Review Article

MR imaging for acute pancreatitis: the current status of clinical applications

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Abstract: Acute pancreatitis is a common clinical acute abdomen. Imaging examinations play an important role in the management of acute pancreatitis. MR imaging is a noninvasive examination with high tissue contrast and a variety of acquisition sequences that can help determine the diagnosis, complications and severity of acute pancreatitis. The acute pancreatitis classification working group modified the Atlanta classification in 2012 to improve clinical evaluations and standardize the radiologic nomenclature for acute pancreatitis. In particular, the redefinition of necrotizing pancreatitis offers a new understanding of this disease. In clinical practice, there is still a lack of unifying standards between radiologists and physicians, such as for the imaging features of pseudocysts, walled-off necrosis, peripancreatic necrosis and especially for the MR imaging features of acute pancreatitis. In this article, we review the 2012 revised Atlanta classification of acute pancreatitis and recent advances in the clinical applications of MR imaging (MRI) in acute pancreatitis by showing how MRI can provide more optimized information for clinical diagnosis and treatment plan.

Keywords: MR imaging (MRI); acute pancreatitis (AP); local complication

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Introduction

Acute pancreatitis (AP) is an acute chemical inflammation of the pancreas caused by activated pancreatin. The main clinical manifestations include abdomen pain, nausea and vomiting. Mild AP cases are mainly due to pancreatic edema and have a good prognosis, while severe AP cases are associated with hemorrhage, necrosis, infection, multiple organ failure and even shock (1). AP is usually caused by choledocholithiasis, hyperlipidemia and alcoholism. The disease course, clinical manifestations and prognosis vary greatly between individuals, a timely diagnosis and accurate evaluation of the disease severity is vital for clinical management (1). Furthermore, the complications of AP are closely related to the prognosis of the patients. The Acute Pancreatitis Classification Working Group modified the Atlanta Classification in 2012 (2). The 2012 revised Atlanta classification divided AP into interstitial edematous pancreatitis (IEP) and necrotizing pancreatitis (NP) and introduced two phases of the disease: early and late. The disease severity is classified as mild, moderately severe or severe (2). IEP refers to pancreatitis without necrosis in the pancreatic parenchyma and peripancreatic region, while NP involves necrosis in the pancreatic parenchyma or/and in any part of the peripancreatic region. In the 2012 revised

Atlanta classification of AP, the course of the disease within 1 week is known as the early stage and after a week is known as the late stage. Regarding the severity classification, patients without organ failure are classified as having mild disease, patients with organ failure that recovered within 48 hours are classified as having moderately severe disease, and patients with organ failure that do not recover within 48 hours is classified as having severe disease (2). In addition, the new classification revised the terminology of local complications and subdivides the early complications into acute peripancreatic fluid collections (APFCs) and acute necrotic collections (ANCs) based on the necrotic debris. Delayed complications are also classified into pseudocysts and walled-off necrosis (WON) (3). The new classification clarifies physicians' misconceptions about local complications in different specialties and provides a better background for combined multidisciplinary treatment.

CT is the first choice for diagnosing AP and its local complications (4). CT scans are fast and have high spatial resolution. CT scans can clearly display the pancreas and the surrounding tissues and overcome the limitations that ultrasound examinations are susceptible to, such as the influence of intestinal gas (5). However, CT has some shortcomings in evaluating the severity of AP. On enhanced CT scans, small nonenhanced areas may either be necrosis or local effusion in the pancreas, but it is difficult to distinguish them accurately by CT. Therefore, the possibility of overestimating the scope of necrosis may exist in the diagnosis of pancreatic necrosis with enhanced CT. Furthermore, focal adipose deposition in some elderly people may be misjudged as necrotic foci in the pancreas (6). Thus, the value of enhanced CT for small necrotic pancreases is limited, and the range of necrosis cannot be estimated correctly. As an alternate method for diagnosing acute pancreatitis, MR imaging (MRI) and MR cholangiopancreatography (MRCP) shows great potential in clinical applications (7). The benefits of MRI for acute pancreatitis are as follows: (I) the MRI sequence for T2-weighted image (T2WI) is highly sensitive to liquid and thus can visualize a small amount of liquid in mild pancreatitis, T2WI can characterize non-liquid materials in the pancreatic collections better than CT (6); (II) MRI clearly shows the areas of necrosis without enhancement and is safe for patients who are unable to receive iodinated contrast material because of allergies or kidney failure (8,9); (III) MRCP can noninvasively evaluate the changes of the bile ducts (especially the distal bile duct, which is difficult for ultrasound to show) and pancreatic duct system and help diagnose the etiology of the disease (10); (IV) recent studies have found that in addition to the air bubbles, high signals on the MRI diffusion-weighted imaging (DWI) sequences can also indicate the existence of infection to preliminarily determine whether the accumulation is infected (11); (V) novel MRI techniques such as intravoxel incoherent motion (IVIM) imaging can assess both perfusion and diffusion of the diseased tissues (12-14); (VI) MRI can be used to stage AP and clearly show local complications. It has been reported that non-enhanced MRI is more reliable and accurate than CT in estimating the severity of AP (9); and (VII) finally, MRI does not require ionizing radiation and does not lead to adverse effects on the human body.

In this article, we review the 2012 revised Atlanta classification of AP and recent advances in the clinical applications of MRI in acute pancreatitis by showing how MRI can provide more optimized information for clinical diagnosis and treatment plans.

MRI for the diagnosis of acute pancreatitis

Interstitial edematous pancreatitis

According to the 2012 revised Atlanta classification of AP, AP can be divided into interstitial edema pancreatitis and necrotizing pancreatitis (15). The former accounts for most AP cases and has a mild and self-limiting clinical course. The pancreas has diffuse or localized enlargement due to inflammatory edema, and this type of AP has no pancreatic necrosis or peripancreatic necrosis (2). The parenchyma of the pancreas may display normal or slight hypointensity on T1-weighted images (T1WI) and hyperintensity on T2WI. Patchy-like hyperintensity on T2WI could be observed in the peripancreatic region, perirenal space, and lesser omental bursa. There may be acute peripancreatic fluid collections. The pancreas shows homogeneous enhancement after an intravenous administration of a contrast agent such as Gd-DTPA (*Figure 1*).

Necrotizing pancreatitis

In necrotizing pancreatitis (NP), MRI has higher soft tissue contrast than CT and is superior to CT in showing hemorrhage and tissue necrosis. The sensitivity rate is approximately 76.5% (5). The necrotic area displays hypointensity on T1WI, hyperintensity on T2WI and no enhancement after an injection of contrast agent such as Gd-DTPA. NP can be divided into three subtypes based on the location of necrotic involvement: pancreatic

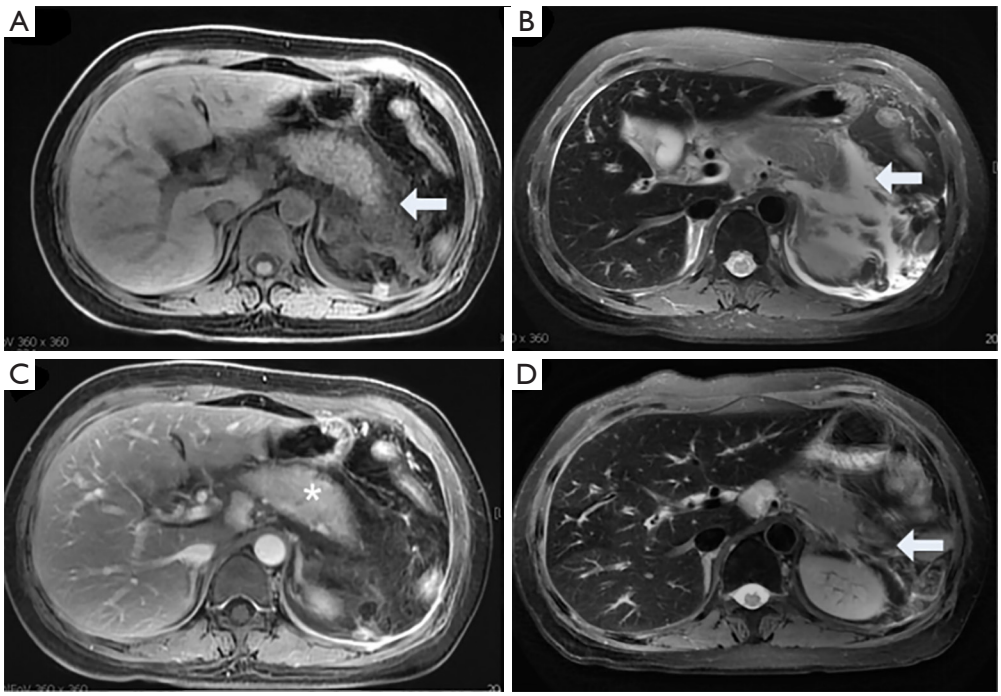

Figure 1 A 61-year-old male with acute edematous pancreatitis. On the third day after onset, the pancreas showed swelling and there was homogeneous peripancreatic fluid collection on the axial T1WI (A, arrow) and the T2WI (B, arrow). The pancreas was homogeneous enhanced after contrast injection (C, asterisk). After 9 days of treatment, the peripancreatic collection was absorbed (D, arrow).

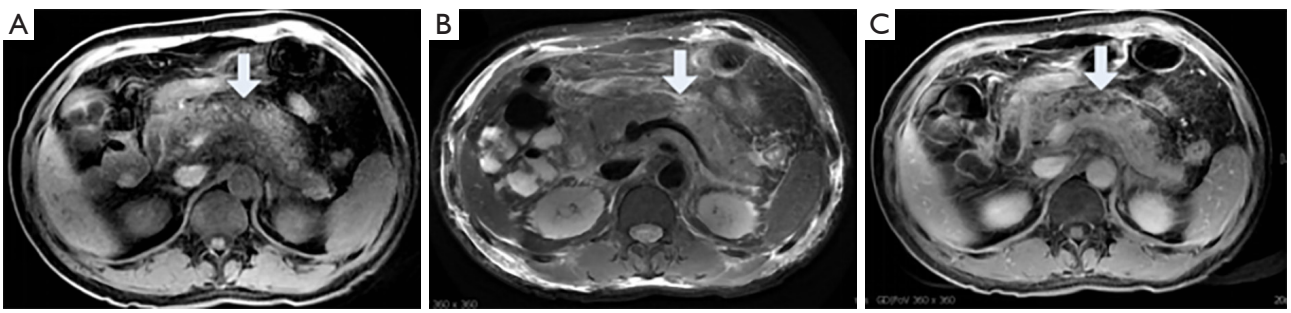

Figure 2 A 45-year-old male with acute necrotizing pancreatitis within 3 days after onset (pancreatic parenchymal necrosis alone). The neck and body of the pancreas show hypointensity combined with patchy isointensity (arrow) on the T1WI (A), high and low mixed signal intensity on the T2WI (B, arrow), and no enhancement after an intravenous injection of contrast material (C, arrow). The extent of necrosis is approximately 30–50% of the pancreatic gland. In the peripancreatic tissue, stripe-like hyperintensity is observed on the T2WI (B).

parenchymal necrosis alone (PN), peripancreatic necrosis alone (PPN), and both pancreatic parenchymal necrosis and peripancreatic necrosis (3). The first type accounts for approximately 5% of all NP cases. The necrotic parenchymal area is not enhanced, and there is no necrotic area in the peripancreatic tissue (*Figure 2*). The extent of necrosis can be stratified as <30%, 30–50% and >50% to grade the severity of pancreatitis (3). The 2012 revised Atlanta classification of AP notes that since the evolution of pancreatic parenchymal necrosis often takes several days, it is uncertain whether the areas with reduced enhancement are necrosis if the enhanced scanning occurs during the first few days (3). To determine whether the early low enhancement area is necrotic, we can only observe whether the area eventually evolves into a completely nonenhanced area through follow-up, but the optimal follow-up time is

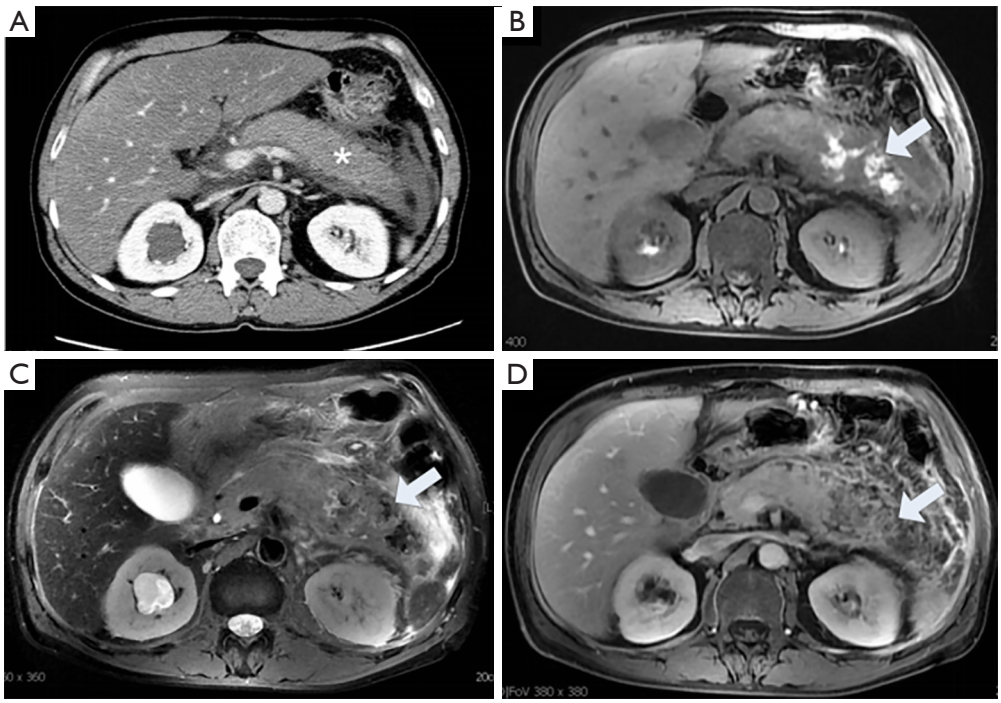

Figure 3 A 55-year-old male with acute necrotizing pancreatitis. On the first day after onset, the enhanced CT image (A) showed enlargement of the body and tail of the pancreas, collections of peripancreatic fluid, and homogeneous enhancement of the pancreatic parenchyma (asterisk). However, on the 9th day after onset, the abdominal MRI T1WI (B, arrow) and T2WI (C, arrow) showed patchy hyper/hypo-intensity, which refers to pancreatic hemorrhages, and a large slightly hypo-/hyperintensity area represented necrosis. After enhancement (D), large nonenhancement areas are found in the body and tail of the pancreas and peripancreas (arrow). MRI, MR imaging.

not clear (*Figure 3*). When the pancreatic neck and body are totally necrotic, these signs typically indicate "disconnected pancreatic duct syndrome" (16).

The second type can be seen in approximately 20% of necrotizing pancreatitis cases. Studies have suggested that the clinical course of PPN is milder than PN but more severe than IEP; therefore, PPN should be considered as a separate type different from the former two (17). The parenchyma of the pancreas shows a homogeneous signal on the T1WI and T2WI and is evenly enhanced (*Figure 4*). Nonfluid components can be found in the peripancreatic collections, which do not show enhancement after an administration of contrast agent. In most instances, the necrosis within the collections cannot be easily confirmed, which creates a difficulty in diagnosing this type of disease and means that PPN can only be diagnosed by fine needle aspiration. Since the morphological changes of extrapancreatic fat may only indicate the accumulation of fluid rather than fat necrosis, the accuracy of this definition in judging PPN demands further exploration. Meyrignac *et al.* (18) defined extrapancreatic fat infiltration, fluid accumulation or the mixed accumulation of liquid and solid components as PPN. Dellinger *et al.* (19) suggested that any accumulation with peripancreatic heterogeneity should be considered peripancreatic necrosis until confirmed. These two definitions may overestimate the incidence of PPN. Sternby *et al.* (20) found that different observers had poor consistency in the diagnosis of PPN, which indicated that the diagnosis of PPN was too subjective. There is no uniform standard for the quantitative analysis of PPN necrosis. A process to accurately identify the necrotic substances of PPN by objective methods still needs to be further studied in the future. Koutroumpakis *et al.* (21) described PPN as "limited" and "diffuse" in terms of the extent of PPN and the number of sites involved. A limited extent refers to an area of peripancreatic necrosis <5 cm, and diffuse extent refers to an extent of peripancreatic necrosis >5 cm or if >3 sites are involved. Rana *et al.* (17) also described PPN as "limited" and "diffuse", but in the literature "limited" extent refers to necrosis confined to the peripancreatic tissue, and the necrosis is "diffuse" if it extends to the paracolic sulcus or involves the pelvic cavity. Additionally, Meyrignac *et al.* (18) have reported that the PPN volume is correlated

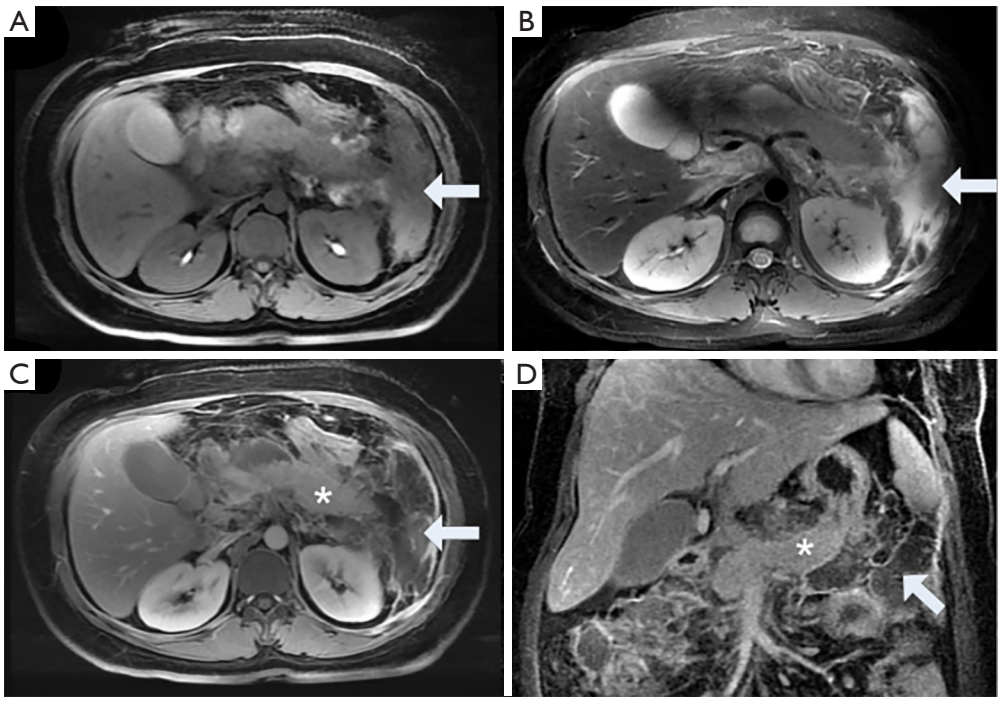

Figure 4 A 52-year-old woman with acute necrotizing pancreatitis 7 days after onset (peripancreatic necrosis alone associated with hemorrhage). Collections of nonfluid components around the body and tail of the pancreas show heterogeneous hypo-/hyper-intensity (arrow) on the T1WI (A) and T2WI (b), and there was no enhancement after a contrast agent administration (C,D) (arrow). The parenchyma of the pancreas shows homogeneous enhancement (asterisk) (C,D).

with the prognosis of AP, and a volume of PPN ≥100 mL may be more reliable than the current scoring systems in assessing organ failure, infection, length of stay and mortality.

The last type is the most common type and accounts for approximately 75–80% of necrotizing pancreatitis (3). The necrosis in the pancreatic parenchyma is not enhanced, and the collections around the pancreas show mixed intensity on T1WI and T2WI but no enhancement after contrast injection (*Figure 5*). The complication and mortality rates of the last type are higher than those of the first two, and this type more likely requires surgical interventions than the other two types (22).

It should be noted that confirming necrosis is difficult at the early stage, even with enhanced scans. Early enhancement is likely to underestimate the final extent of necrosis. Low perfusion injury of the pancreas and the development of peripancreatic fat necrosis occurs after several days, and while hypoperfusion areas in prophase can be restored or can also deteriorate into necrosis (*Figure 3*). Therefore, the assessment of pancreatic necrosis is best performed 4 days after onset using enhanced MRI or CT (23). Moreover, the appearance of nonenhanced parenchyma after one week should be considered as necrosis (6) (*Figure 3*). Hemorrhage can demonstrate complex MRI signals. It is generally considered that the slightly high signal intensity on the T1WI suggests hemorrhage. MRI is superior to CT in visualizing hemorrhages, which can appear as iso-density on CT (24) (*Figure 6*). Necrotizing pancreatitis, especially the third type, has a high prevalence of co-occurring with bacterial infections. Infections are the second leading cause of death in patients with pancreatitis. As soon as the co-infection is identified, antibiotic therapy and surgical treatment (percutaneous drainage or open surgery) are necessary (1). Gas in the necrotic zone on the MRI may indicate infection (25).

MRI for local complications

Acute peripancreatic fluid collections (APFCs)

APFCs usually occur in IEP within 4 weeks of the onset of symptoms (26). APFCs lack cystic walls and solid components and are homogeneously hypointense on T1WIs and hyperintense on T2WI. These collections are

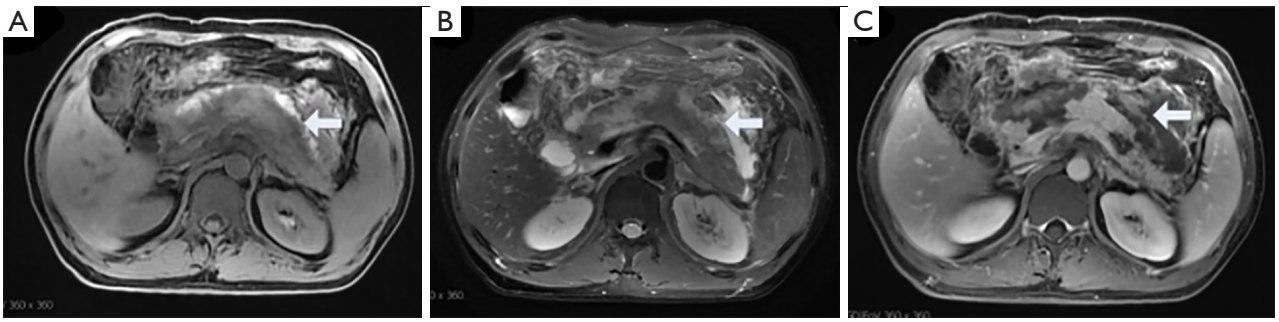

Figure 5 A 49-year-old man with acute necrotizing pancreatitis on the third day after onset (both pancreatic parenchymal and peripancreatic necrosis). The T1WI (A) and T2WI (B) shows heterogeneous signal intensity and small patches of hyperintensity hemorrhage (arrows). After enhancement, both the pancreas and peripancreatic adipose tissue show non-enhanced areas consistent with necrosis or hemorrhage (arrow) (C).

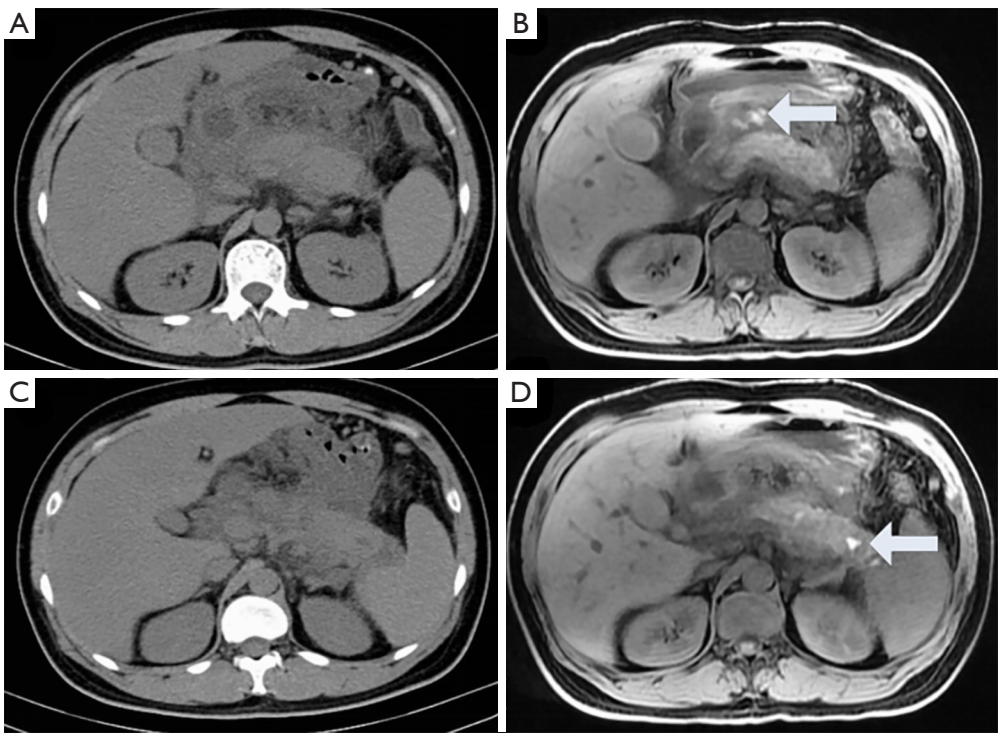

Figure 6 A 32-year-old female patient with acute necrotizing pancreatitis who underwent both CT (A) and MRI (B) examinations within 3 days of onset. The CT image (A,C) show an enlarged pancreas with homogeneous density and peripancreatic collections; however, the MRI T1WI (B,D) clearly shows patchy hyperintensity in the tail and around the pancreas (corresponding to hemorrhages) (arrow). MRI, MR imaging.

limited in the peripancreatic fascial planes and are most commonly found in the lesser omental sac and left pararenal space (*Figure 1*) (3). APFCs are usually sterile, and if the acute edematous pancreatitis is cute, 50% of the collections can be spontaneously absorbed within 2–4 weeks (27). When the protracted course of APFCs is more than 4 weeks, they may develop into pseudocysts (28).

Pancreatic pseudocysts

After the occurrence of acute pancreatitis, pancreatic juice and various inflammatory exudates appear around

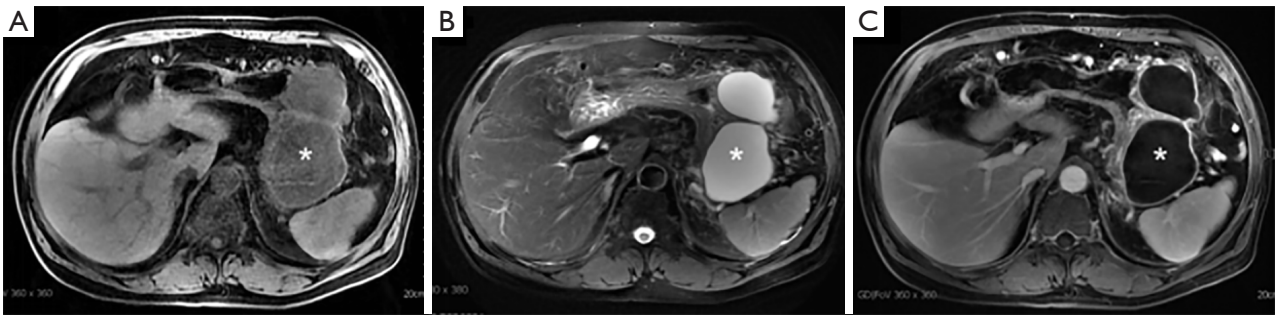

Figure 7 A 68-year-old man with acute pancreatitis 4 months after onset. Two peripancreatic encapsulated fluid collections (asterisk) show hypointensity on the T1-weighted image (A) and hyperintensity on T2-weighted image (B). After contrast injection, only the wall was enhanced (C, asterisk). Fine needle aspiration for the collections confirmed that they were pseudocysts.

the pancreas. If the collection is not absorbed after approximately four weeks, the collection may be surrounded by a capsule due to an inflammatory reaction, which forms a PPC. Because the cyst does not have a true epithelial tissue covering, it is called a pseudocyst. There are different studies on the risk factors of PPC in acute pancreatitis. Some researchers have found that the male sex, alcoholic pancreatitis and ascites are risk factors for PPC formation (29-31). PPCs larger than 6 cm in diameter may be associated with multiple complications, such as mass effect, infection, rupture and bleeding (3,32). In line with the 2012 revised Atlanta classification of AP, there is no non-liquid substance in pseudocysts. MRI can depict pancreatic pseudocysts to reveal a thin smooth wall, and the liquid content displays homogeneous intensity on T1WIs and T2WIs (*Figure* 7). Pancreatic juice constantly overflows from the ruptured pancreatic duct, leading to a gradual enlargement of the cysts and a long period of illness. Due to the need for surgical indications, it is important to visualize where the pancreatic duct breaks and its extent of rupture (33). T2WI, MRCP, and multiplanar reconstruction helps to visualize the broken pancreatic duct and its connection with pseudocysts. Pseudocyst infection is rare. When the pseudocyst is infected, the clear liquid becomes pus but still has no solid content, and the pseudocyst shows a slightly hyperintense signal on T2WIs and DWI scans. Studies have shown that patients who were only complicated with pseudocysts without infection or size-related symptoms should be treated with conservative treatment (34).

Acute necrotic collections (ANCs)

ANCs are only observed in necrotic pancreatitis within 4 weeks of onset and contain variable amounts of fluid and necrosis. Necrosis can involve the pancreatic parenchyma or/and peripancreatic tissues (*Figure 5*). Collections that are in the pancreatic parenchyma and around the necrotic pancreas are still termed ANCs (2,3). ANCs have no capsules. ANCs show mixed signals on T1WI and T2WI. On T2WIs, there can be flocculent low signal necrosis areas within the collections, which are not enhanced (*Figure 5*). It is very difficult to identify the ANCs and APFCs in the first week of onset, and the CT evaluation of ANC is subjective and cannot objectively judge whether there is necrotic tissue in the heterogeneous collections in peripancreatic tissue. Although MRI is more sensitive in differentiating different components in the accumulation of peripancreatic tissue, when the necrotic tissue fragments are small, MRI cannot determine whether the liquid that appears with high signal on the liquid-suppressed T2WI is entirely inflammatory fluid without doping necrotic cells, and thus, MRI still cannot accurately determine ANCs (35) (*Figure 8*). ANCs require drainage with a catheter to prevent infection and possible sepsis (36).

Walled-off necrosis (WON)

A severe and persistent clinical course or delayed deterioration of disease in necrotizing pancreatitis suggests the presence of WON, which is the maturation stage of ANCs (3). The most characteristic manifestation of WON on MRI is that the encapsulated effusion contains non-liquid substances, which are flocculent, with banded tissue fragments that are free and floating, and there is no enhanced signal on the enhanced scans. This non-liquid substance is the residue of the pancreas and extrapancreatic tissue, which are difficult to distinguish with CT while can

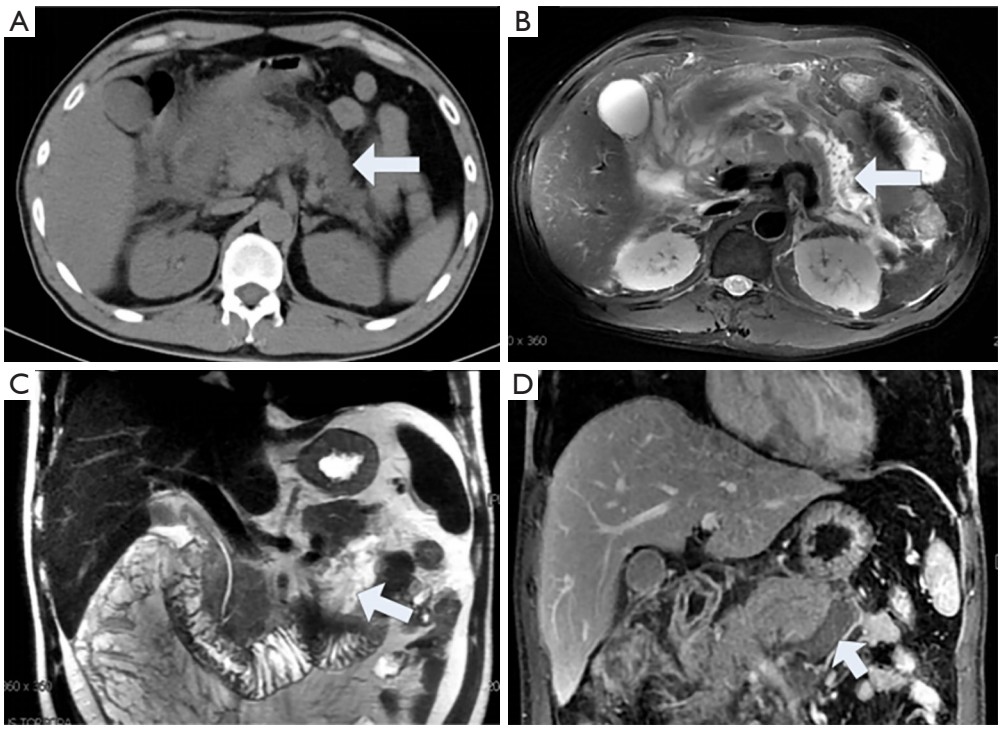

Figure 8 A 39-year-old female with peripancreatic necrosis underwent CT and MRI within 7–10 days after onset. On plain CT scans (A), the peripancreatic collections are uniform (arrows), indicating acute peripancreatic fluid collections. However, on the MRI fs-T2WI (B) and COR-T2WI (C), there are shadows within the patchy hypointense areas of the collections around the body and tail of the pancreas (arrow), and these areas show no enhancement after an administration of contrast agent (D, arrow), indicating acute peripancreatic fluid collections. MRI, MR imaging.

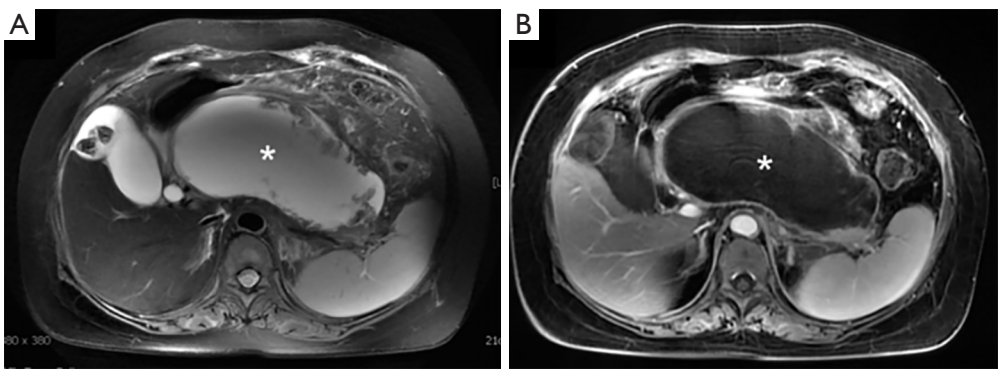

Figure 9 WON of the pancreatic head, body, and a portion of the tail in a 43-year-old woman with necrotizing pancreatitis 24 days after onset. The axial T2-weighted, fat-suppressed image (A) and enhanced T1-weighted image (B) reveals a marginal cystic envelope with uneven thickness and patchy necrosis with no enhancement of the necrotic tissue (asterisk). WON, walled-off necrosis.

be clearly visualized with MRI (5) (*Figure 9*). Patients with WON are commonly complicated with infection. Studies show that the mortality of patients with WON co-infection is higher than that of non-infected patients (37). Patients with sudden fevers, leukocytosis, sepsis or bubble signs, and gas-liquid interface on MRI suggest an infection (11) (*Figure 10*). When there is no characteristic imaging sign of infection but there is a clinical suspicion of co-

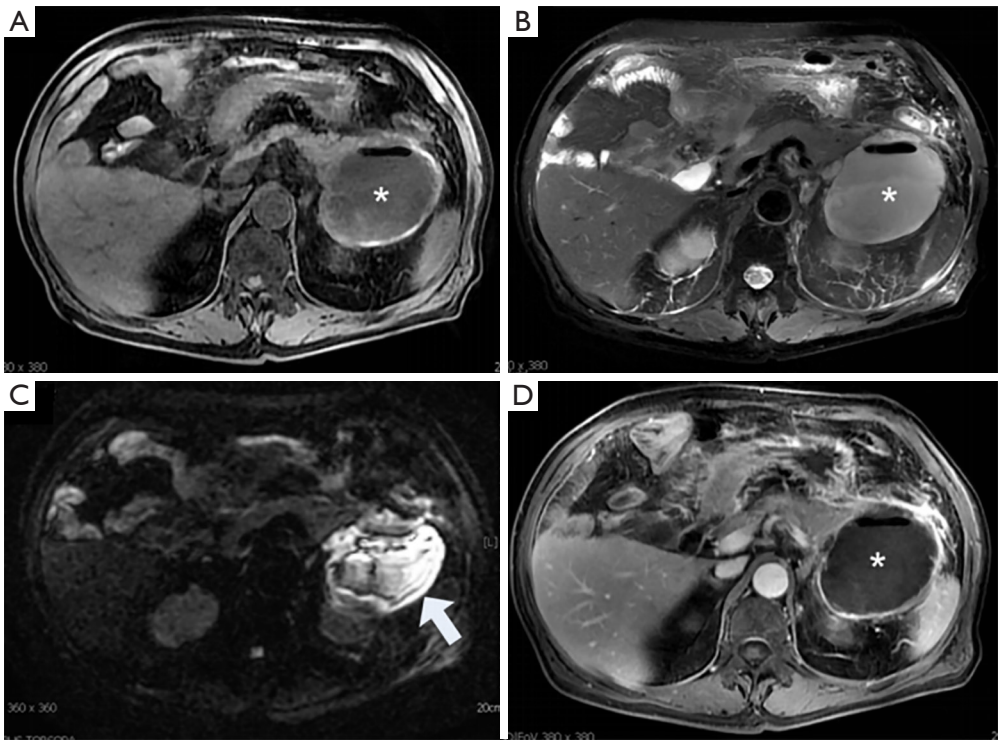

Figure 10 A 67-year-old female with acute pancreatitis 7 weeks after onset. Infected WON in the tail of the pancreas was confirmed by fine needle aspiration biopsy. The T1WI (A) and T2WI (B) show an encapsulated, ill-defined collection (asterisk) in the tail of the pancreas with wall thickening and gas within the collection. With DWI, the collection shows hyperintensity (C, arrow) (C), and the wall was enhanced after contrast injection (D). WON, walled-off necrosis; DWI, diffusion-weighted imaging.

infection, a fine needle aspiration biopsy is necessary for a definite diagnosis (11). Surgical treatment can be used only when WON is clinically diagnosed with an infection (infective necrosis) or to treat the increasing volume of WON or complications with bleeding, such as secondary pseudoaneurysms that cause obvious abdominal pain and distention (38).

Other complications

AP can involve the adjacent organs, such as the stomach, intestines, spleen, kidneys, and peripancreatic vessels. Changes of these organs and structures are reactions to inflammatory substances in the early stage. With the stabilization and improvements of the disease, these changes will gradually decrease or disappear. In the late stages, some changes may persist or even aggravate the complications of AP, such as including pseudoaneurysm of the peripancreatic vessels, venous thrombosis, spleen infarction, intestinal fistula.

Disconnected pancreatic duct syndrome (DPDS)

Severe necrotic pancreatitis with pancreatic duct disruption has a prevalence of approximately 10% to 31% (39). Persistent pancreatic fluid overflow causes various local complications, such as pancreatic pseudocysts, pancreatic ascites, pancreatic pleural fistulas, and pseudoaneurysms. ERCP is generally regarded as the gold standard for the diagnosis of pancreatic duct interruption. However, because ERCP is invasive, which is not suitable for acute pancreatitis (especially severe cases), and cannot show the pancreatic duct at the other end of the rupture, the evaluation of the main pancreatic duct interruption by ERCP is incomplete (40). The combination of MRI and MRCP provides a noninvasive method that can not only show the pancreas and peripancreatic changes but can also analyze the proximal and distal ends of the ruptured main pancreatic duct. Therefore, the use of MRI and MRCP is more advantageous to evaluate the main disconnected pancreatic duct syndrome caused by acute necrotizing pancreatitis (41). The visualization ratio of the pancreatic duct on MRCP

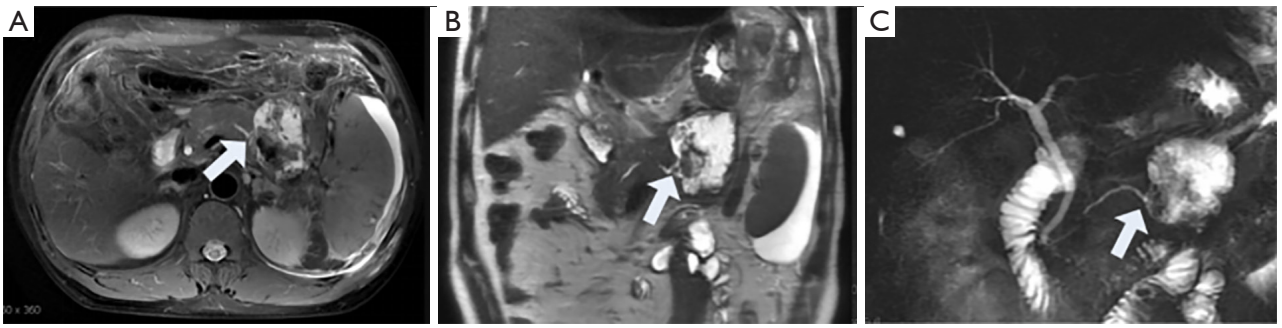

Figure 11 Disconnected pancreatic duct syndrome in a 37-year-old woman with acute necrotizing pancreatitis. The axial T2-weighted image (A) and coronal T2WI (B) obtained 7 weeks after onset show an area of WON with diameter of 6 cm located in the pancreatic body and tail. The pancreatic duct in the pancreatic body is dilated, abruptly cut off, and connected with the area of WON (C, arrow). WON, walled-off necrosis.

is lower in acute pancreatitis than that in normal subjects, but the diameter of the main pancreatic duct is still within the normal range. The visualization of the pancreatic duct usually demands a combination of the coronal and axial SSFES T2WIs, axial FRFSE T2WI and MRCP image (42) (*Figure 11*). Studies have reported that the diagnosis of pancreatic duct disruption should be considered if the following occurs: (I) peripancreatic necrosis area at least 2 cm, and (II) MRCP shows that the main pancreatic duct of the upstream pancreatic tissue travels to the WON area of the intra and/or extrapancreatic tissue and approaches with a right angle into the fluid or necrotic tissue (33). Our previous study showed that as the severity increases, the incidence of pancreatic duct rupture also increases. The development of a disruption and a ruptured pancreatic duct on the MRI scan can be another credible indicator of the severity of acute pancreatitis (43), and the severity determines the clinical treatment method and predicts the short-term and long-term complications.

Hemorrhage and peripheral vascular invasion

Hemorrhage in acute pancreatitis mainly includes gastrointestinal bleeding, intra-abdominal blood accumulation and bleeding in the pseudocysts. The main mechanism is that the tissue-dissolving enzymes released by the pancreas can corrode the wall of the peripancreatic vessels, which causes chemical inflammation of the vascular wall, formation of venous thrombosis, and massive hemorrhage due to the destruction of the blood vessel wall (44). Hemorrhages on CT show a slightly high density (>35 HU). Over time, the density gradually decreases. Compared with CT, MRI is more sensitive to visualizing hemorrhages, which are hyperintense on T1WI during acute phase and have signals that persist longer than on CT images (24) (*Figures 3*,*6*).

Involvement of the peripancreatic vessels, which includes a series of vascular abnormalities, is common in AP and especially in NP; the prevalence is approximately 16.9% (45). The pathogenesis is probably direct exudative damage to the pancreatic vessels. Arteritis and pseudoaneurysm occur in arterial involvement; phlebitis, venous thrombosis, and pancreatic portal hypertension/portal vein cavernous changes occur in venous involvement (46). The main MRI manifestations are loss of normal flow in the lumen, and the signal void on the T1WI and the T2WI is replaced by local hyperintensity. The enhanced scan shows inhomogeneous enhancement and segmental poor enhancement in the affected segments (*Figure 12*). As the acute pancreatitis worsens, the vascular complications become more common. A study showed that for splenic vein thrombosis and mesenteric venous thrombosis, the occurrence of splenic artery and vein inflammation was positively correlated with the MRSI scores.

Gastrointestinal tract involvement

Studies show that approximately 63% of patients with AP have gastrointestinal abnormalities (47). The abnormalities are characterized by gastrointestinal dilatation, mild thickening of the gastrointestinal wall, stratification of the bowel wall during its enhancement on the arterial phase after an intravenous injection of a contrast agent. The thickening of the intestine wall is uniform, which is different from the heterogeneous and eccentric thickening of

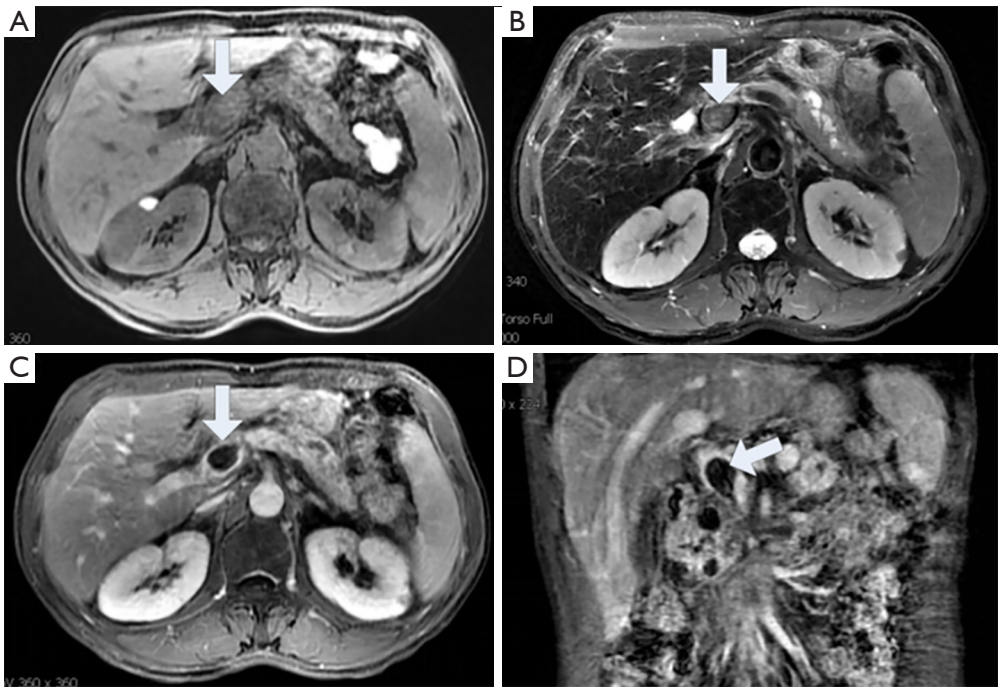

Figure 12 Portal vein thrombosis in a 78-year-old male with acute pancreatitis 4 weeks after onset. There are strips of hyperintense signals on T2WI around the head and neck of the pancreas. The portal vein shows hyperintensity on the axial T1WI (A, arrow) and T2WI (B, arrow) and has filling defect on the delayed phases after enhancement (C,D) (arrow).

neoplastic lesions. The middle wall shows high signals, and the internal and external ring show hypointensity; together, these form the so-called "target sign" on T2WI, and the post-contrast scan also shows stratified enhancement (48) (*Figure 13*). Ji *et al.* (47) hypothesized that the closer to the pancreas, the more serious the abnormalities in the tissues and organs; they found that the colon and duodenum were the most vulnerable organs. Gastrointestinal tract involvement plays an important role in assessing the severity and monitoring the efficacy of treatment (47). If attention is not paid to intestinal injuries in the early stage of the disease, with the progression of the disease a large number of toxins and inflammatory mediators can be released, and bacteria can even be translocated. Once "intestinal infection" occurs, there may be fatal consequences. Therefore, clinicians should actively observe changes in the gastrointestinal tract during the course of disease with MRI, and adjust the therapy according to the recovery of the gastrointestinal tract.

Mesenteric and retroperitoneal fascia space changes

AP can involve the mesentery and manifest as the spread of inflammation from the root of the mesentery to the mesentery along with transverse-mesocolon edema and effusions (49). Mesenteric edema and effusion are characterized by stripes and small slices of hyperintensity on T2WI (*Figure 14*). The changes in the blood vessels include wall thickening, rough texture, enlarged lumen and collateral circulation. Studies have shown that small intestinal mesentery and transverse mesocolic invasion are common in AP, which are positively correlated with a high MRSI score, and both of these are aggravated with increases in the severity of AP (50).

The pancreas is located in the retroperitoneal anterior pararenal space, so the inflammatory pancreatic juice of AP can penetrate into the anterior pararenal space and can also spread into the perirenal space, the posterior pararenal space, the abdominal cavity, and even the subphrenic and pelvic cavities (51,52). In addition to the above-mentioned retroperitoneal space, Molmenti *et al.* (53) proposed a new anatomical concept for the structure of the retroperitoneal space, the interfascial plane. The retroperitoneal space is composed of several discontinuous layers and potential interfascial planes. Interfascial plane involvement is a common MRI manifestation of acute pancreatitis on both sides, but the rate of left side involvement is significantly

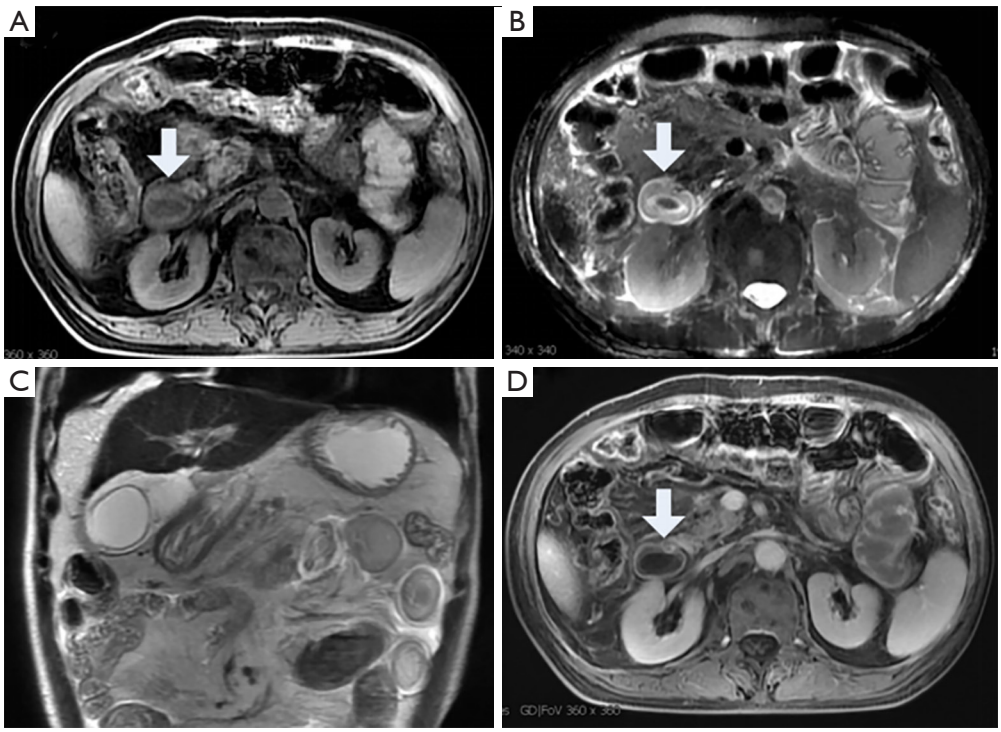

Figure 13 A 44-year-old female with acute pancreatitis. An MRI was performed on the 7th day after the onset of pancreatitis. The duodenum shows thickening on the T1WI (A, arrow). The "target sign" (arrow) of intestinal wall thickening can be seen on the T2WI (B). The axial (B) and coronal T2WI (C) show dilatation of the duodenum and small intestine, thickening of the bowel wall and edema (arrows). After contrast administration, the signal of the bowel wall was enhanced evenly (D, arrow). MRI, MR imaging.

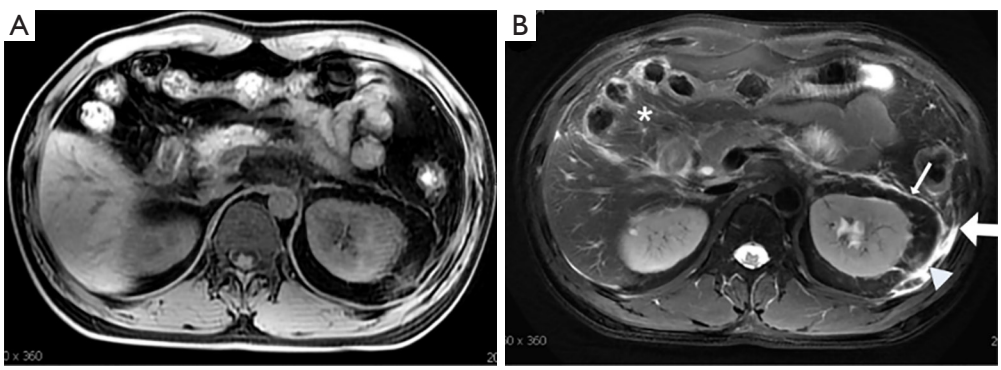

Figure 14 A 39-year-old male with acute edematous pancreatitis. Inflammation involved the transverse mesocolon (asterisk) and left retroperitoneal interfascial plane, left retromesenteric plane (thin arrow), the lateroconal plane (thick arrow) and retrorenal plane (arrowhead). The inflammation appeared hypointense on the T1WI (A) and hyperintense on the fat-suppressed T2WI (B).

higher than that of the right side (51). MRI can accurately depict the extent of interfascial plane involvement in pancreatitis of different severities and infer the route of inflammation transmission through the sequence of fascial involvement (*Figure 14*) (50,51).

Complications of the liver and biliary tract system

AP leads to abnormal peripancreatic microcirculation, systemic inflammatory response syndrome (SIRS) and bile duct obstructions, which may lead to abnormal liver changes (54). Routine MRI findings of AP with liver

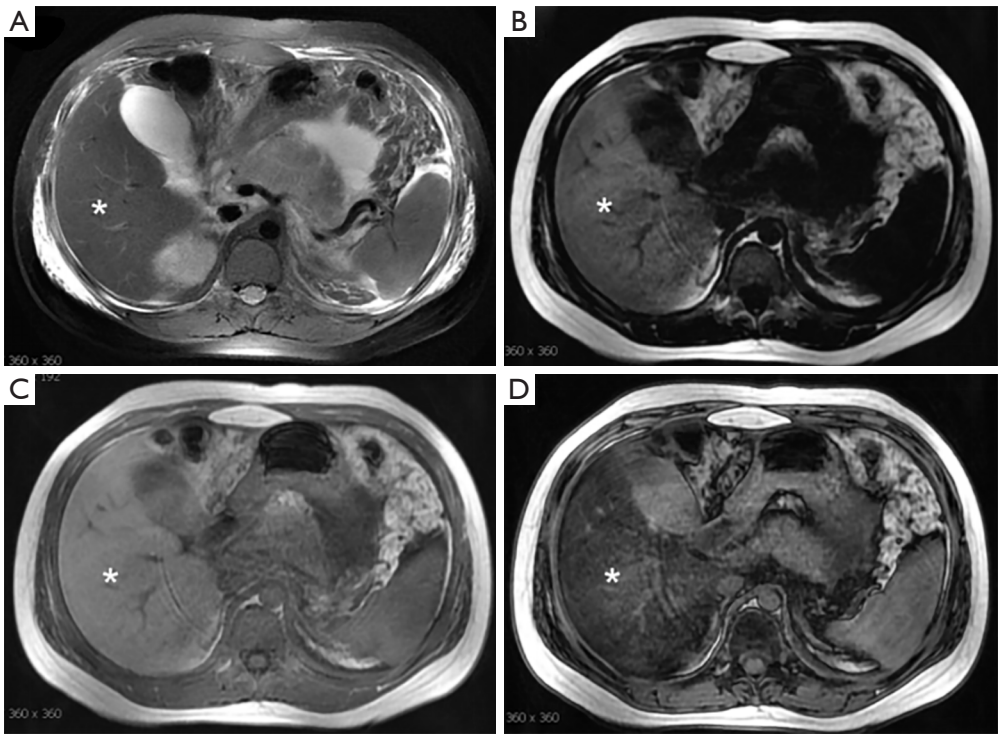

Figure 15 A 44-year-old male patient with acute pancreatitis and fatty liver. The MRI T2WI shows fluid collections around the pancreas (A). On the LAVA-Flex fat-only phase image (B), the liver intensity is high (asterisk). In addition, the intensity of the liver parenchyma on the in-phase image (C) is significantly higher than that on the out of phase image (asterisks) (D). MRI, MR imaging.

abnormalities include hepatic steatosis, transient hepatic perfusion abnormalities, periportal lymphatic stasis and perihepatic effusion (*Figure 15*). These abnormalities can reflect different degrees of liver injury, and the incidence of hepatic steatosis is the highest out of the incidences of all abnormalities (55). Liver injuries caused by AP can not only aggravate the severity of pancreatitis but can also develop into liver failure, which results in the rapid death of the patient. The fatty liver finding on MRI is positively correlated with the triglyceride level, and fatty liver can also be alleviated with the improvement of pancreatitis (56). As the lower part of the common bile duct travels in the head of the pancreas, most of the diseases of the head of the pancreas can cause the narrowing and obstruction of the lumen of the common bile duct. Early decompression of the biliary tract system can prevent progression of the disease and avoid recurrent pancreatitis (57).

Others

AP can also involve the urinary system, respiratory system, bone and skin. Acute renal failure is an early complication of severe AP and has a high mortality rate. The incidence of organ failure in other systems increases significantly after acute renal failure (58). There is more perirenal involvement in AP on MRI than renal parenchyma involvement, and the morbidity of perirenal involvement has been noted to be positively correlated with a high MRSI score (51). MRI findings of abnormal changes in the renal and perirenal space include abnormal perfusion of the renal parenchyma, swelling of perirenal fat, fluid collections in the perirenal space, renal venous thrombosis, etc. (51). Respiratory complications are very common in patients with acute pancreatitis. Hypoxemia can be the only manifestation in mild cases, and respiratory failure can occur in severe cases (59). Studies have shown that abdominal hypertension and systemic inflammatory response syndrome associated with acute pancreatitis are the main causes of early lung injury. Abdominal hypertension can lead to increased intrathoracic pressure, pleural effusion, atelectasis, pneumonia, airway trapping and other pulmonary complications; all of these can then lead to abnormal gas exchange, which is one

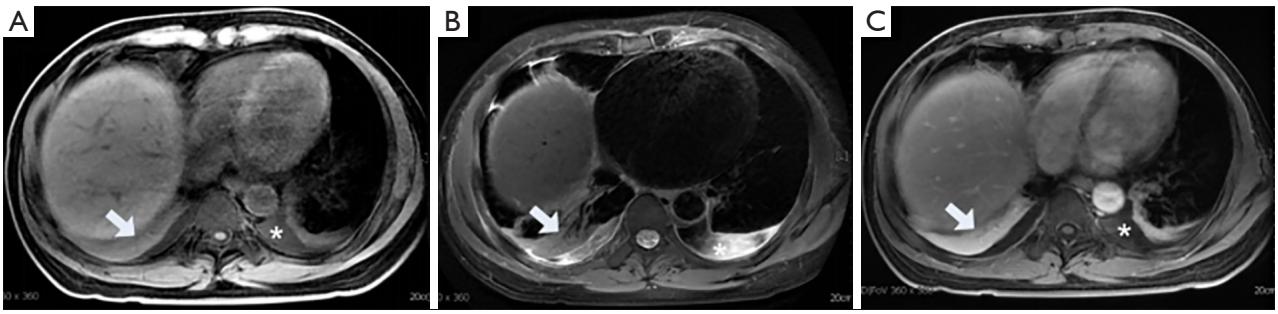

Figure 16 A 72-year-old male with acute pancreatitis complicated with pneumonia and pleural effusion underwent an MRI examination 5 days after onset. The T1WI (A) and T2WI (B) show iso-intense flakes (representing pneumonia) in the bilateral lower lung (arrows), which were enhanced after an administration of contrast agent (C, arrow). On the T2WI (B), the bilateral pleural effusion shows hyperintensity (asterisk). MRI, MR imaging.

of the most common causes of early lung injury in acute pancreatitis (60,61). In addition, systemic inflammatory response is also an important mechanism that leads to lung injury in acute pancreatitis. In acute pancreatitis, vasoactive substances and cytokines are released into the blood flow and into the lung tissue, which can lead to endothelial damage and capillary leakage; additionally, a large number of protein-rich exudates are released into the alveolar space, leading to interstitial pulmonary edema (59). MRI shows diffuse pulmonary exudative patches, bilateral lobular consolidation and pleural effusions (62) (*Figure 16*). Injuries of the bone and skin secondary to pancreatitis may be caused by high pancreatic lipase in the blood. On MRI, these injuries can be characterized by inflammation of the joints, focal bone destruction, and abdominal wall edema (*Figure 17*). Furthermore, a few patients may have pancreatic ascites, that is, pancreatic juice that directly or indirectly enters the peritoneal cavity after pancreatic duct rupture. Physicians should treat these injuries as early as possible to reduce mortality (63). In acute pancreatitis, the spleen is also a commonly involved organ; common splenic abnormalities are splenomegaly and splenic infarction. In addition to routine sequences, quantitative analysis of the spleen with IVIM sequences might be useful for visualizing splenic perfusion changes in AP (14).

MRI for the severity of acute pancreatitis

The MRSI derived from the CT severity index (CTSI) is used to assess the severity of AP by combining inflammation around the pancreas and necrosis of the pancreas parenchyma to evaluate the local conditions, and the MRSI is comparable to the CTSI in evaluating the severity of AP (5). It has been shown that the MRSI is superior to the Acute Physiology and Chronic Health Evaluation II (APACHEII) in assessing local complications from pancreatitis but has a limited role in determining systemic complications, where the APACHE II score excels (64).

Certain imaging signs beyond the MRSI scoring system can also indicate the severity of AP. Studies have shown that MRI can display positive correlations between the time of improvement of the gastrointestinal tract abnormalities and the time of hospitalization, the disappearance time of abdominal pain and abdominal distention, and the normal time of amylase recovery and the recovery time of the diet (47). MRI can also show peripancreatic vascular changes, and the severity of vascular involvement is positively correlated with the severity of AP (14). In addition, the incidence of liver and kidney dysfunction, pulmonary inflammation, perirenal space involvement, interfascial plane involvement and abdominal wall edema increases with the severity of AP (50-52). It has been reported that the morphological changes of the main pancreatic duct (flexion degree and curve length) are related to AP occurrence. The greater the flexion of the main pancreatic duct, the greater the possibility of the occurrence of AP. The incidence of pancreatic duct rupture increases with the severity of AP and can be used as another simple auxiliary index to assess and predict the severity of AP (43). In terms of clinical treatment, surgical indications and the timing of the surgery are important for patients who require surgical intervention. MRI can help surgical planning by demonstrating the scope of necrotic tissue, the increase or decrease in the amount of peripancreatic retroperitoneal fluid, and the clear demarcation of the necrotic tissue and

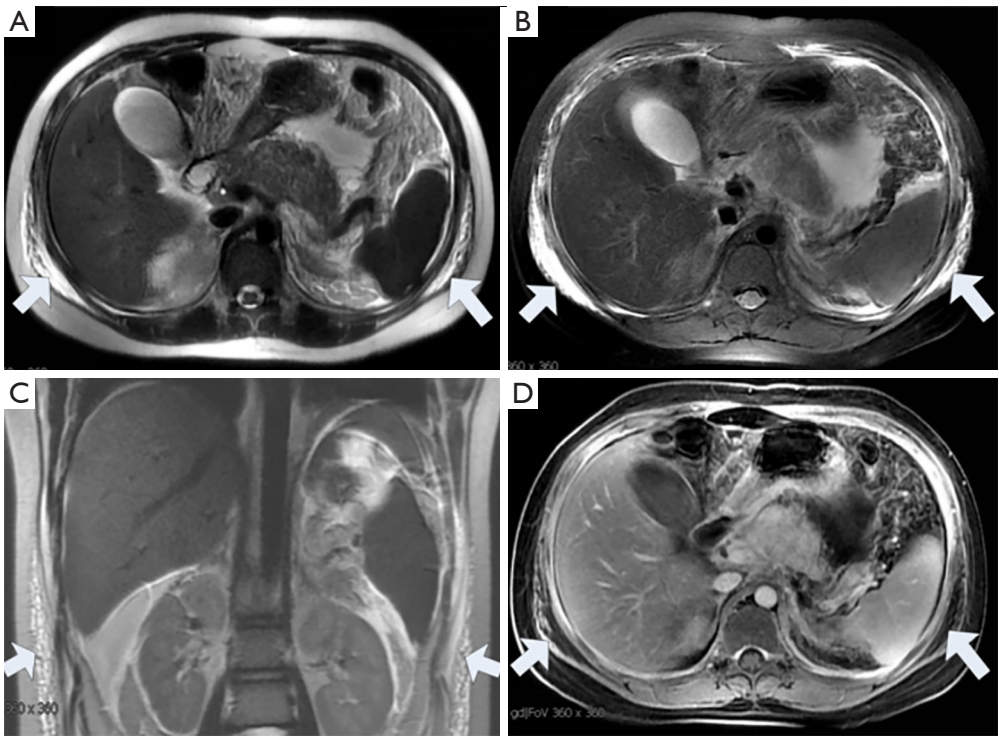

Figure 17 A 38-year-old female with acute pancreatitis. The MRI T2WI (A,C) and fs-T2WI (B) show irregular, patchy, strip-like hyperintense signals, a blurred muscle space and disordered soft tissue structures on both sides of the abdominal wall (arrows). There was no enhancement after contrast injection (D, arrow). MRI, MR imaging.

the normal tissue.

In recent years, DWI, ADC values and intravoxel incoherent motion (IVIM) have been applied in the abdomen, all objectively reflect the movement of water molecules in tissues and their biological behavior (65). During AP, there is the presence of swollen pancreatic acinar cells, interstitial congestion, edema and inflammatory cell infiltration (66). de Freitas Tertulino *et al.* (65) found that the ADC value can help to distinguish mild inflammation from necrosis of the pancreas, which is significant for evaluating the severity of acute pancreatitis. Kovalska *et al.* (66) suggested that microcirculatory disturbances can cause ischemia, reperfusion injury, and even pancreatic tissue damage. The severity of AP actually hinges on the severity of microcirculation disturbance. MR perfusion imaging can reflect the changes in early pancreatic blood flow, which can diagnose pancreatic necrosis in the early stage of disease by quantitatively analyzing pancreatic blood perfusion (67). Furthermore, Hu *et al.* showed that evaluation of pancreatic perfusion with DCE-MRI was helpful in grading AP severity. They found that pancreatic perfusion decreased with the aggravation of AP (68). Sahani *et al.* (69) used animal models to confirm the feasibility and accuracy of computed tomography perfusion in detecting pancreatic necrosis. In the necrotic area, perfusion parameters such as blood flow, blood volume and surface permeability were significantly reduced, but these parameters did not change significantly in the edema area. Moreover, diffusion tensor imaging (DTI) has the capability to detect changes in the degree of diffusion anisotropy and molecular diffusion in AP patients by calculating the FA and ADC values of the pancreas to evaluate the severity of AP (70).

Conclusions

In summary, MRI has advantages in the diagnosis of AP and its complications. The severity of AP can be estimated by combining the morphological changes of the pancreas, peripancreatic tissues and other organs on MR images. The rapid development of functional imaging techniques based on MRI has greatly increased the possibility of radiologists accurately evaluating the severity and progression of acute pancreatitis.

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Footnote

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

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