Review Article

Role of computed tomography and magnetic resonance imaging in local complications of acute pancreatitis

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Abstract: Acute pancreatitis (AP) represents a pancreas inflammation of sudden onset that can present different degrees of severity. AP is a frequent cause of acute abdomen and its complications are still a cause of death. Biliary calculosis and alcohol abuse are the most frequent cause of AP. Computed tomography (CT) and magnetic resonance imaging (MRI) are not necessary for the diagnosis of AP but they are fundamental tools for the identification of the cause, degree severity and AP complications. AP severity assessment is in fact one of the most important issue in disease management. Contrast-enhanced CT is preferred in the emergency setting and is considered the gold standard in patients with AP. MRI is comparable to CT for the diagnosis of AP but requires much more time so it is not usually chosen in the emergency scenario. Complications of AP can be distinguished in localized and generalized. Among the localized complications, we can identify: acute peripancreatic fluid collections (APFC), pseudocysts, acute necrotic collections (ANC), walled off pancreatic necrosis (WOPN), venous thrombosis, pseudoaneurysms and haemorrhage. Multiple organ failure syndrome (MOFS) and sepsis are possible generalized complications of AP. In this review, we focus on CT and MRI findings in local complications of AP and when and how to perform CT and MRI. We paid also attention to recent developments in diagnostic classification of AP complications.

Keywords: Acute pancreatitis (AP); computed tomography (CT); magnetic resonance imaging (MRI)

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Introduction

Acute pancreatitis (AP) represents a pancreas inflammation of sudden onset; it can occur with different degrees of severity ranging from mild gland inflammation to massive pancreatic necrosis. The annual incidence of acute pancreatitis varies from 5 to 70 new cases per 100,000 people and, in the USA is responsible for over 200,000 hospital admissions each year (1-3). Biliary calculosis and alcohol abuse cause about 70% of cases of acute pancreatitis (4). Less common causes are metabolic disorders (hypertriglyceridemia and hypercalcemia), drugs (azathioprine and mercaptopurine), infections (paramyxovirus, coxsackievirus, ascaris lumbricoides), tumours (pancreatic adenocarcinoma and lymphoma), abdominal trauma (especially in children), endoscopic retrograde cholangiopancreatography (ERCP), functional alteration of the sphincter of Oddi and congenital anomalies (coledococele, pancreas divisum and duodenal duplication cyst). Furthermore, twenty percent of cases

of acute pancreatitis are idiopathic, although most are thought to be caused by the passage of biliary sludges and microlithiasis (4,5). Acute pancreatitis can simulate other clinical disorders like inferior wall myocardial infarction and other causes of acute abdomen (intestinal obstruction, mesenteric ischemia or infarction, perforation of gastric or duodenal ulcer, biliary colic and aortic dissection) (6). According to the recent guidelines, to diagnose this condition it is necessary fulfilling two of the following three criteria (7): (I) epigastric pain, more or less intense, often radiating to the back and generally associated with nausea and vomiting; (II) increase of serum amylase and lipase values at least three times compared to normal limits; (III) characteristic findings of acute pancreatitis on contrast-enhanced computed tomography (CT) and less commonly magnetic resonance imaging (MRI) or ultrasound (US). Diagnosis of acute pancreatitis can be confirmed even without the use of radiological imaging. CT or MRI may be required to identify the cause, to assess the degree of severity, to predict the course and to identify possible complications.

Severity level of acute pancreatitis

AP severity assessment is one of the most important issue in disease management. Atlanta Classification was formulated in 1992 in the attempt to classify acute pancreatitis and its complications (8). This classification was modified and implemented in 2012 and 2016. The revised Atlanta classification subdivides acute pancreatitis into two types: interstitial edematous pancreatitis (IEP) and necrotizing pancreatitis. These changes have allowed both to improve the differences between acute IEP and acute necrotising pancreatitis, and to implement the definitions of its complications. The clinical evaluation of severe AP is often complex and unreliable; it is estimated that even experienced doctors in less than half of the cases (40%) will be able to predict which patients will develop severe AP based only on clinic results (9). The most used clinical scores developed with the aim of more accurately and reproducibly assessing the severity of AP are the APACHE II criteria, Ranson criteria and Glasgow criteria (10).

When and how to perform CT and MRI

In most cases the symptoms of AP are nonspecific and, as reported in the literature, serum lipase and amylase levels do not correlate with the severity of the disease. Contrast-enhanced CT (using iodinated contrast medium injected intravenously at a flow rate of 3–5 mL/sec) is recommended when is necessary to confirm the diagnosis, identify (where possible) cause and complications, rule out alternative causes of abdominal pain, assess the extent of acute pancreatitis and also for the preoperative planning (1,11).

CT acquisition protocol consists of:

(I) unenhanced acquisition, if possible preceded by the oral administration of 500 mL of water acting as negative contrast increases the difference between the second duodenal portion and the head of the pancreas. This phase allows also the identification of some causes of acute pancreatitis (e.g., biliary microlithiasis);

(II) parenchymal phase (40 seconds) is the optimal phase for the identification of pancreatic necrosis areas. In fact, in this phase the healthy pancreatic tissue has the maximum enhancement;

(III) portal phase (70–80 seconds) extended to the whole abdomen useful for identifying some complications (e.g., venous thrombosis) and associated pathologies.

To these phases we can add the arterial phase (20 seconds) and a delayed phase (3–5 minutes) for the detection of haemorrhage and pseudoaneurysms. It is common to use a double-phase technique (parenchymal and portal phases) but with this protocol we risk missing the haemorrhagic collections (11). MRI is comparable to CT for the diagnosis of acute pancreatitis but requires much more time so it is not usually chosen in the emergency scenario. In clinical practice we use MRI when the patient is allergic to iodine contrast, when we want to better evaluate the pancreatic ductal system and biliary three, the bile duct and also for a better characterization of peri-pancreatic collections (12). CT is considered the gold standard in patients with AP; however, it exposes patients to radiation burden, increased by follow-up examinations; furthermore, the use of iodinated contrast media can potentially aggravate acute pancreatitis (13).

Advantaged of MRI use are:

(I) MRI is a diagnostic imaging method with no radiation hazard, which might be suitable for patients with multiple follow-up controls;

(II) MRI has fewer contraindications than CT and is a reliable method for severity staging of acute pancreatitis, which has predictive value for the prognosis of the disease;

(III) MRI cholangiopancreatography (MRCP) has the unique capability of providing non-invasive images of the pancreatic ducts and can demonstrate possible communication of a pancreatic pseudocyst

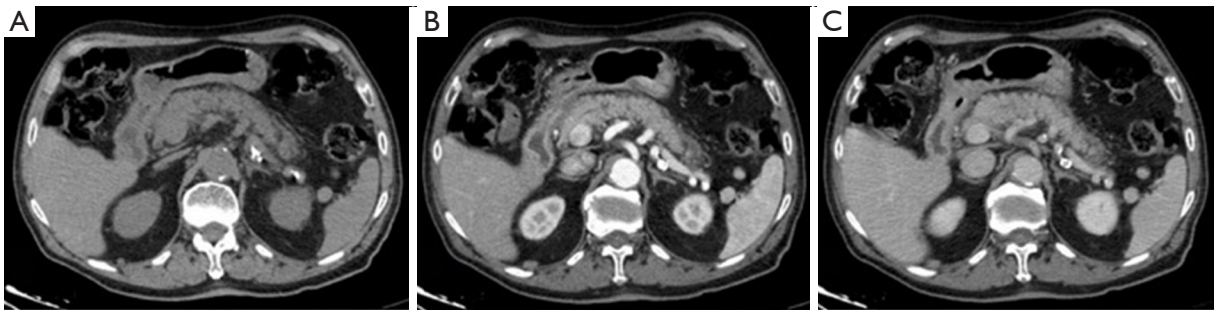

Figure 1 IEP in unenhanced phase (A), parenchymal phase (B) and portal phase (C). IEP, interstitial edematous pancreatitis.

with pancreatic ducts (14);

(IV) MRI is useful for assessing signal intensity of fluid exudation or pseudocysts; to identify local haemorrhage or pseudoaneurysm, which might help plan the surgery.

Regarding the MRI protocol today, the introduction of the fat-suppression techniques, breath-hold fast sequences and phased-array coils has permitted to increase contrast resolution of pancreatic and peripancreatic tissues.

Consequently, the use of MRI has become more frequent in patients with AP complications. The acquisition protocol requires the combined use of T1-weighted (T1-w), T2-weighted sequence (T2-w), MRCP sequences and T1-w sequences with fat suppression [e.g., fast spin-echo (FSE)] imaging with multiple breath-hold acquisitions or single-breath-hold gradient echo imaging to improve the delineation of pancreatic borders and the pancreas itself; T1-w sequences allow also the evaluation of haemorrhagic complications of acute pancreatitis (15-17). T2-w sequences [e.g., fast recovery fast spin-echo (FRFSE) triggered or breath-hold single-shot fast spin-echo (SSFSE)] imaging has significant advantages in demonstrating fluid-filled lesions in or around the pancreas and the pancreatic duct (17,18). SSFSE T2-w sequences can be used to guide acquisition of an MRCP series which (obtained before gadolinium administration) allows non-invasive evaluation of pancreatic ducts and the whole extrahepatic biliary tract, and provides few respiratory artefacts or susceptibility effects (17,19). Diffusion weighted imaging (DWI) can display the manifestations of AP with water molecules restriction in an earlier phase compared to other imaging modalities and without radiation hazard (20).

Dynamic imaging after intravenous administration of gadolinium performed with T1-w acquisition [e.g., liver acquisition with volume acceleration (LAVA)] with the same timing of contrast-enhanced CT gives a comprehensive evaluation of the extent of the necrosis and the full range of the inflammatory extension for the initial staging of acute pancreatitis. Moreover, MR angiography, the post-processing technique after MR LAVA, can be performed to supplement the information for visualization of pancreatic vascular network and vascular complications of acute pancreatitis (17,19).

Different forms of pancreatitis

IEP

IEP is the most common type of AP, found in 90–95% of cases. CT detects a focal or diffuse parenchymal enlargement caused by inflammatory oedema and, after contrast administration of contrast medium, the pancreatic parenchyma will show a homogeneous enhancement. In addition to this, generally the peripancreatic fat will show fluid imbibition, which is often associated to peripancreatic effusion (*Figure 1*). MRI shows an increased signal of the pancreas and peripancreatic tissues in T2-w sequences, a low signal in T1-w sequences in the same locations, and restriction of the water molecules on DWI (*Figure 2*). Clinical symptoms of interstitial pancreatitis usually resolve within the first week (21,22).

Necrotizing pancreatitis

Necrotizing pancreatitis occurs in 5–10% of cases; it is characterized by a protracted clinical course, a high incidence of local complications, and a high mortality rate.

There are three subtypes of necrotizing pancreatitis; the subtypes are based on the anatomic area of necrotic involvement: (I) pancreatic only; (II) peripancreatic only; and (III) combined pancreatic and peripancreatic. The latter subtype is the most common (75% of cases). In this type of pancreatitis one or more areas of pancreatic parenchyma

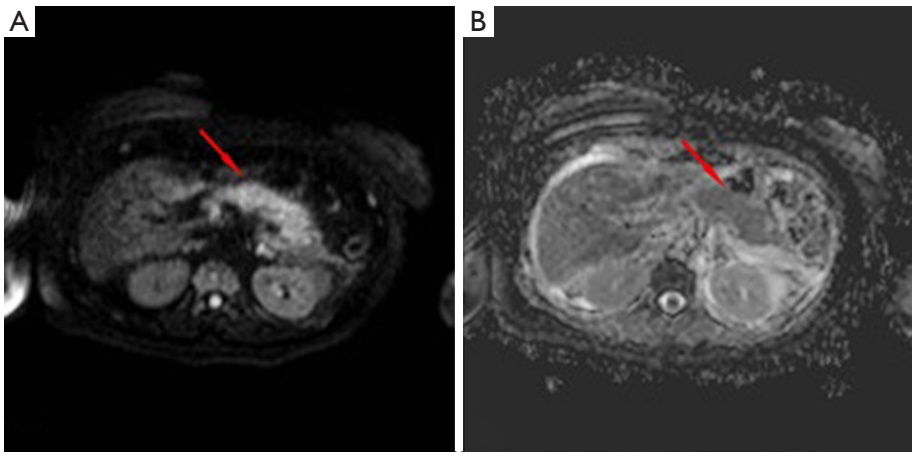

Figure 2 IEP (arrows) red arrows indicate signal restriction at the body-tail of the pancreas in DWI image (b800, A) and ADC map (B). IEP, interstitial edematous pancreatitis. DWI, diffusion weighted imaging; ADC, apparent diffusion coefficient.

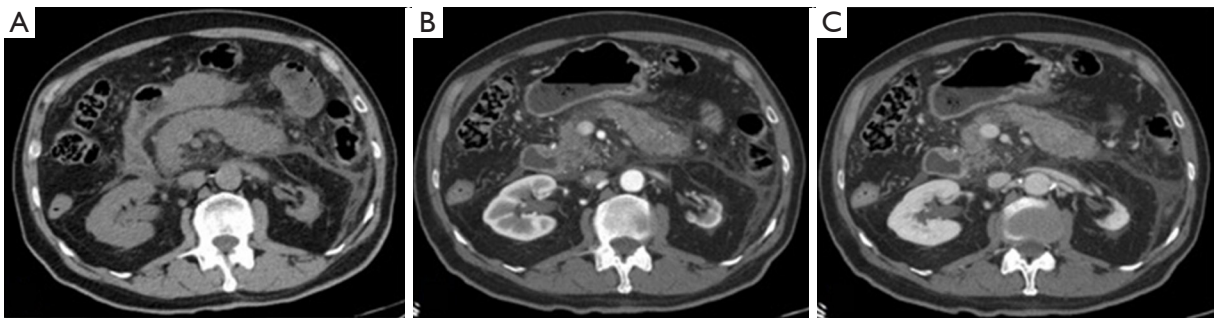

Figure 3 Necrotizing pancreatitis in unenhanced phase (A), parenchymal phase (B) and in portal phase (C).

or peripancreatic tissue show an unenhanced or minimally enhanced (<30 HU) areas on contrast-enhanced CT or gadolinium enhanced MR images (10,19,23) (*Figure 3*). This imaging is established within a few days, this explains why an early CT can underestimate the extent of necrosis (23-26). The evolution of pancreatic necrosis is variable, can remain solid or liquefy, remain sterile or become infected, disappear or persist over time. These patients have higher morbidity than patients with IEP.

Complications

Complications of AP can be distinguished in localized and generalized. Among the localized complications we can identify: acute peripancreatic fluid collections (APFC), pseudocysts, acute necrotic collections (ANC), walled off pancreatic necrosis (WOPN), venous thrombosis, pseudoaneurysms and haemorrhage. The old term pancreatic abscess is abandoned because any collection can be sterile or infected, otherwise the infection occurs more often in the necrotic collections (27). Multiple organ failure syndrome (MOFS) and sepsis are possible generalized complications of AP.

APFC and pseudocyst

The acute peripancreatic fluid collection represents an early complication (<4 weeks) of acute IEP. APFCs can occur in the first hours after the onset of symptoms and are composed exclusively of fluid material. Contrast-enhanced CT will show fluid, capsule-free collections arranged around the pancreas (*Figure 4*). MRI, with fat suppressed T1- and T2-w sequences, can accurately depict APFCs with liquid signal performance of hypointensity on T1-w images and hyperintensity on T2-w images (*Figure 5*) (16). In some cases, APFCs will be placed in the anterior

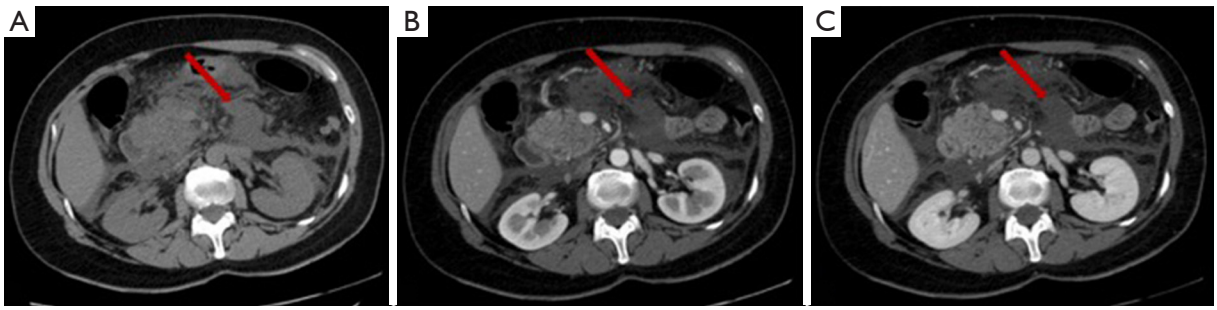

Figure 4 APFC CT examination in unenhanced phase (A), parenchymal phase (B) and in portal phase (C); red arrows showing APFC. CT, computed tomography; APFC, acute peripancreatic fluid collections.

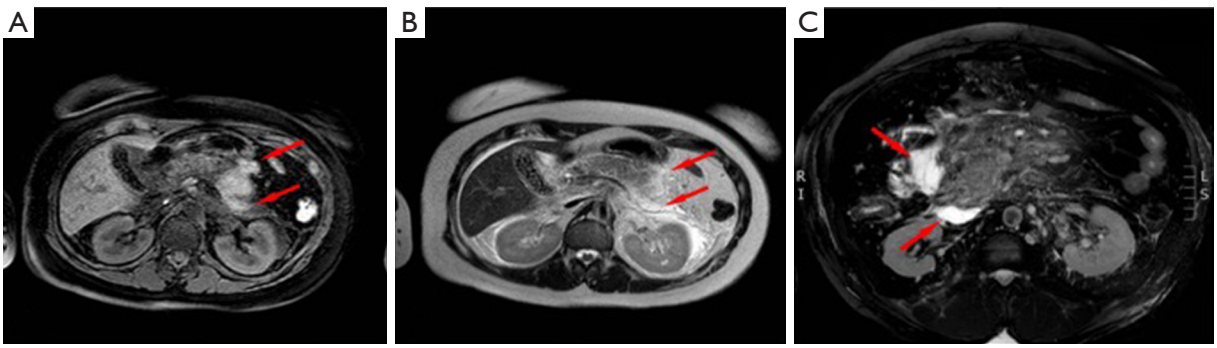

Figure 5 APFC in unenhanced axial magnetic resonance T2-w images with (A,C) and without (B) fat-suppression. Red arrows showing peripancreatic fluid collections. T2-w, T2-weighted; APFC, acute peripancreatic fluid collections.

pararenal space (more commonly on the left), transverse mesocolon, mesenteric root, gastro-hepatic ligaments, gastrosplenic and gastrocolic (18,28,29). APFCs remain sterile and disappear spontaneously within 2–4 weeks in 50% of patients. If APFCs are sterile, it is not appropriate to drain them because they usually resolve spontaneously and their aspiration may cause infection (30). Only infected APFCs have to be drained. If an APFC did not resolve after 4 weeks, it becomes more organized and develops a fibrous tissue capsule. This collection is called pseudocyst and represents the late complication (>4 weeks) of IEP. Pseudocysts develop in less than 10% of IEP cases (18). According to spatial locations, pancreatic pseudocysts are classified as intraparenchymal or extrapancreatic. The intraparenchymal pseudocyst might be communicating with pancreatic ducts and associated with partial pancreatic ductal obstruction (29,31). Among the most important complications of pseudocysts we can identify infection, compression on adjacent organs (stomach, duodenum, biliary system) and rupture (peritonitis). At contrast-enhanced CT the pseudocysts appear as collections of well-circumscribed peripancreatic fluids, usually round or oval of homogeneously low density, surrounded by a well-defined wall with enhancement (*Figure 6*). MR findings of a simple pseudocyst include a round or oval fluid collection surrounded by a thin wall, with liquid signal performance of hypointensity on T1-w images and hyperintensity on T2-w images (*Figure 7*). Pancreatic pseudocysts can also present as complex pseudocysts associated with mucus, protein, and haemorrhage with heterogeneous hyperintense signal on T1-w images with fat suppression (29,31). The three main treatment modalities are endoscopic drainage (the preferred treatment), percutaneous drainage and surgical drainage.

ANC and WOPN

The ANCs represent an early complication (<4 weeks) of acute necrotizing pancreatitis. Unlike the APFC their content is heterogeneous and consists of fluid, solid debris and fat. They can be single or multiple and be in a peripancreatic position or spread throughout the

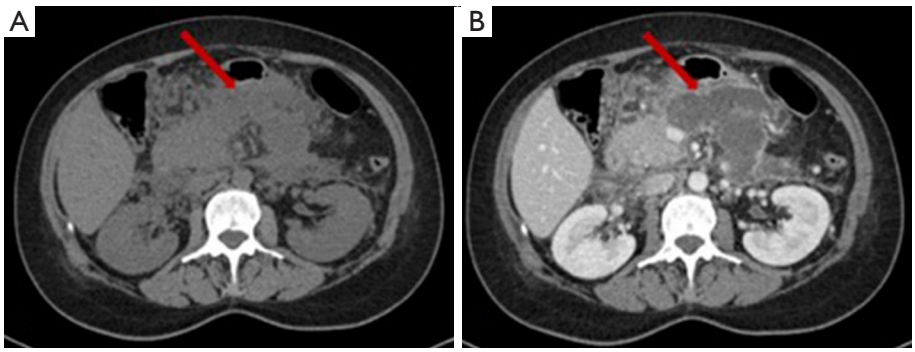

Figure 6 Pseudocyst: red arrows indicate pseudocyst in unenhanced (A) and in enhanced portal phase (B) CT examination. CT, computed tomography.

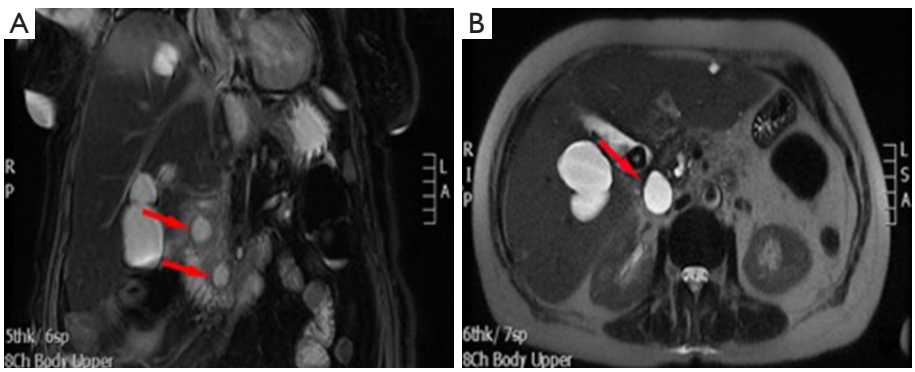

Figure 7 Pseudocyst (red arrows) in unenhanced coronal and axial magnetic resonance T2-w images with (A) and without (B) fat suppression. T2-w, T2-weighted.

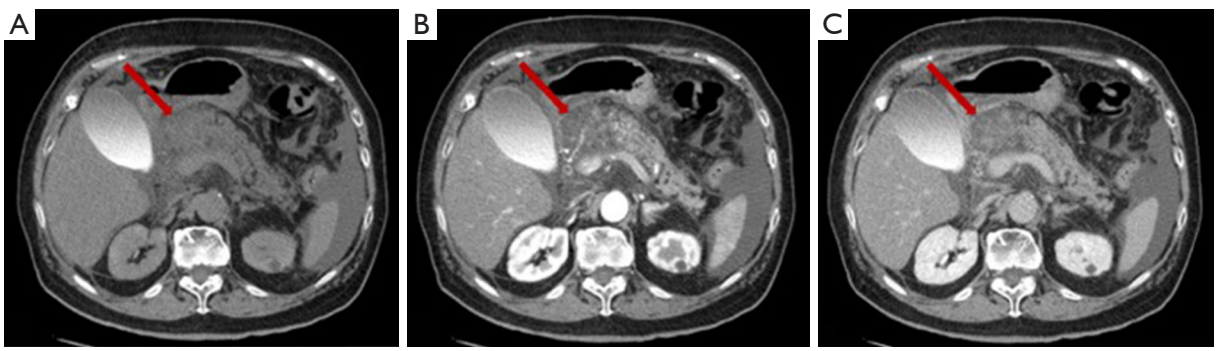

Figure 8 ANC (arrows) in unenhanced phase (A), parenchymal phase (B) and portal phase (C). ANC, acute necrotic collections.

abdomen. The contrast-enhanced CT will allow us to see heterogeneous, wall less collections in a patient with acute necrotic pancreatitis (*Figure 8*). MRI (with fat suppressed T1- and T2-w sequences) can accurately depict ANC with liquid signal performance of hypointensity on T1-w images and hyperintensity on T2-w images with areas of haemorrhage or necrosis of pancreas and peripancreatic fat tissue which appear with iso-hyperintensity on T1w images (*Figures 9,10*).

The contents of the collection can be sterile or infected. Sterile pancreatic necrosis does not require treatment, especially if the patient has a stable clinical status. However,

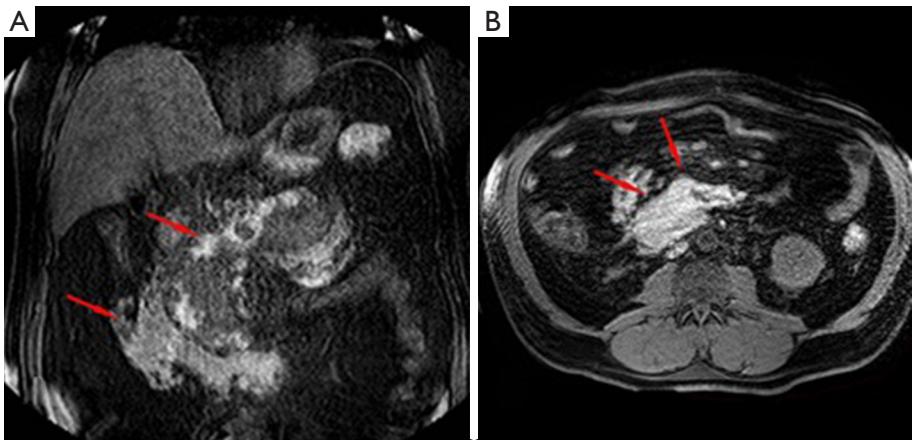

Figure 9 ANC in unenhanced T1-w fat-sat coronal (A) and axial (B) images with evidence of haemorrhagic collection (red arrows). ANC, acute necrotic collections; T1-w, T1-weighted.

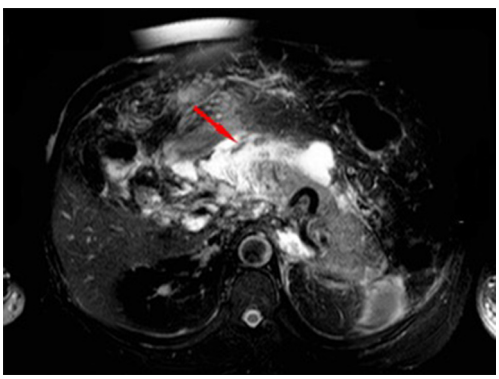

Figure 10 ANC with necrosis of peripancreatic fat tissue (arrow) in unenhanced axial magnetic resonance T2-w image with fat suppression. ANC, acute necrotic collections; T2-w, T2-weighted.

the possibility of infection increases with the time. After 4 weeks the acute necrotic collection will develop a thick capsule visible at contrast-enhanced CT. This formation will be called WOPN and represents the late complication (>4 weeks) of acute necrotizing pancreatitis. WOPN is an irregular, partially liquefied collection, which may contain solid and fat debris (*Figure 11*). MR T1- and T2-w images with and without fat suppression, can easily and better than CT, depict necrotic debris or haemorrhagic components of the pancreatic parenchyma with signal hyperintensity on T1-w sequences and hypointensity on T2-w sequences (*Figure 10*) (15).

WOPN can be sterile or infected. The differentiation of WOPN from the pancreatic pseudocyst is essential because management differs. WOPN may need aggressive treatment (most centres prefer the treatment with operative necrosectomy in the infected or symptomatic cases) to avoid complications.

Venous thrombosis

Splanchnic venous thrombosis is a rare complication of AP. Venous thrombosis often involves the splenic vein, the portal vein and the superior mesenteric vein, both in combination or separately.

Splenic vein thrombosis is the most common form and is due to the inflammatory intimal injury or to the ab-extrinsic compression by the fluid collections (32). This can cause portal hypertension, development of venous ectasia and splenic infarction (33).

Pseudoaneurysm

Pseudoaneurysm is a rare but serious complication of acute pancreatitis and occurs in 4% to 10% of cases (34). Erosion of the arteries is caused by the proteolytic enzymes released by the pancreas (35-37). Pseudoaneurysms can break in the peritoneal cavity, in the retroperitoneum, in adjacent collections and rarely in the pancreatic duct (35). For their diagnosis it is useful to perform CT in the arterial phase.

Haemorrhage

Like pseudoaneurysms, haemorrhage is caused by the release of proteolytic enzymes from the pancreas. Fortunately, it is a rare complication but it is often lethal.

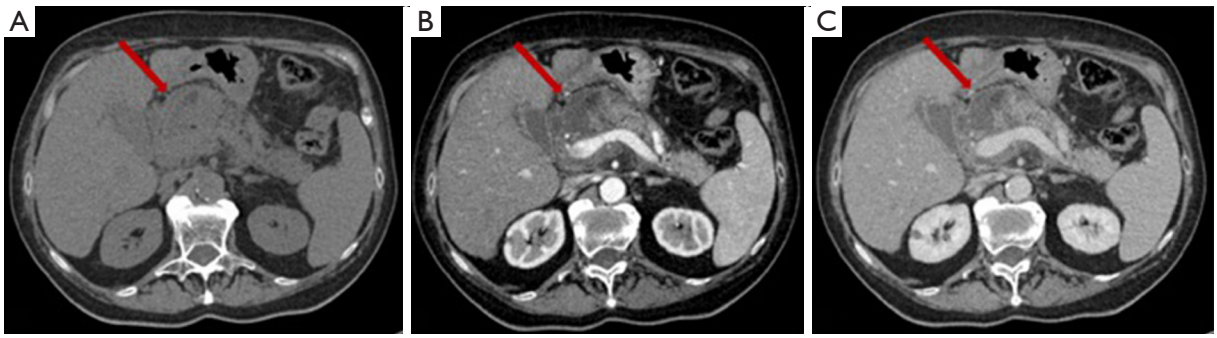

Figure 11 WOPN in unenhanced phase (A) parenchymal phase (B) portal phase (C) CT examination (red arrows). WOPN, walled off pancreatic necrosis; CT, computed tomography.

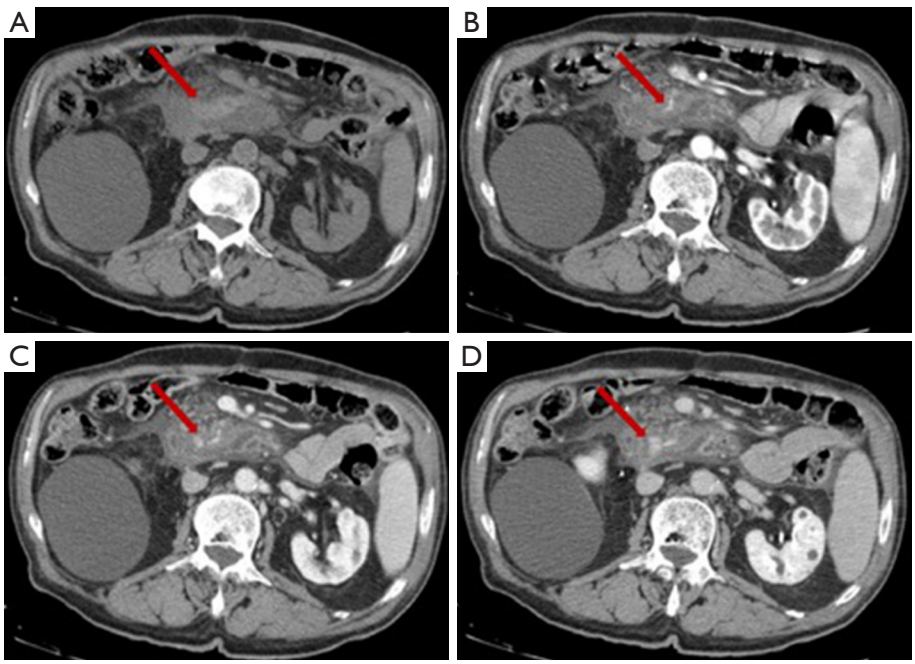

Figure 12 Haemorrhage on CT examination (red arrows): in unenhanced phase (A), parenchymal phase (B), portal phase (C) and in delayed phase (D).

Splenic artery, gastroduodenal artery and pancreatic-duodenal artery are the most frequent involved arteries (38). In this case it is useful to perform CT in the arterial phase (*Figure 12*). T1-w fat suppression images may reveal the presence of bleeding prior contrast agent administration.

Abdominal compartment syndrome

Abdominal compartment syndrome can be defined as an "acute elevation of the intraabdominal pressure with organ dysfunction". Haemorrhagic pancreatitis and large amount of pancreatic ascites can be one of the causes of abdominal compartment syndrome. The prevalence of intraabdominal hypertension in patients with severe acute pancreatitis is about 40–50% (39). Diagnosis of abdominal compartment syndrome is often complicated; multiorgan failure, sepsis and acute respiratory distress syndrome are often seen in these patients. Radiological findings are few and non-specific. Between the radiological findings that can be found in these patients we can describe: elevated diaphragm, rounded configuration of abdominal wall (anteroposterior-to-lateral girth ratio >0.8), hemoperitoneum, flattened inferior vena cava, flattened renal veins, mosaic liver perfusion, increased bowel enhancement, increased gastric

wall enhancement, gastric distention, reduced diastolic flow in portal, hepatic or renal veins on sonography (40).

Conclusions

Acute pancreatitis represents a frequent cause of acute abdomen and its complications are still a cause of death. CT and MRI represent the best clinical and surgery friend in the identification of acute pancreatitis and its complications. Imaging findings of acute pancreatitis are crucial for the timing and management of acute pancreatic complications in the emergency setting.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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