REVIEW / Gastrointestinal imaging

Imaging of acute pancreatitis and its complications. Part 1: Acute pancreatitis

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KEYWORDS
Acute pancreatitis; Ultrasound; Computed tomography; Magnetic resonance imaging

Abstract  Acute pancreatitis is an acute inflammatory disease of the pancreas that may also involve surrounding tissues or remote organs. The Atlanta classification of acute pancreatitis was introduced in 1992 and divides patients into mild and severe groups based on clinical and biochemical criteria. Recently, the terminology and classification scheme proposed at the initial Atlanta Symposium have been reviewed and a new consensus statement has been proposed by the Acute Pancreatitis Classification Working Group. Generally, imaging is recommended to confirm the clinical diagnosis, investigate the etiology, and grade the extent and severity of the acute pancreatitis. Ultrasound is the first-line imaging modality in most centers for the confirmation of the diagnosis of acute pancreatitis and the ruling out of other causes of acute abdomen, but it has limitations in the acute clinical setting. Computed tomography not only establishes the diagnosis of acute pancreatitis, but also enables to stage severity of the disease. Magnetic resonance imaging has earned an ever more important role in the diagnosis of acute pancreatitis. It is especially useful for imaging of patients with iodine allergies, characterizing collections and assessment of an abnormal or disconnected pancreatic duct. The purpose of this review article is to present an overview of the acute pancreatitis, clarify confusing terminology, underline the role of ultrasound, computed tomography and magnetic resonance imaging according to the proper clinical context and compare the advantages and limitations of each modality.

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Acute pancreatitis is an acute inflammatory disease of the pancreas characterized by autodigestion of the pancreatic parenchyma, interstitial fat necrosis and necrotising vasculitis, resulting from the inappropriate intracellular activation of proteolytic pancreatic enzymes. The inflammatory process may be limited to the pancreas, spread to surrounding tissues or even involve the remote organs, resulting in multiorgan failure and occasional death [1]. Imaging of acute pancreatitis requires not only an understanding of the disease subtypes and associated complications but also familiarity with the appropriate radiologic nomenclature as defined by the Atlanta symposium in 1992 [2] and, more recently, modified by the Acute Pancreatitis Classification Working Group in 2008 [3]. Correct use of the terms describing the radiological findings is crucial for management decision-making in patients with acute pancreatitis.

In patients with acute pancreatitis, imaging is recommended to confirm the clinical diagnosis, investigate the etiology, and grade the extent and severity of the disease. Ultrasound (US) is the first-line imaging modality in most centers for the confirmation of the diagnosis of the disease and the ruling out of other causes of acute abdomen, but it has limitations in the acute clinical setting. Contrast-enhanced computed tomography (CT) plays a significant role in evaluating the extent and evolution of the disease and its complications. Magnetic resonance imaging (MRI) has earned an even more important role in the diagnosis of acute pancreatitis. It is especially useful for imaging of patients with iodine allergies, characterizing collections and assessment of an abnormal or disconnected pancreatic duct.

The objective of this review article is to present an overview of the acute pancreatitis, clarify confusing terminology, underline the role of US, CT and MRI according to the proper clinical context and compare the advantages and limitations of each modality.

**Etiology and clinical presentation**

The two most common causes of acute pancreatitis are gallstones (30–45%) and alcohol abuse (30–35%) [4,5]. Less common causes include hypertriglyceridemia, hypercalcemia, viral infections (mumps, coxsackie), biliary parasites (ascaris), drugs (azathioprine, mercaptopurine, didanosine), oddi dysfunction, tumor, trauma, surgery, endoscopic retrograde cholangiopancreatography (ERCP) and congenital abnormalities (pancreas divisum, annular pancreas, choledochocele, duodenal duplication cyst). Acute pancreatitis is idiopathic in up to 20% of all cases, although about two-thirds of these cases are now thought to be caused by biliary sludge or microlithiasis [5,6].

The main presenting symptom of acute pancreatitis is abdominal pain, localized in the epigastrium in the majority of the cases, and radiated to the back in half of the cases. The clinical picture is often accompanied by nausea, vomiting, fever and tachycardia. The typical laboratory finding is the increase in the serum and/or urine levels of amylase and lipase [7]. However, elevated amylase and lipase levels are not specific to acute pancreatitis and may be caused by bowel obstruction, infarction, cholecystitis, or perforated ulcer [8]. As an indicator, increased serum lipase is accepted to be more sensitive and specific in the diagnosis of the disease than increased serum amylase [9]. The serum level of the alanine aminotransferase enzyme also increase in biliary acute pancreatitis [6]. Other laboratory findings include leukocytosis and elevated acute phase reactants, such as interleukin-6, C-reactive protein and procalcitonin [9]. For the clinical diagnosis of acute pancreatitis, at least two of the following three features must exist:

- presence of abdominal pain strongly suggesting acute pancreatitis;
- at least three-fold increase in the serum levels of amylase and/or lipase activity;
- presence of the characteristic imaging findings of acute pancreatitis [10].

**Natural course ans clinical severity scoring**

In the 1992 Atlanta Symposium, acute pancreatitis was divided into two groups as “mild” and “severe” based on the clinical and biochemical findings [2]. In 2008, this classification was revised by the “Acute Pancreatitis Classification Working Group” [2] developing a new morphological classification based on the imaging findings, and acute pancreatitis was divided into two groups as “interstitial edematous pancreatitis” and “necrotizing pancreatitis” [3].

The majority of the patients with acute pancreatitis (70–80%) are in the interstitial edematous (mild) group, which gives a rapid response to the conservative treatment and usually limits itself within 48 to 72 hours. In this group, organ failure and local complications are generally not observed and progression to the severe form is quite rare. It is histologically characterized by interstitial edema, infrequently, by pancreatic necrosis [1,2,5].

Necrotizing (severe) pancreatitis that is accounted for 20–30% of the patients with acute pancreatitis is histologically characterized by focal or diffuse pancreatic necrosis, fat necrosis, and hemorrhage in the pancreatic or peripancreatic tissues. The revised Atlanta classification introduces two phases of acute pancreatitis with a bimodal distribution for mortality [3]. The early phase occurs within the first week of onset of disease and is characterized by dynamically expanding pancreatic and peripancreatic inflammation with ischemia. These changes can either resolve or progress to irreversible necrosis and liquefaction, which may be associated with the development of fluid collections in and around the pancreas. Organ failure is the main determinant of the clinical course and disease outcome. Patients with organ failure that resolves within 48 hours of onset have been shown to have zero mortality rate. However, development of exaggerated inflammatory response (systemic inflammatory response syndrome) and subsequent multiorgan failure are responsible for approximately 50% of all deaths [1,2,5]. The late phase usually starts in the second or subsequent weeks. The course and outcome of this phase is mainly related to possible infection of the pancreatic necrosis. Pancreatic necrosis itself is not usually the cause of death in these patients, however, the necrotic tissue serves as a focus for infection in 40 to 70% of cases and mortality is related to infection. Disease progression is marked by increasing necrosis, infection, persisting systemic inflammatory response syndrome and multiorgan failure, causing a significant increase in mortality [1,2,5].

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A new subgroup of acute pancreatitis called “moderately severe acute pancreatitis” has recently been described as the group consisting of the patients who have local complications similar to those with severe acute pancreatitis but with lower morbidity, which is believed to be due to more transient organ dysfunctions [11,12].

There are several scoring systems, such as Ranson [11] and APACHE II (Acute Physiology and Chronic Health Evaluation) [13] to assess the severity of acute pancreatitis based on the clinical and laboratory findings, although each of them has some limitations. Calculation of the Ranson’s score cannot be completed before 48 hours after admission, and APACHE II score can be calculated after 24 hours. There are 11 Ranson criteria and 12 APACHE II criteria with 1 point for each parameter and the rate of the morbidity and mortality increases with the score. A Ranson score of 3 or more, or an APACHE II score of 8 or more suggests the presence of severe acute pancreatitis [11,13]. Ranson and APACHE II criteria are shown in Table 1. According to the 1992 Atlanta Symposium, at least, one of the following criteria must exist for the diagnosis of severe acute pancreatitis:

- organ failure (systolic blood pressure < 90 mmHg, PaO₂ ≤ 60 mmHg, serum creatinine > 2 mg/dL, gastrointestinal bleeding > 500 mL/day);
- presence of local complications (necrosis, infection, abscess, pseudocyst);
- Ranson score ≥ 3;
- APACHE II score ≥ 8 [2].

### Imaging methods

The purpose of imaging in acute pancreatitis is to confirm the clinical diagnosis, investigate the etiologic and evaluate the extent and complications of the disease. Imaging modalities available for the diagnosis of acute pancreatitis include US, CT, MRI, magnetic resonance cholangiopancreatography (MRCP) and ERCP. The modality to be selected depends on the reason for investigation. The main purpose of the imaging in acute clinical situations is to detect the disease, to recognize the complications and to make a differential diagnosis from the other acute abdomen causes (gastrointestinal perforation, acute cholecystitis, acute aortic dissection and mesenteric arterial occlusion, etc).

### Ultrasound

US is the first-line imaging modality in most centers for the confirmation of the diagnosis of acute pancreatitis and the ruling out of other causes of acute abdomen, because it is a quick and easy to perform examination and which is repeatable, free of radiation and can be carried out at the bedside. However, this modality has technical limitations related to paralytic ileus accompanying in the first 48 hours of the disease. The advantage of US in the early period is that it allows to evaluate the gallbladder and bilary tract, and to detect gallstones and dilatation of the bile ducts. Pancreas may be seen normal in the cases of mild acute pancreatitis. In 30% of the cases, pancreatic enlargement and decreased parenchymal echogenicity due to interstitial edema may be seen (Fig. 1). Focal ill defined hypo/hyperchoic areas (edema/hemorrhage), which may be observed in the parenchyma (Fig. 2). Blurring of the pancreatic contours due to the edema of the surrounding adipose tissue and the fluid collections in the peripancreatic region, especially in the lesser sac and the left anterior pararenal space may be seen. US is used in the characterization of the contents of the fluid collections and the

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**Table 1** Criteria of Ranson and APACHE II for severity of acute pancreatitis (severe pancreatitis: Ranson score ≥ 3 or APACHE II score ≥ 8).

<table>
<thead>
<tr>
<th>Ranson Criteria</th>
<th>APACHE II Criteria</th>
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<tbody>
<tr>
<td>At admission</td>
<td></td>
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<tr>
<td>Age &gt; 55 years</td>
<td>Age &gt; 55 years</td>
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<tr>
<td>WBC &gt; 16,000/mL</td>
<td>WBC &lt; 3000 or &gt; 14,900/mL</td>
</tr>
<tr>
<td>Glucose &gt; 200 mg/dL</td>
<td>Rectal temperature &lt; 36 °C or &gt; 38.4 °C</td>
</tr>
<tr>
<td>LDH &gt; 350 IU/mL</td>
<td>Mean arterial pressure &lt; 70 or &gt; 109 mmHg</td>
</tr>
<tr>
<td>AST &gt; 250 IU/L</td>
<td>Heart rate &lt; 70 or &gt; 109 bpm</td>
</tr>
<tr>
<td>During initial 48 hours</td>
<td></td>
</tr>
<tr>
<td>Hct decrease &gt; %10</td>
<td>Respiratory rate &lt; 12 or &gt; 24</td>
</tr>
<tr>
<td>BUN increase &gt; 5 mg/dL</td>
<td>pH &lt; 7.33 or &gt; 7.49</td>
</tr>
<tr>
<td>Calcium &lt; 8 mg/dL</td>
<td>Na⁺ &lt; 130 or &gt; 149 mmol</td>
</tr>
<tr>
<td>PO₂ &lt; 60 mmHg</td>
<td>K⁺ &lt; 3.5 or &gt; 5.4 mmol</td>
</tr>
<tr>
<td>Base deficit &gt; 4 mEq/L</td>
<td>PO₂ &lt; 70 or &gt; 200 mmHg</td>
</tr>
<tr>
<td>Fluid sequestration &gt; 6 L</td>
<td>Kreatinin &lt; 0.6 or &gt; 1.4 mg/100 mL</td>
</tr>
<tr>
<td></td>
<td>Htc &lt; %30 or &gt; %45.9</td>
</tr>
<tr>
<td></td>
<td>Glasgow coma score = 15</td>
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<td></td>
<td>Chronic health points</td>
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**Figure 1.** Interstitial edematous pancreatitis in a 50-year-old man known with chronic alcohol abuse. US image, obtained at admission, reveals enlargement and decreased parenchymal echogenicity of the whole pancreas (P) with poorly defined contours due to interstitial edema. Note also a small amount of peripancreatic fluid.
pseudocysts [14–16]. One of the limitations of US is the inability to make differential diagnosis of the interstitial and the necrotizing pancreatitis, because it does not allow the evaluation of the parenchymal perfusion. However, the studies performed with US contrast agents are promising [17–19].

Endoscopic US enables to identify common bile duct stones and occult pancreatic tumors that cannot be detected on CT or MRI in patients with recurrent acute pancreatitis. Fine-needle aspiration biopsy guided by endoscopic US may allow to differentiate focal pancreatitis from pancreatic tumors [20–22]. Color Doppler US enables to evaluate vascular complications, such as arterial pseudoaneurysm or thrombosis of the portal venous system [15,23].

**Computed tomography**

Contrast-enhanced CT examination that is considered as the gold standard in the evaluation of the patients with acute pancreatitis not only establishes the diagnosis of acute pancreatitis, but also allows to stage the severity of the disease [24,25]. In the cases with acute pancreatitis, CT examination should be performed if the clinical diagnosis is uncertain, clinical findings suggest severe acute pancreatitis (Ranson score ≥ 3, APACHE II score ≥ 8), or there is suspicion of necrotizing pancreatitis, and for patients who do not improve clinically within 72 hours of the initial conservative medical therapy or for patients who demonstrate improvement during the initial medical therapy but then manifest acute change in clinical status with fever, pain, decrease in hematocrit or hypotension, and any complication is suspected [26–28]. The ideal time for assessing these complications with CT is after 72 hours from onset of symptoms. Follow-up CT is not recommended for those patients whose initial CT scan showed Balthazar grade A–C pancreatitis or a CT severity index (CTSI) score < 3. However, if the patient shows change in clinical status that suggests a developing complication, CT scan should be performed. A follow-up CT is recommended at 7–10 days and/or before the discharge if the initial CT shows Balthazar grade D or E pancreatitis or CTSI score 3–10 at presentation [26–28]. CT is also useful to guide catheter placement for drainage and to assess success of treatment in patients who underwent percutaneous drainage or other interventions. Moreover, in patients with their first episode of acute pancreatitis who are over 40 years of age and have no identifiable cause for pancreatitis, contrast-enhanced CT should be used to exclude a possible neoplasm.

A thin-slice (≤ 3 mm) contrast-enhanced CT at the portal venous phase, obtained 60–70s after the intravenous contrast agent administration, allows to identify pancreatic necrosis as well as evaluating the extra-pancreatic complications [27–31]. Adding the arterial phase to this protocol makes vascular complications, such as hemorrhage and pseudoaneurysms to be revealed more clearly [15,32]. Unenhanced CT allows to define the calcified gallstones and the parenchymal calcifications in chronic pancreatitis more easily is usually unnecessary if there is no suspect of pancreatic or intraabdominal hemorrhage [32].

CT findings of acute pancreatitis depend on the severity and extend of the inflammatory process. A CT scan which is performed within the first 48 hours of the onset of symptoms may be completely normal. CT findings of acute pancreatitis include enlargement of the pancreas (localised or diffuse), ill defined parenchymal contours, decreased density and inhomogeneity of the pancreatic parenchyma and fluid collections in the peripancreatic region (Fig. 3). The inflammatory reaction can produce increased attenuation of the peripancreatic fat tissue commonly described as “stranding” [33–35] (Fig. 4). The inflammatory process is usually diffused and involves all the gland. However, the inflammation may be localized only in the head part in 18% of the patients [15]. Normal pancreatic parenchyma has CT attenuation values of 40–50 Hounsfield units (HU) on the unenhanced CT. A normal pancreas should demonstrate

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**Figure 2.** Interstitial edematous pancreatitis in a 59-year-old woman with gallstones. US image, obtained at admission, shows focal hypoechoic areas (arrows) in the pancreas (P) due to interstitial edema. There is a small amount of peripancreatic fluid.

**Figure 3.** Interstitial edematous pancreatitis in a 31-year-old woman with gallstones. Contrast-enhanced CT image, performed 3 days after onset of acute attack, reveals enlargement, diffusely decreased parenchymal density and loss of normal lobular contour of the pancreas (P) due to interstitial edema. Acute peripancreatic fluid collection and numerous gallstones (arrow) in the gallbladder are also seen.
a homogeneous increase in attenuation with intravenous contrast agent to 100–150 HU [25]. Parenchymal necrosis is seen as unenhanced or minimally enhanced (< 30 HU) hypodense areas on the contrast-enhanced CT [25–27] (Fig. 5). Contrast-enhanced CT scan performed 48–72 hours after the onset of acute attack, enables the definition of necrosis areas with a high accuracy (80–90%) [27]. Pancreatic enhancement often is decreased in fatty infiltration of the gland and pancreatic edema or interstitial pancreatitis. Sometimes it may be difficult to differentiate the focal necrosis areas from the intraparenchymal small focal fluid collections (Figs. 6 and 7). False-negative rate of the contrast-enhanced CT is 21% in the minor (under 30%) necrosis [27]. There is not a specific finding on CT out of the air bubbles for the infected pancreatic necrosis [26]. Because CT cannot reliably diagnose retroperitoneal fat necrosis, it has been suggested that all heterogeneous peripancreatic collections should be considered as areas of fat tissue necrosis unless proven otherwise [27] (Fig. 8). The inflammatory process in acute pancreatitis usually expands toward left of the pancreatic tail and the left pararenal space. A relative decrease in the density of the perirenal fat tissue due to an increase in the density of Gerota fascia and the pararenal

**Figure 4.** Interstitial edematous pancreatitis in a 59-year-old woman with gallstones. Contrast-enhanced CT image performed 3 days after onset of acute attack, shows extensive stranding of peripancreatic fat (arrows), acute peripancreatic fluid collection (F) extending to left anterior pararenal space and thickening of the left Gerota fascia (P: pancreas).

**Figure 5.** Pancreatic necrosis (> 75%) in a 68-year-old woman with gallstones. Contrast-enhanced CT images at the portal venous phase (a, b), obtained 3 days after the onset of acute attack, show full width necrosis (N) of the pancreatic neck, body and proximal tail. Parenchyma of the head and distal tail is seen to enhance normally. Acute peripancreatic fluid collection (F) extending to left anterior pararenal space, thickening of the left Gerota fascia, and a gallstone (arrow) in the gallbladder are also seen (P: pancreas).

**Figure 6.** Focal pancreatic necrosis (< 30%) in a 63-year-old man with gallstones. Contrast-enhanced CT images at the portal venous phase (a, b), obtained 3 days after the onset of acute attack, reveal focal non-enhancing areas of pancreatic necrosis (short white arrows) in the head of the pancreas (P). There are acute peripancreatic fluid collection (F) and thickening of the posterior parietal peritoneum (long white arrow) due to inflammation. Fluid in the gallbladder fossa and high attenuation content of the gallbladder (GB) due to prior ERCP examination are also seen.
space resulting from the inflammatory process leads to the ‘renal halo’ sign [36] (Fig. 8a). Ascites and reactive pleural or pericardial effusion may be seen even in the early stages of the acute pancreatitis.

The main advantages of CT against MRI include being much more available, less expensive, faster and used more easily in the patients with a poor general condition, having a higher sensitivity in definition of the small air bubbles and calcifications and being more practical to be used as a guide in the drainage applications [15]. The main disadvantages of CT are to include ionizing radiation and to require the use of iodinated contrast agents. Iodinated contrast agents have been found to aggravate acute pancreatitis, increase the complication rate and prolong the recovery time in animal models by impairing the pancreatic microcirculation, although this issue has not been confirmed in humans [37].

**Magnetic resonance imaging**

Today, the increase of the contrast resolution of pancreatic and peripancreatic tissues by the use of the fat-suppression techniques, breath-hold fast sequences and phased-array coils has increased the use of MRI in patients with acute pancreatitis. MRI of acute pancreatitis requires the combined use of T1-weighted sequence (e.g. fast spin echo imaging with multiple breath-hold acquisitions or single breath-hold gradient echo imaging), T2-weighted sequence (e.g. fast recovery fast spin echo or single shot fast spin echo imaging), and MRCP sequence (e.g. a thick slab, single shot fast spin echo T2-weighted sequence) [38–41]. Dynamic imaging after intravenous administration of gadolinium gives a comprehensive evaluation of the extent of the necrosis and the full range of the inflammatory extension. However, this protocol is difficult to be applied to acute pancreatitis patients with a poor general condition, since it requires 15–20 minutes. However, MRI should be preferred to CT in the cases for which exposure to ionizing radiation is important (children, young patients, pregnant women and patients with recurrent acute pancreatitis) and in the cases having iodinated contrast agent allergy or renal failure in which iodinated contrast agent administration is contraindicated. Because repetitive CT scans increase the dose of radiation patients with complicated acute pancreatitis are exposed to, using MRI in the follow-up of such cases is more appropriate. However, it should not be ignored that contrast-enhanced MRI scans performed by intravenous administration of gadolinium may create a risk for nephrogenic systemic fibrosis, especially in patients with renal failure [42].

The normal pancreas appears slightly hyperintense than the liver on T1-weighted images due to the acinar protein content, and isointense or slightly hypointense on T2-weighted images. While the pancreas is maximally enhanced at 20–40 seconds of intravenous gadolinium administration, it becomes isointense with the liver in the later phases [43]. In the mild pancreatitis, signal intensity of the pancreas...
may be normal. On T1-weighted with fat-suppression images, focal or diffuse pancreatic enlargement, decreased parenchyma intensity or parenchymal heterogeneity may be observed [39]. Sensitivity of T2-weighted with fat-suppressed sequence in the definition of the edema in the pancreatic parenchyma is higher [44,45]. In this sequence, pancreatic parenchyma is seen more hyperintense than the liver due to inflammation and edema [39–41] (Fig. 9). The necrotic pancreatic tissue is seen as hypointense on T1-weighted images and it does not show enhancement after intravenous gadolinium administration [39]. On T2-weighted images, necrosis may be hypointense or when liquefied, hyperintense. The most sensitive sequence in the demonstration of fluid collections is T2-weighted images [39–41]. Several studies have also demonstrated the superiority of MRI in the characterization of fluid collections, due to its ability to establish the presence of solid debris of necrosis [46]. Simple pseudocysts are uniloculated, encapsulated fluid collections that are seen as hypointense on T1-weighted and hyperintense on T2-weighted images (Fig. 10). Complex fluid collections are seen as heterogeneous lesion dominated by hyperintensity on fat-suppressed T1-weighted images [41]. T2-weighted images are useful to show the gallstones. Air bubbles are seen as hypointense both on T1-weighted and T2-weighted images [39]. Hemorrhage in the parenchymal or peripancreatic areas is shown best with T1-weighted gradient echo sequence [41]. Parenchymal hemorrhage is seen as the spotted or patchy areas of hyperintensity on T1-weighted images with fat-suppression. A hypointense hemosiderin rim is typically seen on T2-weighted images [39,41].

MRCP sequence shows small (>2 mm) bile duct stones, enabling to determine the etiology of acute pancreatitis [39–41] (Fig. 11). Congenital anomalies that may cause repeating acute pancreatitis, such as pancreas divisum and annular pancreas, are easily diagnosed with MRCP [38]. Another advantage of MRCP is that it allows the evaluation of the integrity of pancreatic duct. Whether pseudocysts are connected with the pancreatic duct or not can be demonstrated with MRCP [39–41].

MRI is at least as effective as CT in determining the presence and the extension of pancreatic necrosis and in showing the presence, localization and the extension of fluid collections, while this method is superior to CT in the evaluation of the content of fluid collections [38]. However, MRG has a lower success rate in definition of the air bubbles and calcifications.

**CT Severity index**

A significant progress has been achieved in the evaluation of the patients with acute pancreatitis with CTSI classification described by Balthazar et al. in 1990 (Table 2), which divides the severity of acute pancreatitis into five groups (Grades

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**Figure 9.** Interstitial edematous pancreatitis in a 38-year-old woman with gallstones. Axial T2-weighted MR images without fat suppression (a, b), obtained at admission, show increased signal intensity in the head, isthmus and proximal body of the pancreas (P) due to interstitial edema. A small amount of peripancreatic fluid and thickening of the left Gerota fascia are also seen.

**Figure 10.** Pancreatic pseudocyst (Ps) in a 41-year-old man known with gallstones. Axial CT (a), T1-weighted (b) and T2-weighted (c) MR images, obtained 6 weeks after the onset of acute attack, reveals a well defined, homogenous fluid collection (Ps) with a fibrous capsule (P: pancreas).
A—E [24,25] (Fig. 12). Balthazar CTSI allows staging of the severity of the inflammatory process, evaluation of the pancreatic necrosis and definition of the local complications, enabling differentiation of the mild (interstitial/edematous) acute pancreatitis from severe (necrotizing) acute pancreatitis and making decision for the correct treatment.

Table 2 Balthazar CT severity index for severity of acute pancreatitis (mild acute pancreatitis = 0–3, moderate acute pancreatitis = 4–6, severe acute pancreatitis = 7–10).

<table>
<thead>
<tr>
<th>Prognostic indicator</th>
<th>Score</th>
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<tbody>
<tr>
<td><strong>Pancreatic inflammation</strong></td>
<td></td>
</tr>
<tr>
<td>Grade A: normal pancreas</td>
<td>0</td>
</tr>
<tr>
<td>Grade B: focal or diffuse enlargement of the pancreas</td>
<td>1</td>
</tr>
<tr>
<td>Grade C: pancreatic ± peripancreatic fat inflammation</td>
<td>2</td>
</tr>
<tr>
<td>Grade D: single peripancreatic fluid collection</td>
<td>3</td>
</tr>
<tr>
<td>Grade E: two or more peripancreatic fluid collection ± retroperitoneal air</td>
<td>4</td>
</tr>
<tr>
<td><strong>Pancreatic necrosis</strong></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>&lt;30 %</td>
<td>2</td>
</tr>
<tr>
<td>30–50 %</td>
<td>4</td>
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<tr>
<td>&gt;50 %</td>
<td>6</td>
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<tr>
<td><strong>CT Severity Index</strong></td>
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Balthazar et al. found an excellent correlation between necrosis, the length of hospitalization, development of complications, and death: patients with a CTSI ≤ 3 showed a morbidity rate of 8% and mortality rate of 3%. However, in patients with CTSI ≥ 7, the morbidity rate was 92% with a 17% mortality rate [24,25].

“The Modified CTSI” described by Mortele et al. in 2004 simplifies the evaluation of fluid collections and necrosis.

Figure 11. A 38-year-old woman with biliary acute pancreatitis. MRCP sequence image reveals multiple signal-void stones surrounded by high signal intensity bile in common bile duct (arrow). A large multiloculated fluid collection (F) is also seen.

Figure 12. Balthazar CT scoring system. a: grade A: normal pancreas; b: grade B: pancreatic enlargement; c: grade C: pancreatic and peripancreatic fat inflammation (long arrow) [small superficial necrotic areas (small arrows) are also seen]; d: grade D: single peripancreatic fluid collection (F); e: grade E: two fluid collections (F).
rate and adds the extra-pancreatic complications (pleural effusion, ascites, vascular complications, parenchymal complications and gastrointestinal system involvement) (Table 3) [47]. There is no significant difference between the CTSI and modified CTSI in evaluating the severity of acute pancreatitis [48]. Compared to APACHE II, both CT indexes more accurately diagnose clinically severe disease and better correlate with needed for intervention and pancreatic infection [48]. Another scoring system used to evaluate the severity of acute pancreatitis is extra-pancreatic inflammation on computed tomography (EPIC) scoring system (Table 4) [49]. The definition of the risk can be carried out with EPIC scoring system within the 24 hours of admission without the need for contrast agent to be administered.

### Conclusion

The comprehensive evaluation of acute pancreatitis is based on clinical, laboratory and imaging findings. CT is an excellent non-invasive modality of choice to stage the severity of the inflammatory processes, to detect the presence and extent of the pancreatic necrosis and to identify local complications. MRI has earned an even more important role in the diagnosis of acute pancreatitis. MRI is at least as effective as CT in the detection of the presence and extent of pancreatic necrosis and in showing the presence, localization and extension of fluid collections, while this method is superior to CT in the evaluation of the content of fluid collections. The choice of the proper imaging modality in acute pancreatitis exactly depends on available time, technology, and clinical condition of the patient.

### Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

### References
