Imaging acute pancreatitis – the role of computed tomography

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The incidence of acute pancreatitis is increasing in Europe, with significant medical, surgical and financial implications. The overall incidence of acute pancreatitis is 22.4 per 100,000 population in England with a hospital admission rate of 9.6 per year per 100,000 population in the United Kingdom (1;2). In more than 80% of patients, acute pancreatitis is secondary to gallstone disease or alcohol abuse. Recognising the importance of this condition, the UK Working Party on Acute Pancreatitis has issued guidelines for the management of acute pancreatitis that provide an overview of the clinical perspective and management.

Acute pancreatitis is an acute inflammatory disease of the pancreas caused by inappropriate intracellular activation of proteolytic enzymes and subsequent autodigestion of the pancreatic parenchyma and surrounding tissues. The majority of patients have mild interstitial oedematous pancreatitis (IOP) which is self-limiting and responds rapidly to conservative management. However, 20% have severe acute pancreatitis (SAP) which can progress to a systemic inflammatory response syndrome (SIRS) and result in septic systemic complications with significant morbidity and mortality. This sub-group requires immediate medical care to prevent life-threatening complications.

Imaging, most commonly with contrast-enhanced computed tomography (CE-CT), plays a significant role in the identification of local and systemic complications and in planning further management. Image-guided interventional procedures tend to be less invasive than surgery, often reducing the need for surgical intervention, and thereby improving outcome.

Severity scoring

There are several clinical and biochemical scoring systems that can be employed to assess the severity of acute pancreatitis. In 1985, Balthazar et al introduced a scoring system based on radiological findings by grading the severity of pancreatitis into five different groups on unenhanced CT (table 1, figures 1a-e). To improve the prognostic value of this system, CE-CT findings of the degree of necrosis were incorporated to give the CT severity index (CTSI) (table 1). The system was further modified by Mortelé et al in 2004, combining pancreatic inflammation, necrosis and extra-pancreatic complications in a 10-point scale to give the modified CTSI (table 2) which shows a stronger correlation between outcome and severity than the original.

Contrast-enhanced computed tomography

CE-CT is considered to be the gold standard imaging modality in the evaluation of patients with acute pancreatitis. MRI and ultrasound (US) are used in specific clinical situations and can be useful in determining aetiology.

Ideally, CE-CT should be performed 48-72 hours after

FIGURE 1
Balthazar Scoring System.

(a) Balthazar grade A – The pancreas appears normal on CT. Note is made of a subtle secondary sign of inflammation with thickening of the left Gerota’s fascia (white arrow). (b) Balthazar grade B – The pancreatic gland is swollen on CT. Note the presence of intrahepatic biliary dilatation (black arrowheads) which was also secondary to gallstone disease, the cause of pancreatitis in this patient. (c) Balthazar grade C – Pancreatic oedema with standing in the peri-pancreatic tissues (white arrows). Note the thickening of Gerota’s fascia on the left and the presence of part-calcified gallstones in the gall bladder (black arrows). (d) Balthazar grade D – Single peripancreatic collection (*). (e) Balthazar grade E – Two peripancreatic fluid collections (*) and extensive peripancreatic fat stranding.
A higher accuracy in the depiction of necrotising pancreatitis is characterised by enlargement of the pancreas (which can be localised or diffuse) and homogeneous enhancement of other organs. Imaging at 60 seconds optimises vascular opacification and contrast administration (enhancement of less than 30 HU correlates well with necrosis).

**TABLE 1**
Radiological Scoring System based on the Balthazar classification system and the CT Severity Index – correlation with morbidity and mortality.<sup>15</sup>

<table>
<thead>
<tr>
<th>Prognostic indicator</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatic inflammation</td>
<td></td>
</tr>
<tr>
<td>Normal pancreas</td>
<td>0</td>
</tr>
<tr>
<td>Intrinsic pancreatic abnormalities with inflammatory changes in the peri-pancreatic fat</td>
<td>2</td>
</tr>
<tr>
<td>Pancreatic or peri-pancreatic fluid collection or peri-pancreatic fat necrosis</td>
<td>4</td>
</tr>
<tr>
<td>Pancreatic necrosis</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>&lt;30%</td>
<td>2</td>
</tr>
<tr>
<td>&gt;30%</td>
<td>4</td>
</tr>
</tbody>
</table>

Extra-pancreatic complications

- One or more of pleural effusion, ascites, vascular, haemorrhage or pseudoaneurysm formation, parenchymal complications (infarction, haemorrhage or sub-capsular fluid collection) or GI involvement (inflammation, perforation or intramural fluid collection)

Where mild acute pancreatitis is 0-2 points, moderate is 4-6 points and severe is 7-10 points.

**TABLE 2**
Modified CT severity index.<sup>14</sup>

The onset of an acute attack. With this delay, the scan yields a higher accuracy in the depiction of necrotising pancreatitis. However, in patients with an acute abdomen of unknown aetiology, CE-CT is often performed at admission and the diagnosis of acute pancreatitis is made at this time.

**CE-CT technique**

At our institution, the standard protocol for imaging acute pancreatitis is 100ml intravenous (IV) contrast at a rate of 3ml/second, with image acquisition at approximately 60 seconds (optimal pancreatic enhancement is at 40 seconds, but imaging at 60 seconds optimises vascular opacification and enhancement of other organs).

**Morphological stages of acute pancreatitis**

**Interstitial oedematous pancreatitis (IOP)**

IOP is characterised by enlargement of the pancreas (which can be localised or diffuse) and homogeneous enhancement of pancreatic parenchyma following IV contrast administration. Adjacent peri-pancreatic and retroperitoneal tissues can appear entirely normal or may demonstrate haziness and stranding with varying amounts of associated peri-pancreatic fluid.

**Necrotising pancreatitis**

The Atlanta Classification defines necrotising pancreatitis as being associated with more than 30% parenchymal necrosis. Pancreatic necrosis (figure 2) is seen as diffuse or focal areas of non-viable pancreatic tissue, indicated by the lack of parenchymal enhancement following IV contrast administration (enhancement of less than 30 HU correlates well with necrosis).<sup>16</sup>

**FIGURE 2**

Pancreatic necrosis – CE-CT demonstrating pancreatic necrosis 11 days following admission with acute abdominal pain. The pancreas is swollen and the majority of the parenchyma is non-enhancing/poorly enhancing (black arrows). Only the pancreatic head and uncinate process enhance normally (*). Necrosis develops early in the course of SAP (typically within the first 48 hours) and is usually well established by 96 hours after onset of clinical symptoms. CE-CT can be equivocal in the first 12 hours. The process starts initially as solid necrosis and progresses to liquefaction. Complete resolution of necrosis can occur with eventual reabsorption of this fluid component. Patients with sterile necrosis do not generally require intervention unless they remain persistently unwell (usually secondary to mass effect) for more than four weeks after onset.

Pancreatic necrosis is sub-divided into three types:<sup>15,16</sup>

1. Organised pancreatic necrosis (OPN): An evolving collection characterised by encapsulation and inhomogeneous contents of liquefied, necrotic fatty tissue and solid necrotic pancreatic and extra-pancreatic debris.<sup>15</sup>

2. Central gland necrosis/disconnected duct syndrome: Full width necrosis of the pancreatic neck with or without involvement of the pancreatic body but with sparing of the head and the tail (due to collateral blood supply). This pattern of involvement results in disruption of the pancreatic duct and persistent collections due to continued secretions from the tail, often requiring distal pancreatectomy for definitive treatment.

3. Extra-pancreatic necrosis (EXPN): Necrosis in the peri-pancreatic tissues in the absence of pancreatic necrosis. The pancreas is seen to enhance homogeneously following contrast administration.<sup>15</sup>

**Complications of acute pancreatitis**

**Pancreatic/peri-pancreatic fluid collections**

Acute peri-pancreatic fluid collection (APFC): Collection of enzyme-rich pancreatic juice in or near the pancreas occurring early in the course of acute pancreatitis. APFCs lack solid components and are low attenuation on CT. They conform to the anatomical boundaries of the retroperitoneum and do not have an inflammatory or fibrous capsule. These collections generally remain sterile and resolve spontaneously in about 50% of patients.<sup>14</sup> Intervention at this
stage can be detrimental and may convert a sterile collection into an infected one.27

Pseudocyst: APFCs that persist for more than four weeks after the onset of acute pancreatitis are termed pancreatic pseudocysts. They are defined as a collection of pancreatic juice enclosed by a well-defined wall of fibrous or granulation tissue and are usually sterile (figure 3).14-24 Pseudocysts occur as a complication of pancreatitis in 10-20% of patients.25 Approximately 50% of pseudocysts resolve spontaneously without becoming symptomatic, while 25% will result in clinical complications, eg pain and infection.25

Asymptomatic pseudocysts do not require treatment.

FIGURE 3
Pancreatic pseudocyst – CE-CT demonstrating a well defined peri-pancreatic fluid collection with a fibrous capsule (*) six weeks following the patient’s acute episode of pancreatitis.

Post-necrotic pancreatic/peri-pancreatic fluid collection (PNPFC): Develop as a sequel of pancreatic necrosis. They are evolving collections characterised by inhomogeneous contents of liquefied, necrotic fatty tissue and solid necrotic pancreatic and extra-pancreatic debris.26-28 PNPFC are often associated with necrosis of a segment of the main pancreatic duct. If there is concern regarding duct disruption, initial imaging with magnetic resonance cholangiopancreatography (MRCP) or secretin-stimulated MRCP provides non-invasive evaluation of the pancreatic duct.22,27,28

Walled-off pancreatic necrosis (WOPN): Formed as the PNPFC matures in a similar way to the development of a pseudocyst from APFC.21 WOPN is an irregular, partially liquefied collection containing solid luminal content that develops as a late consequence of necrotising pancreatitis where areas of liquefactive necrosis are not reabsorbed. These fluid collections often have a thickened non-epithelialised wall, can extend into the peri-pancreatic space and may be sterile or infected.12,29

When PNPFC and WOPN become infected, the associated mortality more than doubles.13-15 Clinical and radiological differentiation between sterile and infected collections is often difficult. However, the presence of locules of gas within the collection is highly suspicious for infection (figure 4).

Vascular complications
Vascular complications occur in approximately 25% of patients with acute pancreatitis.31

Venous thrombus: Splenic vein thrombosis is the commonest vascular complication of acute pancreatitis and occurs in 10-40% of patients.32 Long term, thrombosis may result in portal hypertension, subsequent variceal formation and splenic infarction.

Pseudoaneurysm formation: Pseudoaneurysms result from the digestive effects of pancreatic enzymes on local arteries causing weakness of the arterial wall. Pseudoaneurysms have a tendency to enlarge and ultimately rupture, potentially resulting in life threatening haemorrhage. Arterial phase CT (combined with maximal intensity projections) allows optimal delineation of pseudoaneurysm anatomy and identification of active bleeding, providing a roadmap for subsequent intervention.

FIGURE 4
Infected pancreatic necrosis - CE-CT showing a gas-containing fluid collection in and around the pancreatic bed (white arrowheads). Locules of gas within the collection raise the index of suspicion for associated infection. A scan seven days previously demonstrated extensive pancreatic necrosis with large peripancreatic fluid collections (Balthazar grade E). Only the pancreatic head and uncinate process enhance normally following IV contrast medium (*).

Role of interventional radiology
Late complications of acute pancreatitis significantly add to the degree of morbidity. Image-guided intervention has an important role in the management of these complications. Percutaneous drainage procedures
Image-guided drainage procedures have been found to be an effective alternative to surgical debridement in patients with pancreatitis-associated complications.33

Indications for drainage include:
- Symptomatic collections which are exerting mass effect, causing pain or have become infected.34
- Pseudocysts larger than 5cm that have been present for longer than six weeks, which are unlikely to resolve spontaneously.25
- Fluid contained in collections caused by pancreatic necrosis is often viscous, therefore adequate drainage requires multiple side holes and large bore catheters (up to 28-Fr). Meticulous irrigation (ideally with sterile saline at least three times a day) is required to maintain patency.

Endovascular treatment of pseudoaneurysms
The endovascular approach to pancreatitis-associated pseudoaneurysms depends on the location and morphology of the pseudoaneurysm.35 The first-line approach is coil embolisation where the pseudoaneurysm sac is occluded by the placement of metallic coils. This technique is employed in aneurysms with narrow necks and in locations where coils can be deployed safely without the risk of non-target embolisation. In cases where the configuration of the pseudoaneurysm neck is suboptimal or where there is a high risk of non-target embolisation, a covered stent can be placed to exclude the pseudoaneurysm from the circulation.

Conclusion
Acute pancreatitis can be severe and life-threatening. Imaging is central in the identification of complications and radiological scoring systems can predict prognosis. With the current move towards minimally-invasive treatment, the role of image-guided therapy is increasing and the need for surgical intervention is decreasing.

References