevated lipase. CECT **a** shows diffuse poor enhancement and regional stranding. **b** 6 days later deteriorated, requiring vasopressor support. Heterogenous enhancement of < 50% of the pancreas consistent with necrotizing pancreatitis. **c** 21 days later, acute necrotic collections with internal locules of fat necrosis. **d** 35 days later, acute necrotic collections evolve into walled off necrosis with areas of rim enhancement and increased internal necrotic debris/fat

typically resolve with supportive care; however, 20% of patients will progress to more advanced disease [8].

Moderately severe acute pancreatitis is associated with transient organ failure lasting less than 48 h and/or local or systemic complications (which include exacerbation of patient comorbidities). RAC suggests that the cardiovascular, renal, and respiratory organ systems are assessed with the Modified Marshall scoring criteria to determine if failure is present (Table 1) [9]. Once organ failure resolves, these patients tend to have a similar disease course as the mild acute pancreatitis group [10]. Severe acute pancreatitis, however, is associated with increased mortality and may involve single or multiple organ failure. Additionally, those that develop infected necrosis have a higher mortality rate compared to those with sterile collections in the setting of acute pancreatitis [11].

Pancreatitis complications

Local complications

Local complications of acute pancreatitis should be suspected when there is persistent or recurrent abdominal pain, secondary increase in serum pancreatic enzymes, development of fever or leucocytosis, and increasing organ dysfunction. Local complications include acute peripancreatic fluids collections, pancreatic pseudocysts, acute necrotic collections (ANC), walled off necrosis (WON) (Fig. 2), and infection of pancreatic necrosis [6] (Fig. 4).

Acute peripancreatic fluid collections

Acute peripancreatic fluid collections (APFC) occur within the first 4 weeks of interstitial edematous pancreatitis (IEP) in the absence of peripancreatic necrosis and features of pseudocysts. On CECT, the criteria for APFC as per RAC are as follows: occurring in the setting of IEP, presence of a homogenous collection of fluid density, no distinct wall enclosing the collection, and no intra-pancreatic extension

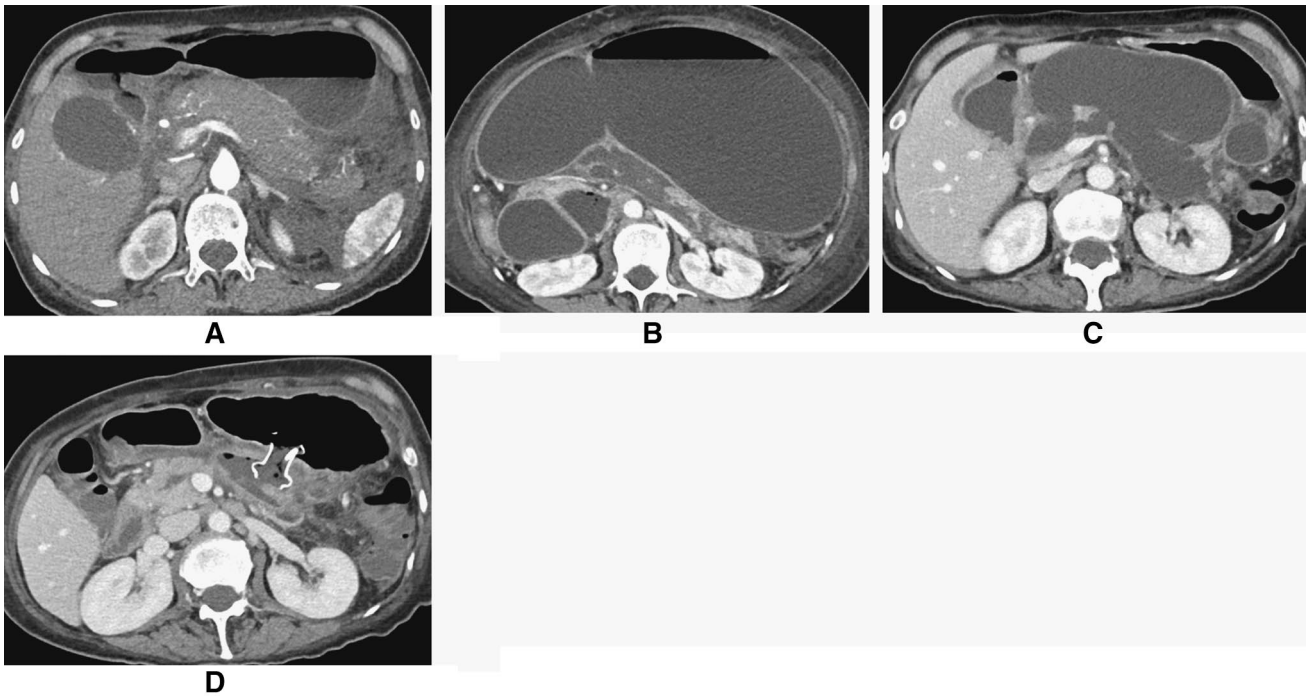

Fig. 3 Necrotizing pancreatitis with acute necrotic collection and secondary duodenal stenosis. **a** Initial CT in a 65-yo-F with severe upper mid abdominal pain radiating into her back which has been constant for about 12 h. **b** 9 days later after the patient started liquids and solid food intake, the patient developed worsening nausea, vomiting, and abdominal pain. The stomach and proximal duodenum are marked distended secondary to duodenal stenosis. **c** 20 days later, increasing size of acute necrotic collection with developing areas of rim enhancement. The stomach and duodenum are decompressed by nasogastric tube. **d** 26 days later, cystogastrostomy stent was placed, and acute necrotic collection was successfully drained and significantly decreased in size

Table 2 Revised Atlanta classification grading of acute pancreatitis

Grade of pancreatitis	Findings
Mild	No organ failure
	No local or systemic complications
Moderately severe	Organ failure that resolves within 48 h and/or local or systemic complications without persistent organ failure
Severe	Persistent (> 48 h) single or multiple organ failure

[6]. Most APFCs resolve spontaneously and can be followed by clinical monitoring and repeated imaging [12].

Pancreatic pseudocysts

If an APFC does not resolve within 4 weeks, it becomes more organized and develops an enhancing non-epithelialized wall on CECT, termed a pseudocyst [12]. Pseudocysts are well circumscribed, have no debris, and contain a homogeneous fluid density and may occur anywhere from the mediastinum to the pelvis, though most commonly are seen in the lesser sac [6, 13]. Approximately half of pseudocysts resolve spontaneously and also remain asymptomatic. Only 50% of persistent pseudocysts will cause clinical symptoms or complications, which can include secondary infection, pain, hemorrhage secondary to erosion into adjacent vessels, decompression or rupture, or local mass effect. Pseudocysts should be treated with endoscopic drainage or surgery if they are symptomatic, measure greater than 5 cm or increasing in size, and persist for more than 6 weeks [13].

Acute necrotic collection

ANCs present within the first 4 weeks of necrotising pancreatitis and are poorly organized necrotic collections. Necrosis can involve either the pancreatic parenchyma or may be peripancreatic tissue. On CECT, ANCs are

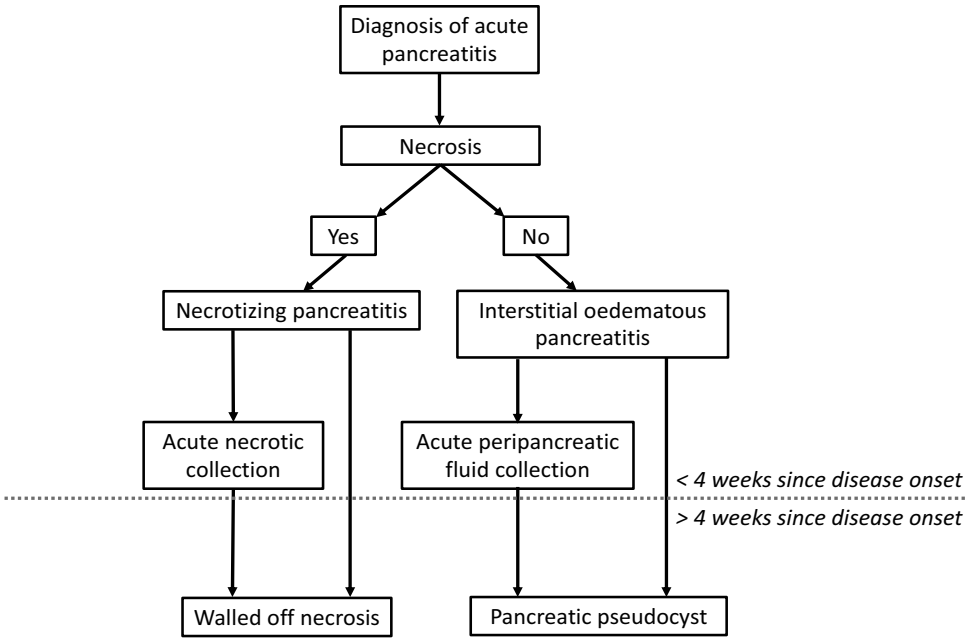

Fig. 4 Collections associated with acute pancreatitis

heterogeneous in appearance and have no definable wall enclosing the collection; however, even if the collection is homogenous, it is considered ANC when associated with known pancreatic parenchymal necrosis [6, 12].

Prior to 2 weeks, it may be difficult to distinguish ANCs from APFCs; however, ANCs will typically ultimately contain non-liquified debris or fat globules (Fig. 2). On unenhanced CT, the presence of fat attenuation within a pancreatic collection is helpful at identifying necrosis and can also help differentiate between ANCs and APFCs [6, 12].

Walled off necrosis

After 4 weeks of necrotising pancreatitis, similar to pseudocysts, ANCs become WON as a well-defined inflammatory wall develops. Consequently, like ANCs, WONs may be intra- or extra-pancreatic [6]. Although differentiating WON from pseudocysts may be simple when pancreatic necrosis is present, WONs may develop in the setting of a normally enhancing pancreas on CECT. In these cases, a T2-weighted MRI or ultrasound may be necessary to help identify the presence of debris in the fluid collection to distinguish WON from pseudocyst [3, 13].

A recent retrospective review has demonstrated that the majority of asymptomatic WONs resolve spontaneously but approximately a third of the patients will require intervention [14]. For those requiring treatment, minimally invasive approaches like percutaneous or endoscopic drainage have superior outcomes compared to open surgical debridement (Fig. 3). Since WON varies in extent, algorithmic management guidelines have been published on the suggested treatment approaches depending on the degree of necrosis and location; more accessible collections are treated with endoscopic transmural drainage, while deeper collections inaccessible to endoscopic drainage are managed with deep laproscopic necrosectomy and/or percutaneous drainage [15].

Infected pancreatic necrosis

Any of the aforementioned collections may be sterile or infected, though necrotic collections are much more likely to be infected. Infection is rare in the first week. The probability of infection of pancreatic necrosis increases with prolonged hospital stay and is usually secondary to Gram-negative enteric bacilli. Infection should be suspected in the setting of clinical sepsis. On CECT, air locules may be present within the necrotic pancreatic tissue [6, 13]. Debate regarding the value of fine need aspiration to confirm the presence of infection is ongoing, with some sites preferring early conservative management with percutaneous drainage allowing concurrent fluid culture over treatment based on clinical presentation alone [12].

Pancreatic duct complications

Necrosis of the central pancreas results in the disruption of the main pancreatic duct in 40% of cases. In the setting of residual functional pancreatic tissue upstream from the ductal disruption, persistent leakage of pancreatic fluid is seen. Peripancreatic ascites and the formation of pancreaticopleural fistula may also develop. If pancreatic duct injury is suspected, this can be confirmed with ERCP or pancreatic

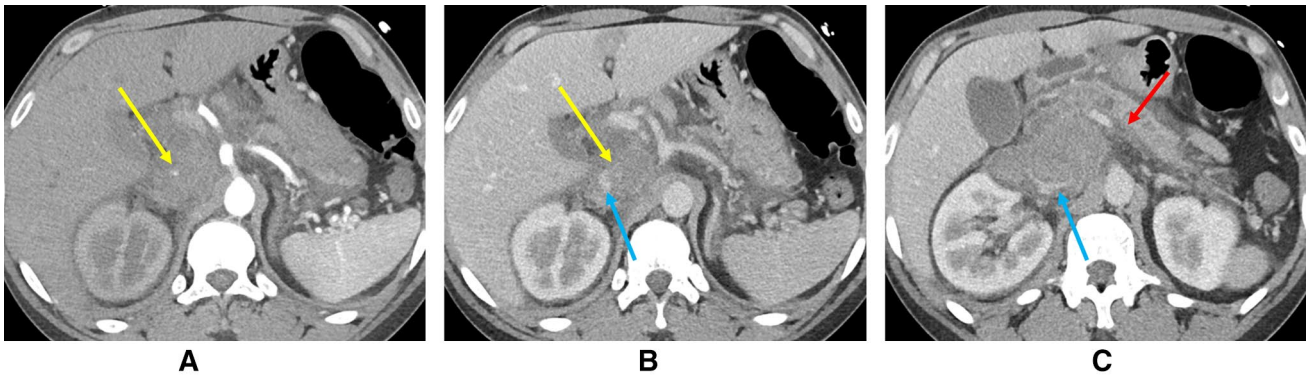

Fig. 5 Acute recurrent pancreatitis resulting in acute peripancreatic collection with spontaneous hemorrhage and splenic vein thrombosis. 35-yo-M with abdominal/back pain with history of prior pancreatitis. **a** Arterial phase CT shows acute peripancreatic fluid collection in portocaval space containing punctate amorphous enhancement (yellow arrow), which becomes more ill-defined in venous phase (**b**). More inferior image in venous phase **c** shows fluid collection in portocaval space contains radiodense content, compatible with hematoma. IVC (blue arrow) and portal vein/SMV confluence are compressed by fluid collection. Splenic vein is thrombosed (red arrow)

MRI and MRCP. Another late complication of necrotizing pancreatitis are pancreatic duct strictures, which may develop secondary to inflammation or healing following successful drainage of necrotic collection(s) [16].

Vascular complications

Vascular complications occur in about 25% of patients with acute pancreatitis. Local inflammation, reduced venous flow, and mass effect on venous structures from adjacent collections can lead to thrombosis. The splenic vein is the most common site for thrombus development with the superior mesenteric and portal veins being less commonly affected (Fig. 5).

The release of pancreatic enzymes in acute pancreatitis results in erosion of local vasculature which may lead to pseudoaneurysm formation as well as spontaneous hemorrhage (Fig. 5). The most common sources of bleeding are the splenic artery, portal vein, splenic vein, and other peripancreatic vessels [16].

Imaging timeline

Acute pancreatitis may be imaged with either contrast enhanced computed tomography (CECT) or magnetic resonance imaging (MRI) with or without contrast. Magnetic resonance cholangiopancreatography (MRCP) is a non-contrast sequence that can be added to routine MRI assessment of the pancreas, although MRCP in the early setting—prior to endoscopic retrograde cholangiopancreatography (ERCP)—has been shown to increase length of hospital stay, radiology cost, and hospital expenses with minimal benefit [17, 18]. We focus on the use of CECT in the setting of acute pancreatitis.

In the acute setting, within 72 h of symptom onset, CT imaging is not superior to clinical scoring systems because imaging may underestimate degree of pancreatic/peripancreatic necrosis or severity (Figs. 1, 2). Thus CT imaging during this early time period rarely changes management and does not improve clinical outcomes. Exceptions include equivocal biochemistry: alcohol-induced pancreatitis and hypertriglyceridemia have been shown to present with lower serum amylase and lipase.

In the initial few days, CT may show only equivocal findings, as both necrotic and edematous parenchyma exhibit heterogeneous enhancement on CECT [3, 17, 19, 20] (Fig. 1), and frank necrosis may take 24–48 h to develop [3]. CT obtained 3 days after clinical onset yield higher accuracy in the depiction of necrotizing pancreatitis and better differentiate normal variants or equivocal zones of ischemia from pancreatic necrosis [19]. Delay in clinical evaluation may similarly result in a serum amylase, or less commonly lipase, less than three times of upper limit of normal due to physiologic enzyme excretion or breakdown and can also require imaging to confirm the diagnosis [21]. CT-based scoring systems have been, and are being, developed to help guide the management of patients with acute pancreatitis; these will be discussed in detail below.

In the setting of moderately severe or severe acute pancreatitis, management is entirely guided by clinical scoring methods. Although pancreatic necrosis is a known risk factor for morbidity and mortality, the presence and/or extent of necrosis may not be reliably seen on imaging up to 7 days after initial symptoms, and the morphologic changes that may be present on imaging do not correlate with organ failure. In addition, the presence or absence of fluid collections or necrosis rarely, if ever, results in an urgent intervention within the first week [17].

If symptoms persist greater than 1 week, CECT or MRI to exclude necrosis is appropriate because these patients are suspected to have moderately severe or severe acute pancreatitis; mild pancreatitis usually resolves by this time. Subsequent imaging should be guided by the clinical presentation. New infectious signs/symptoms such as leukocytosis, fever and rigors/chills should prompt additional work up (CECT, MRI) to exclude infected collection or necrotizing pancreatitis complicated by infection, due to the significant increase in mortality in this subset of patients [2, 17].

In the setting of known peripancreatic collections over 4 weeks after symptom onset, imaging should be based on persistent or worsening clinical symptoms to exclude local complications such as secondary infections, or to plan for regional intervention. At this time, CECT or MRI may be considered [17].

CT technique

In the setting of acute pancreatitis, CECT remains the primary imaging modality to assess extent of the disease, identify complications, direct management, and assess disease evolution [16, 20]. Iodinated intravenous contrast is essential to evaluate local pancreatic morphology, the presence and extent of pancreatic necrosis, as well as evaluate for vascular complications such as pseudoaneurysm or splenic vein thrombosis.

Imaging protocols vary by institution, however, a typical CT protocol for evaluation of acute pancreatitis is a single-phase study in the pancreatic parenchymal phase (40 s after the initiation of IV contrast) [16, 22] or portal venous phase (60–80 s) [23, 24] from the top of the diaphragm and including the entire abdomen. A bolus intravenous injection of non-ionic 100–120 mL of iodinated contrast material (at a dose of 1.3–1.5 ml/kg) is performed by using a pressure injector at the rate of 3–5 ml/s [16, 23–26]. This may be followed by a saline chase of 20 ml normal saline at a rate of 2.5–3 ml/s [24, 25] to improve contrast enhancement and the efficiency of contrast medium utilization [26]. Images are typically reconstructed at 3-mm intervals in the axial planes. Thin (0.75–1.5 mm) slice reconstruction can be included to create 3D and multiplanar reconstruction (MPR) post-processed images. For enteric contrast, water may be used as negative enteric contrast.

In suspected arterial complications, such as a pseudoaneurysm or active bleeding, dual-phase imaging including both arterial and venous phase best evaluates for these entities [26]. Arterial phase can be performed at 25–30 s after the initiation of IV contrast or with the use of bolus triggering technique with attenuation monitored within the aorta. [24, 25]. However, for evaluating severity of pancreatic and extra-pancreatic changes, an initial dual-phase abdominal CT performed 72 h or more after onset of symptoms of acute pancreatitis has not been shown to be superior to single-phase CT [24].

Unenhanced images are typically not required. For patients who cannot undergo administration of iodinated contrast material, e.g., renal insufficiency or history of allergy to iodinated contrast material, unenhanced CT or MRI is another option [20]. Although unenhanced CT is suboptimal for evaluation of pancreatic necrosis and vasculature, the extent of pancreatic and peripancreatic inflammatory changes, pancreatic size, mesenteric edema, ascites, and wall thickening of gastrointestinal tract can still be assessed without IV contrast [27].

Dual-energy CT has been described in the setting of acute pancreatitis, though the research is currently limited. Early studies suggest that dual-energy techniques may allow better differentiation of necrotic debris, hematoma, or residual parenchyma with preserved enhancement [28, 29]. Detection of non-calcified gallstones using dual-energy CT with virtual monoenergetic image analysis as the cause of acute pancreatitis is another potential application [28–30]. Further dedicated research is required to determine the potential of dual-energy CT in evaluation of acute pancreatitis.

Alternative classification systems and the CT severity index

Prior to the widespread use of imaging to assess acute pancreatitis, several clinical scoring systems were used including the Ranson, Glasgow, and Acute Physiology and Chronic Health Evaluation (APACHE) II scores. Such scoring systems, though useful for predicting disease severity, cannot be relied upon to predict local complications, organ failure, or mortality. The Determinant Based Classification (DBC) and Revised Atlanta Criteria were developed to incorporate imaging and resolve the shortfalls of the clinical calculations [31].

The DBC was developed through an international multidisciplinary working group to address perceived limitations of the RAC. The DBC stratifies pancreatitis into four categories: mild, moderate, severe, and critical; the critical category was included beyond the RAC to increase accurate detection of patients at the highest risk of mortality [32]. Although both systems are able to predict outcomes of disease at similar rates, the RAC has been demonstrated to be superior in describing the clinical course of acute pancreatitis and it is also more accurate than Determinant Based Classification (DBS) [10, 31, 33–35]. Additionally, RAC has good inter-observer reliability with respect to classifying the type of acute pancreatitis and peripancreatic collections,

Table 3 CT severity index of acute pancreatitis developed by Balthazar et al. [7, 14]

Inflammation		Necrosis		Severity Index*
Grade and definition	Points	%	Additional points	
A: Normal pancreas	0	0	0	0
B: Enlarged pancreas (focal or diffuse)	1	0	0	1
C: Pancreatic inflammation and/or peripancreatic fat	2	< 30	2	4
D: Single peripancreatic fluid collection	3	30–50	4	7
E: Fluid collection > 1 and/or retroperitoneal air	4	> 50	6	10

*Severity Index is defined by points of inflammation plus points of necrosis (score 0–3: mild acute pancreatitis; 4–6: moderate acute pancreatitis; 7–10: severe acute pancreatitis)

Table 4 Correlation between CT severity index of acute pancreatitis and clinical outcome [7]

Severity Index	Morbidity (%)	Mortality (%)
0–1	0	0
2	4	0
7–10	92	17

supporting the widespread adaption of this classification system [36].

Concurrent to the development of the Atlanta criteria and the subsequent RAC, a number of CT scoring systems were developed in order to mirror the previously mentioned clinical scoring systems and give clinicians an accurate prognostic picture based on early imaging [22, 27, 7, 37]. Grading the severity of acute pancreatitis relies on two important aspects: the presence or absence of multi-organ failure, discussed above (Table 1), and the extent of pancreatic parenchymal injury, which can be quantified with CECT [27]. The most commonly used severity indices based on CECT findings are the CT severity index (CTSI) by Balthazar et al. [7] and the modified CTSI (mCTSI) by Mortele et al. [22].

The original CT severity index (CTSI) of acute pancreatitis, developed in 1990, assessed a combination of primary CT findings including peripancreatic inflammation and inflammatory collections and the extent of pancreatic necrosis: less than 30%, 30–50%, and more than 50% of the pancreas (Table 3) [7]. A significant direct correlation has been found between the CTSI and both the morbidity and mortality of acute pancreatitis (Table 4) [7].

In 2004, Mortele et al. [22] suggested a modified version of CT severity index (mCTSI) by simplifying the assessment of pancreatic inflammation and quantification of the amount of necrosis (< 30% or > 30% only) and adding assessment of extra-pancreatic complications including pleural effusion, ascites, vascular, parenchymal, or gastrointestinal tract complications to the conventional CTSI (Table 5).

Recently, comparative studies between the RAC, CTSI and mCTSI have been reported. In general, there is good agreement between RAC, CTSI, and mCTSI [23]; however, the mCTSI was found more sensitive but less specific than the CTSI in differentiating mild from more severe cases of acute pancreatitis [23]. In another series, however, the mCTSI was reported to be more accurate, easier to calculate, and with lower inter-observer variation than the CTSI in the management of patients with acute pancreatitis [38]. Another study considered two distinct clinical uses for the mCTSI and the CTSI, suggesting mCTSI for predicting short-term mortality and CTSI for predicting the need for intervention [39].

There are several shortcomings in the application of the CT scoring system for grading the severity of acute pancreatitis: the inability to detect or accurately quantify retroperitoneal fat necrosis on CT [19, 40], different performance in patients with initial and recurrent acute

Table 5 Modified CT severity index of acute pancreatitis developed by Mortele et al. [27]

Inflammation (points)	Necrosis (points)	Extra-pancreatic complications (one or more, 2 points)
Normal pancreas (0)	None (0)	Pleural effusion, ascites, vascular complications, parenchymal complications, gastrointestinal tract involvement
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat (2)	≤ 30% (2)	
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis (4)	> 30% (4)	

Modified severity Index is defined by points of inflammation plus points of necrosis and points of extra-pancreatic complications. (score 0–2: mild acute pancreatitis; 4–6: moderate acute pancreatitis; 8–10: severe acute pancreatitis)

pancreatitis [39], and—most importantly—the limited efficacy of CT in detecting pancreatic necrosis during early course of acute pancreatitis (< 48 h after the onset of symptoms) [3]. Despite the confirmed clinical utility of available grading systems of acute pancreatitis, a study carried out by Bollen et al. [27], with the findings which were subsequently confirmed in a separate study [41], concluded that because of similar predictive accuracies of CT scoring and clinical scoring systems, CT imaging to assess for severity of pancreatitis is not recommended on admission. Attempts to devise more accurate CT severity scoring systems using novel features for earlier assessment of patients with acute pancreatitis are ongoing [25, 42–44].

Summary

The availability and affordability of CT makes CECT the mainstay of imaging for patients with acute pancreatitis. Although the use of imaging in the first week of symptoms remains limited, imaging is critical to guide the future management of patients with moderately severe or severe acute pancreatitis. Clinical parameters must inform the need for—and timing of—pancreatic imaging in the setting of acute pancreatitis.

Radiologists, clinicians, and surgeons must be comfortable with the terminology and timelines set out by the Revised Atlanta Criteria when discussing patients with acute pancreatitis. Other currently available scoring systems based on CT findings are reproducible and correlate well with morbidity/mortality in severe cases; however, prognostic accuracy of imaging-based scoring systems is currently similar to clinical scoring systems. At this time, further research is required to improve the prognostic value of the aforementioned scoring rubrics beyond that of their clinical scoring counterparts.

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