

The Role of Imaging in Acute Pancreatitis

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ABSTRACT

Acute pancreatitis is an acute inflammatory disease of the pancreas that may also involve surrounding structures and remote organs. Imaging plays an important role as it confirms the diagnosis, identifies causative factors, helps to grade the extent and severity of disease, is crucial for the early detection of complications and helps to guide treatment. Ultrasound is the first-line imaging modality in our center for determining the cause of the disease (e.g., gallstones) and to rule out other causes of acute abdomen. Magnetic resonance cholangiopancreatography is reserved for detection of choledocholithiasis, not shown on other modalities, for further characterization of collections, to help diagnose disconnected pancreatic duct and for patients in whom contrast-enhanced CT is contraindicated. Contrast-enhanced CT is considered the gold standard in the evaluation of acute pancreatitis, as it evaluates the extent and evolution of the disease, stages its severity and detects complications. Imaging of acute pancreatitis requires familiarity with the appropriate radiologic nomenclature as defined by the revised Atlanta classification so that

imaging descriptions are standardized and communication with clinicians and surgeons is precise. The purpose of this review article is to present an overview of acute pancreatitis and the role of different imaging modalities, as well as to emphasize the importance of revised Atlanta classification and CT severity score.

INTRODUCTION

Acute pancreatitis (AP) is an acute inflammation of the pancreas resulting from an autodigestion of the gland. In approximately 80% of patients, AP is a self-limiting disease which subsides spontaneously, while 15–20% of patients develop severe form of disease, characterized by the development of pancreatic or peripancreatic necrosis, resulting in general and local complications responsible for a mortality rate of 8–35% (1, 2). The incidence of AP in Slovenia in 2001 was 40/100.000 with younger patients (between 20 and 40 years) being affected more commonly recently. Gallstones and alcohol are the most common causes of AP, and additional variants occur when patients are stratified by sex (2, 3).

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DIAGNOSIS AND THE ROLE OF IMAGING MODALITIES

According to revised Atlanta classification, two of the following three criteria must be met for the diagnosis of AP: 1) acute onset of persistent, severe epigastric pain, 2) lipase/amylase elevation > three times the upper limit of normal and 3) characteristic imaging features on imaging modalities (4). Imaging modalities available for the diagnosis of AP include US, contrast-enhanced CT (CE-CT), MRI and magnetic resonance cholangiopancreatography (MRCP). Although many patients will meet the criteria for AP by symptoms and laboratory results alone and may not require imaging initially, imaging may be performed early in the disease course when the cause of the disease is unclear, to look for causative factors such as cholelithiasis and pancreatic cancer. Imaging for the diagnosis of pancreatitis is also appropriate when abdominal pain suggests pancreatitis, but the amylase or lipase level is not elevated to the threshold value, which is often the case at delayed presentation. Also, imaging helps to grade the extent and severity of AP, is crucial for the early detection of complications and to help guide treatment (5, 6). The onset of pancreatitis is considered to coincide with the first day of pain, not the day on which the patient presents for care or the day of hospital admission (4).

Imaging of AP requires not only understanding of the disease subtypes and associated complications but also familiarity with the appropriate radiologic nomenclature as defined by the Atlanta symposium in 1992 and more recently, modified by the Acute Pancreatitis Classification Working Group in 2008 (7, 8). It is important for the radiologist to adopt

this new nomenclature so that imaging descriptions are standardized and communication with clinical and surgical colleagues is precise to properly manage the patient (8).

ULTRASOUND

Ultrasound is usually the first-line imaging modality in our center in suspected biliary pancreatitis and to rule out alternative diagnosis, because it is quick, easy to perform, readily available, avoids radiation

and can be carried out at bedside. The advantage of US in the early AP is to evaluate the gallbladder and biliary tract to detect gallstones (Figure 1) and the dilatation of the bile ducts. Changes in pancreatic parenchyma due to AP may only be seen in 30% of cases. In such cases, enlarged gland with obscured margins and hypoechoic parenchyma due to interstitial edema may be seen. Focal ill-defined hypo- or hyperechoic areas of edema or hemorrhage in the parenchyma and peripancreatic fluid collections may be seen. Also, US is used to characterize the contents of the



Figure 1. Ultrasound shows gallstones (arrow) in the lumen of the fully distended gallbladder with no signs of acute cholecystitis

fluid collections. One of the major limitations of US is the inability to make differential diagnosis of the interstitial and necrotizing pancreatitis (9, 10).

COMPUTED TOMOGRAPHY

CE-CT is considered the gold standard in the evaluation of AP. It plays a significant role in evaluating the extent of disease and evolution of the disease and its complications (11). In 2012, International Association of Pancreatology/American Pancreatic Association (IAP/APA) evidence-based guidelines provided recommendations concerning key aspects of medical and surgical management of AP. According to these guidelines, the indications for initial CT assessment

in patients with AP are 1) diagnostic uncertainty, 2) confirmation of severity based on clinical predictors of severe AP, or 3) failure to respond to conservative treatment or in the setting of clinical deterioration. Optimal timing for initial CT assessment is at least 72–96 hours after onset of symptoms (12). Routine early CT in AP is not recommended for the following reasons: 1) there is no evidence that early CT improves clinical outcome or that early detection of necrosis will influence treatment, 2) CT scoring systems are not superior to clinical scoring systems in predicting prognosis and severity of disease (13), 3) there is evidence to suggest that an early (inappropriate) CT may increase the duration of hospital stay (14), has low yield without direct management implications (15), does not improve clinical outcomes (16), and poses risks of contrast allergy and nephrotoxicity. Early CT (before 72 hours) may be useful to rule out bowel ischemia or intra-abdominal perforations in patients presenting with both AP and acute abdomen. Follow-up CT (or in some cases MRI) is indicated when there is a lack of clinical improvement, clinical deterioration, or especially when invasive intervention is considered (12).

It is recommended to perform multidetector CT with thin collimation and slice thickness (i.e., 5 mm or less), 100–150 mL of non-ionic intravenous contrast material at a rate of 3 mL/s, during the pancreatic and portal venous phase (i.e., 50–70 seconds delay). During follow-up, only a portal venous phase is sufficient (12). Adding the arterial phase to this protocol makes vascular complications, such as hemorrhage and pseudoaneurysms to be detected more clearly. Unenhanced CT helps in detecting calcified gallstones and pancreatic calcifications in chronic pancreatitis and adds to diagnosis of suspected intra-abdominal or pancreatic hemorrhage (17).

MAGNETIC RESONANCE IMAGING

According to the revised Atlanta classification, MRI imaging is reserved for detection of choledocholithiasis (Figure 2a) not visualized on CE-CT images (18, 19) and to further characterize collections for the

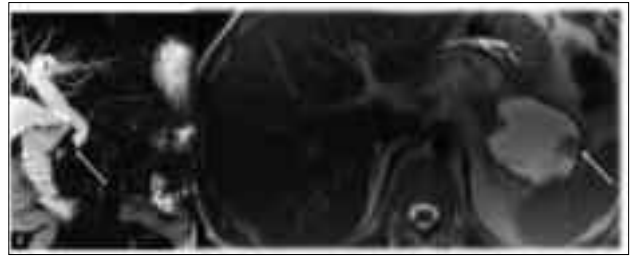


Figure 2. Magnetic resonance cholangiopancreatography. A – coronal plane showing gallstone (arrow) in the common biliary duct, just before papilla Vateri, B – T2 sequence in axial plane showing walled off necrosis with non-liquefied material (arrow) inside it

presence of non-liquefied material (debris and necrotic fatty tissue) (Figure 2b) (20). MRI has an important role in patients in whom CE-CT is contraindicated (e.g., due to allergy to iodinated intravenous contrast agents or pregnancy) (20, 21). It may also better show the disconnection of the pancreatic duct that creates persistent fistulation and inflammation with an increased incidence of infection (22).

REVISED ATLANTA CLASSIFICATION

In the revised Atlanta classification system, new definitions were created to stratify AP into two subcategories based on imaging findings: interstitial oedematous pancreatitis (IEP) and necrotizing pancreatitis. The imaging-based revised classification involves careful assessment of CT images for collections of fluid and non-liquefied material in and around the pancreas (i.e., areas of pancreatic parenchymal and peripancreatic necrosis). The terminology for fluid collections is completely revised. It is important for the radiologist to adopt this new nomenclature so that imaging descriptions are standardized and communication with clinical and surgical colleagues is precise. The classification also points out other important findings to be evaluated with imaging such as causes of pancreatitis, including cholecystolithiasis and choledocholithiasis, or complications related to AP (extrahepatic biliary dilatation; splenic, portal, and mesenteric venous thrombosis; varices; arterial pseudoaneurysm; pleural effusion; and ascites). Also, inflammatory changes due to pancreatic secretions in other intra-abdominal organs need to be reported.

Interstitial oedematous pancreatitis and necrotizing pancreatitis

IEP is more common and represents non-necrotizing inflammation of the pancreas. At CE-CT, the entire pancreas will enhance, with no unenhanced (necrotic) areas (Figure 3a). Necrotizing pancreatitis accounts for 5–10% of cases of AP (4). There are three subtypes of necrotizing pancreatitis; the subtypes are based on the anatomic area of necrotic involvement: 1) pancreatic only, 2) peripancreatic only, and 3) combined pancreatic and peripancreatic, which is the most common (75% of cases). The combined subtype

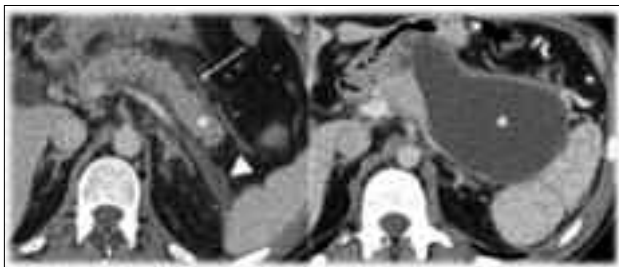


Figure 3. Interstitial oedematous pancreatitis. A – axial contrast-enhanced CT in venous phase, shows enlarged, heterogeneously enhanced gland (*), with indistinct margins and some stranding and minimal fluid (arrow) in the peripancreatic fat; acute peripancreatic fluid collection in the anterior pararenal space (arrowhead) is also present. B – five weeks later, the collection becomes more organised with capsule and is termed pseudocyst



Figure 4. Acute necrotizing pancreatitis – combined pancreatic and peripancreatic necrosis. A – axial CT image in venous phase shows pancreas that is almost completely non-enhancing (*), only a small part of processus uncinatus is enhancing; non-enhancing peripancreatic acute necrotic collections in the left and right pararenal spaces are also present (arrow). B – axial CT image obtained four weeks after onset, capsule is evident and some heterogeneity (*) is seen within this collection, reflecting the presence of non-liquefied material in walled-off necrosis. C – walled-off necrosis in left anterior pararenal space (*) and left paracolic space (arrow) are seen on axial CT images

demonstrates non-enhancing pancreatic parenchyma, as well as non-enhancing heterogeneous peripancreatic collections, and typically accumulating in the lesser sac and anterior pararenal space (Figure 4a) (23).

Pancreatic and peripancreatic collections

The revised Atlanta classification makes an important distinction between collections that contain purely fluid (those encountered in IEP) and collections that contain necrotic debris in addition to fluid (those encountered in necrotizing pancreatitis) and includes

Table 1. Revised Atlanta classification of fluid collections in acute pancreatitis. IEP – interstitial edematous pancreatitis, APFC – acute peripancreatic fluid collection, ANC – acute necrotic collection, WON – walled-off necrosis

Type of acute pancreatitis	Fluid collections
< 4 weeks after onset	
IEP	APFC
Necrotizing pancreatitis	ANC <ul style="list-style-type: none"> • parenchymal necrosis alone • peripancreatic necrosis alone • pancreatic and peripancreatic necrosis
≥ 4 weeks after onset	
IEP	pancreatic pseudocyst
Necrotizing pancreatitis	WON

new definitions that more accurately describe the various types of collections encountered: acute peripancreatic fluid collection (APFC), pseudocyst, acute necrotic collection (ANC), and walled-off necrosis (WON). The important distinctions for classifying collections correctly are the time course (< 4 weeks or ≥ four weeks from onset of pain and the presence or absence of necrosis at imaging (Table 1).

APFC occur during the first four weeks and are present only in patients with IEP because the pathogenesis involves inflammation without necrosis. They are visualized as homogeneous fluid-attenuation collections that lack a wall (Figure 3a). They are always peripancreatic in location (4). Most APFCs resolve spontaneously, and drainage should not be performed because of the risk of infecting an otherwise sterile collection (24). If an APFC has not resolved after four weeks, it is referred to as a pseudocyst. It becomes more organized and develops a capsule that manifests as an enhancing wall at CE-CT (Figure 3b). Pseudocysts may have a connection to the pancreatic ductal system, which is best seen at MRCP.

ANC are present within the first four weeks of symptom onset and are poorly organized necrotic collections that occur only in necrotizing pancreatitis (Figure 4a). They are often multiple, with a loculated appearance, and may extend inferiorly as far as the pelvic sidewalls. They typically demonstrate a variable amount of fluid and can be distinguished from APFCs by the presence of non-liquefied debris, such as solid-appearing components or fat globules within the fluid. In the early phase of pancreatitis, differentiating between an APFC and an ANC can be difficult, and the diagnosis of necrosis may be uncertain. Imaging in the second week is usually helpful for distinguishing an APFC from an ANC. After four weeks

the collection is referred to as WON, has a thick enhancing wall, contains fluid and necrotic fat and pancreatic tissue, which are well demonstrated at both CE-CT and MRI as non-liquefied debris within the fluid (Figure 4b, Figure 4c, and Figure 2b). WON may be confined to the pancreatic parenchyma but more commonly occurs in the peripancreatic space and can often occur in both locations, with a coalescent collection extending from the lesser sac into a portion of parenchyma (4). There is evidence that MRI outperforms CT with better assessment of the ratio of fluid to necrotic debris in collections older than four weeks. Therefore, MRI is a valuable alternative to CE-CT for planning interventions because it allows determination of the amount of necrotic debris that must be removed using more aggressive interventions (25).

Infection and local complications

Any collection can be sterile or infected, although infection occurs far more frequently in necrotic collections. The only imaging finding of an infected collection is the presence of gas within the collection (Figure 5) (4). Infected collections can also manifest with gas bubbles due to a pancreatic-enteric fistula, which can occasionally be seen when necrotic collections erode through the bowel wall, most commonly in the colon and duodenum (Figure 6) (26).



Figure 5. Infected necrosis. Axial un-enhanced CT shows gas bubbles (arrow) in the acute necrotic collection in bursa omentalis, suggesting secondary infection of the collection



Figure 6. Pancreaticoenteric fistula. Axial contrast-enhanced CT shows fistula (arrow) between walled-off necrosis in the mesentery and small bowel loop. Gas bubbles () due to secondary infection from fistula are seen in walled-off necrosis*

In addition to infection, vascular complications are common, occurring in a quarter of patients with AP, and can cause substantial morbidity and mortality. Inflammatory reactions can lead to splenic vein thrombosis, the most common vascular complication. Second, pancreatic enzymes can cause vessel erosion and lead to either spontaneous arterial hemorrhage or pseudoaneurysm of (in order of decreasing frequency) the splenic, gastroduodenal, and pancreaticoduodenal arteries (Figure 7) (27).

COMPUTED TOMOGRAPHY SEVERITY INDEX

A significant progress has been achieved in the evaluation of patients with AP with computed tomography severity index (CTSI) classification developed by Balthazar et al. in 1990. The classification was found to have an excellent correlation between necrosis, the length of hospitalization, development of complications and death (in patients with CTSI ≥ 7 , the morbidity rate was 92% with the mortality rate 17%) (11). In 2004, Mortelet et al. applied 'The Modified CTSI' which simplifies the evaluation of fluid collections and necrosis rate and adds the ex-

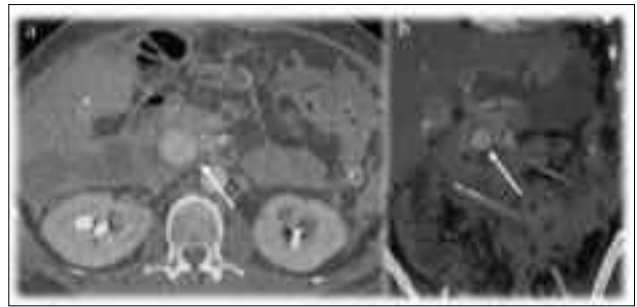


Figure 7. Pseudoaneurysm as a consequence of acute necrotizing pancreatitis. A – axial and B – coronal contrast-enhanced CT shows pseudoaneurysm that is feeding from gastroduodenal and superior mesenteric artery

trapaneatic complications (Table 2) (28). Compared to Acute Physiology and Chronic Health Evaluation (APACHE) II score, CTSI more accurately diagnoses clinically severe disease and better correlates with need for intervention and pancreatic infection (29).

CONCLUSION

Alongside with clinical and laboratory findings, imaging plays an important role in the management of AP. Imaging confirms the diagnosis, identifies etiology, helps to grade the extent and severity of disease and

Table 2. Modified computed tomography severity index for severity of acute pancreatitis. 0–2 – mild acute pancreatitis, 4–6 – moderate acute pancreatitis, 7–10 – severe acute pancreatitis

Prognostic indicator	Points
Pancreatic inflammation	
Normal pancreas	0
Enlargement of the pancreas	1
Peripancreatic inflammation	2
One acute peripancreatic fluid collection	3
Two or more acute peripancreatic fluid collection	4
Pancreatic necrosis	
None	0
30%	2
30–50%	4
>50%	6
Maximum 10 points	

helps detecting complications and to guide treatment. Ultrasound is used to rule out biliary pancreatitis and to exclude other pathologic conditions. Computed tomography is the modality of choice to stage the severity of disease, to detect the presence and extent of fluid collections and necrosis, to identify complications and to guide treatment. MRCP is especially useful for imaging patients with iodine allergies. It may assist in diagnosis of choledocholithiasis, characterizing collections and evaluation of an abnormal or disconnected pancreatic duct. The choice of the proper imaging modality depends on the clinical condition of the patient and the onset of epigastric pain in AP.

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