Usual and unusual forms of pancreatitis: Imaging evaluation with emphasis on MRI

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Purpose

To review the iconographic findings of typical and atypical etiologies of pancreatitis through different imaging techniques, emphasizing in magnetic resonance imaging (MRI) and the key points for the diagnostic approach with it.

Methods and Materials

This is a pictorial review of typical and atypical etiologies of pancreatitis through imaging examples of patients diagnosed and treated in our institution.

Results

INTRODUCTION

Definition:

Pancreatitis is an inflammatory process caused by autodigestion of the gland by pancreatic enzymes. In acute pancreatitis, after the inflammatory process, the gland will often recover without functional or morphological impact. Chronic pancreatitis is, however, a long evolutionary process, sometimes secondary to recurrent attacks of acute pancreatitis, with alteration of the gland structure basically due to fibrosis, which can end up affecting pancreatic functionality.

Etiology:

In acute pancreatitis, the most frequent causes are lithiasis and alcoholism (80-90% of the cases) Fig. 1 on page 19. Atypical causes include: drugs, obstructive tumoral processes, complication after Endoscopic Retrograde Cholangiopancreatography (ERCP), hyperlipidemia and hypercalcemia, abdominal trauma, peptic ulcer, abnormalities of the pancreatic drainage and infections, among others.

In chronic pancreatitis, alcoholism is certainly the most common cause (around 80% in Western countries). Other postulated causes include chronic blockage of the pancreatic duct by neoplastic lesions or stenosis (which can be improved after its clearance) and very
low-protein diets. In certain cases, some of the causes described for acute pancreatitis, if occurred in recurrent episodes, can trigger evolution to chronic pancreatitis. Autoimmune pancreatitis, paraduodenal pancreatitis, tropical pancreatitis and hereditary pancreatitis are also considered atypical forms of presentation for chronic pancreatitis. Imaging examination and exocrine function tests are the ideal combination for diagnosis.

**Epidemiology:**

The incidence of acute pancreatitis in Spain is about 10-40 new cases per 100,000 population. The estimated prevalence of chronic pancreatitis in Europe is 10-30 cases per 100,000.

**Pathology:**

Acute pancreatitis comes as a result of the exudation of fluid with proteolytic enzymes (such as trypsin) in the pancreatic interstitium and the leakage in the surrounding tissue. Thus, histopathologically, in mild cases, pancreatitis is known as edematous or interstitial type; peripancreatic fat necrosis can be present without parenchymal necrosis. In severe cases, there is both necrosis of intrapancreatic and peripancreatic fat and within pancreatic parenchyma, plus hemorrhagic changes.

In chronic pancreatitis there is a recurrent inflammation of the pancreas with irreversible morphologic changes, including injury of acinar cells and ducts. Histopathologically sclerotic and fibrotic changes, stricture and dilatation of the ductal system and atrophy of the parenchyma are found. The injury can be focal, segmental or diffuse. Intraductal calcifications might be present.

**Clinical presentation:**

Clinically, acute pancreatitis presents as moderate or severe abdominal pain in the epigastrium, radiating to the back, with nausea and vomiting; less frequent are fever and abdominal distension. In blood analysis, the elevation of pancreatic enzymes (amylase and lipase) stands out. The main symptom of chronic pancreatitis is chronic pain (80%), often continuous and moderate in intensity, located in epigastrium. As pancreatitis evolves through the years, the pancreatic gland deteriorates and the pain tends to disappear. Other symptoms are related to the progressive damage of pancreatic function (considering advanced disease), such as diarrhea and weight loss, malnutrition and diabetes.
RADIOLOGICAL FINDINGS IN ACUTE PANCREATITIS

Imaging in acute pancreatitis is aimed at:

1. Diagnose pathology.
2. Settle severity.
3. Determine the cause.
4. Assess and manage possible complications.

CT FINDINGS

The computed tomography (CT) is the initial choice in the imaging diagnosis and staging of acute pancreatitis. Ultrasonography is routinely performed in these patients, in order to determine a possible biliary origin. **CT exams within the first 72 hours have limited value** as may underestimate the presence and extent of necrosis.

Acute pancreatitis can manifest itself in a CT scan (in increasing order of severity and according to the classical criteria) as:

1) a normal pancreas

2) a focal or diffuse increase in the gland size

3) pancreatic and peripancreatic inflammatory changes

4) an intra or extrapancreatic single fluid collection

5) several intra or extrapancreatic collections and/or retroperitoneal gas.

We refer to a collection when we find ill-defined and not encapsulated areas of attenuation similar to that of fluid, distinguishable from the pseudocysts, which are encapsulated and develop out of 4 weeks.
After intravenous contrast material administration, the presence of focal or diffuse areas of lack of enhancement is considered diagnostic of necrosis, whose extension is crucial for staging the gravity of the episode. The presence and extent of necrosis are the most important indicators for assessing the severity, although there may be local or systemic complications in patients with acute pancreatitis without necrosis. Fig. 2 on page 19

The rate of local complications of non-necrotizing pancreatitis with collections is around 12% (such as pseudocysts, abdominal bleeding, infection of exudates, intestinal perforation, venous thrombosis, etc). It must be remembered that these complications are more common in patients with severe necrotizing pancreatitis.

**Classification of the severity of pancreatitis CT (Balthazar)**

Balthazar determined a severity scoring in acute pancreatitis, according to findings in CT, which combines the grade of pancreatitis with pancreatic necrosis extension. Mild pancreatitis (80%) includes the interstitial types (without collections; less than 1% mortality) and the exudative type (presence of collections, mortality less than 8%). When there is necrosis (20%) it is considered severe pancreatitis (with a mortality rate of 10-23%). Fig. 3 on page 20

Table 1 on page 38 : Score of severity in acute pancreatitis according to findings by CT

**MR FINDINGS**

MR is, at least, as sensitive as CT in pancreatitis imaging, and may have an additional value in some patients.

In cases of mild pancreatitis, pancreatic resonance signal is similar to the normal one: hyperintense on pre-contrast T1 weighted images and with a peak of enhancement in the pancreatic phase (40 seconds after administration of contrast material); it may show focal or diffuse enlargement that can be subtle. Fig. 4 on page 20

Fluid or peripancreatic exudate are shown as hypointense collections on T1WI (pre and post-administration of contrast material) and hyperintense on T2WI. T2WI single-shot sequences are the most sensitive to show mild peripancreatic exudates. Therefore the RM is very sensitive for the detection of subtle changes in the context of acute pancreatitis, mainly when the pancreas is morphologically normal (good detection of mild peripancreatic inflammatory changes). Literature has shown that the CT scan may
be normal in some cases of patients with pancreatitis, so **MR can play a role in the evaluation of patients with suspected acute pancreatitis and negative CT.**

As the extension of the pancreatitis becomes more severe, the signal of the pancreatic gland becomes more heterogeneous on T1WI pre- and post-contrast sequences, with hypoenhancing areas in the pancreatic phase. Areas of pancreatic necrosis show hypointensity on T1-weighted gradient-echo sequences; when liquefaction occurs, we can appreciate areas of hyperintensity on T2WI sequences. **Fig. 5** on page 21

The dynamic post-contrast study in MRI can detect and quantify pancreatic necrosis similarly to the CT (which is an important factor in the patient prognosis).

MR also detects other complications such as bleeding collections (with high signal on T1-weigthed images, being MRI more sensitive than CT for detection in this case), pseudocysts and abscess formation. Correlation between the extent of high signal tissue on T1WI (correlated with hemorrhagic changes) and severity of pancreatitis has been demonstrated. **Fig. 6** on page 22

**Technical peculiarities in the evaluation of the pancreatitis through RM**

How are the different MRI sequences going to help us in the assessment of acute pancreatitis?

**Fat-suppressed T2-weighted** images show edema in mild pancreatitis and pancreatic or peripancreatic collections.

**T1-weighted in-phase** images can demonstrate peripancreatic exudates.

**Fat-suppressed T1-weighted gradient-echo sequences** are very useful to show hemorrhage and necrosis associated with acute pancreatitis.

**3D fat-suppressed dynamic contrast-enhanced sequences** are useful for assessing necrosis (MR contrast material is less damaging). It is remarkable that the administration of gadolinium may be restricted if there is renal failure, but the MRI without intravenous contrast medium has shown similar results to CT in detecting necrosis.

MR is also superior to CT when assessing the main pancreatic duct (MPD) and its secondary branches (this is important in order to rule out ductal disruption and determine possible pancreatic drainage abnormalities that may be the cause of pancreatitis) using
cholangiographic sequences *(thin-slab 3D with respiratory triggering o thick-slab 2D single-shot sequences)*.

MR is therefore very useful in the assessment of patients with acute pancreatitis, due to its capability to use sequences that can show tissue edema in the mild form and necrosis and hemorrhage in the severe form, as well as accurately characterize collections and assessment of the MPD.

Key findings of acute pancreatitis in MR:

-Mild acute pancreatitis (80%):

(a) normal or enlarged pancreas

(b) parenchyma edema (hyperintense on T2WI, iso or slightly hypointense on T1)

(c) hyperintense peripancreatic fluid on T2WI

(d) peripancreatic exudates on T1-weighted in-phase images

-Severe acute pancreatitis (20%):

(a) quantify necrosis (prognostic factor) as areas with lack of contrast enhancement

(b) pancreatic necrosis as hypointense areas on T1WI, and hyperintense on T2WI when there is liquefaction

(c) increased peripancreatic signal on fat-supressed T1WI, suggestive of hemorrhagic fat necrosis that is associated with increased morbidity and mortality in patients with acute pancreatitis.

**Value of MRI versus CT in acute pancreatitis:**

In the diagnosis and quantification of severity in acute pancreatitis, MRI is comparable to CT. Some advantages of th MRI over CT are:
Absence of radiation (important if there are multiple controls for monitoring) and the potential nephrotoxicity of iodinated contrast agents (though the risk of nephrogenic systemic fibrosis with certain Gadolinium-based contrast media should be taken into account) as well as the potential deterioration of the acute pancreatitis secondary to iodinated contrast agents.

Possibility of cholangiographic sequences, which provide additional information to determine the cause (biliary calculi or duct anomalies) and assessment of the integrity of the pancreatic duct (which has also a prognostic factor).

More precise distinction between exudates and hemorrhagic necrosis of the peripancreatic fatty tissue and the possibility to determine pancreatic hemorrhage; CT can overestimate the degree of severity of the pancreatitis, as it is not possible to differentiate so accurately between peripancreatic fluid collections and hemorrhagic necrosis of the peripancreatic fat.

However, given the higher cost, time of exploration and sensitivity to motion artifacts, MR should be considered globally as a second-line diagnostic modality in patients with acute pancreatitis after the CT.

RADIOLOGICAL FINDINGS IN CHRONIC PANCREATITIS

CT findings

The classic findings of chronic pancreatitis in CT are:

- dilatation of the MPD

- pancreatic parenchyma atrophy

- calcifications

Pseudocysts, areas of pancreatic enlargement, biliary tract dilatation, alteration of peripancreatic fat density or peritoneal fascias can be found less frequently. Fig. 7 on page 23

CT scan may be normal in 7% of the cases.
Calcifications are a late finding, which follows the development of fibrosis.

It should be kept in mind that CT is not sensitive to detect early changes of fibrosis.

**MR findings**

MR can detect the parenchymal and ductal changes of chronic pancreatitis in a more earlier stage than other non-invasive imaging techniques, such as CT.

The findings include:

(a) dilatation of the MPD and its branches, secondary to the traction on the ducts by periductal fibrosis;

(b) focal or diffuse decrease in size of the gland by the atrophy of acinar cells;

(c) decrease of pancreatic signal on fat-suppressed T1WI due to the loss of protein content in the pancreatic acinar cells;

(d) changes in the normal pattern of enhancement, being heterogeneous and decreased in capillary phase and having progressive and delayed enchancement, reflecting fibrous tissue.

**Changes in focal chronic pancreatitis are difficult to differentiate from adenocarcinoma**, as they present a similar semiology: focal enlargement, obstruction with dilatation of the MPD and the common bile duct, atrophy of the proximal gland and even obliteration of the fat around the superior mesenteric artery. Fig. 8 on page 24, Fig. 9 on page 25.

**MR can better distinguish these two entities**, although they share some signal characteristics on fat-supressed T1WI (slightly low signal intensity) and T2WI (mild high signal intensity).

However, in focal pancreatitis, there is heterogeneous initial enhancement in capillary phase, with areas of absence of signal (cysts or calcifications), without demonstrating a well-defined mass; there is often preservation of the normal lobular architecture; and it
is usually associated with diffuse hypointensity of the whole pancreas on fat-suppressed T1WI, including the enlarged area.

The detection of a well-defined and circumscribed mass is more frequent in pancreatic carcinoma, which shows irregular, heterogeneous and reduced enhancement of the remaining pancreas in the capillary phase.

In rare cases, when chronic pancreatitis affects only the swollen portion (no inflammatory changes in the rest of pancreas) or when the swelling is so destructive that the typical underlying stromal pattern is lost, the diagnosis cannot be made by MR and requires histological diagnosis.

Also, MR accurately detects changes by outbreaks of aggravation in chronic pancreatitis. Fig. 10 on page 26

PANCREATITIS OF UNUSUAL OR ATYPICAL ETIOLOGY

Classically, pancreatitis of atypical cause (non-alcoholic, not biliary) accounts for 20-30% of the acute pancreatitis, and is generally considered a disease with better prognosis than the typical one, although with a tendency to recurrence.

Autoimmune pancreatitis

It is exclusively a type of chronic pancreatitis. It is estimated that 30% of chronic pancreatitis are idiopathic. Within them, there is a subgroup associated with autoimmune pathology (such as Sjögren's syndrome, inflammatory bowel disease, primary biliary cirrhosis and sclerosing cholangitis), whose pathology shows chronic inflammation, with lymphoplasmocitary infiltration and periductal fibrosis, leading to obliteration and destruction of ducts. This form of pancreatitis, known as autoimmune pancreatitis or lymphoplasmacytic sclerosing pancreatitis, corresponds to 2-11% cases of chronic pancreatitis.

Clinically, it may present with abdominal pain, jaundice, weight loss and diabetes; patients do not show typical acute pain of pancreatitis. It is more common in men, above 50-years-old, and its main differential diagnosis should be with neoplastic pathology. Characteristic features are elevation of IgG4 values in serum and the good response to corticosteroid therapy.
At histopathological examination there is focal or diffuse inflammation with lymphoplasmocitary component, and diffuse sclerosis and fibrosis in advanced stages, resulting in mass-like appearance in the pancreas, which can lead to misdiagnosis of carcinoma (even more by its clinical behavior and the occasional increase in CEA and CA19.9).

Diffuse enlargement of the pancreas, with loss of lobular architecture may be seen on CT. Parenchyma is iso or hypodense, with a narrow or not dilated duct. Other findings include a hypodense peripheral rim with delayed enhancement, blurring of the peripancreatic fat planes, involution of the pancreatic tail and presence of locoregional lymphadenopathy. Calcifications, pseudocysts and vascular entrapment are rare. There are forms of focal involvement in the mass-like pancreatic type, more frequent in the pancreatic head, that can make it very difficult to differentiate from adenocarcinoma.

Findings in MR include pancreatic enlargement, with moderate hypointensity on T1WI and hyperintensity on T2WI; delayed uptake of contrast material; hypointense on T2WI pseudocapsule/peripheral rim around affected parenchyma with decreased and delayed enhancement; Neither pancreatic atrophy nor peripancreatic exudates are appreciated. On cholangiographic sequences, we can find diffuse, segmental or focal narrowing of the MPD, mild stenosis of intrapancreatic common bile duct, with proximal dilatation. Fig. 11 on page 27, Fig. 12 on page 28.

Extrapancreatic manifestations, such as renal impairment or retroperitoneal fibrosis can coexist.

The differential diagnosis with cancer should be taken into account in its focal mass-forming presentation (vascular entrapment, abrupt stricture of the MPD or significant atrophy of the proximal pancreas, will favor a neoplastic process) and with lymphoma or mild acute pancreatitis in its diffuse presentation.

The increase of IgG4 or specific antibodies are required to confirm the diagnosis.

The histological diagnosis with pancreatic biopsy should be done if the values of laboratory are indeterminate or there is no complete resolution after the treatment with steroids.

Paraduodenal pancreatitis
Also known as **groove pancreatitis**, it is also an exclusive form of chronic pancreatitis, which can simulate a pancreatic carcinoma. There are two forms: one segmental which affects the pancreatic head and develops scar tissue with fibrosis in the groove; and a pure one, affecting only the region of the groove. Its clinical presentation is related to biliary and duodenal obstruction.

Histopahotologically it is characterized by the presence of **fibrotic tissue in the pancreaticoduodenal groove**, respecting or variably affecting the pancreatic head. **Cystic dystrophy of the duodenal wall and groove pancreatitis could be part of different entities of the same spectrum of paraduodenal pancreatitis**, as they have common findings: dilated ducts and cysts on duodenal wall, Brunner glands hyperplasia and hamartomatous tissue in the pancreatic groove.

In CT, notable findings are the presence of **soft-tissue density in the pancreaticoduodenal groove, with delayed enhancement** and small cystic lesions in the medial wall of the duodenum.

**MR is more accurate to characterize paraduodenal pancreatitis**, identifying hypointense on T1WI and iso or slightly hyperintense on T2WI tissue in the pancreaticoduodenal groove, which can be hyperenhancing in delayed phase. The segmental type shows a mass-like appearance in the pancreatic head corresponding to changes of chronic pancreatitis. Duodenal wall thickening can be seen; the presence of **mural cysts in the 2nd duodenal** portion favors the diagnosis of cystic dystrophy of the duodenal wall, which represents a form of paraduodenal pancreatitis. **Fig. 13** on page 29, **Fig. 14** on page 29, **Fig. 15** on page 30.

The differential diagnosis of the segmental type should be carried out with adenocarcinoma, especially in the scirrhous variant with late enhancement. MR cholangiopancreatography may show mild stenosis of the intrapancreatic common bile duct, instead of the abrupt stenosis seen in carcinoma.

The differential diagnosis of the pure form, should be held with entities such as duodenal or periampullar groove carcinoma (visualization of cysts in the mass or duodenal wall goes in favor of the groove pancreatitis and against carcinoma), neuroendocrine tumor of the groove (easy to differentiate, as this would be hipervascular and hyperintense on T2WI) and acute pancreatitis with inflammatory changes in the groove (which would present rapid changes in its findings and greater hyperintensity on T2WI than in paraduodenal pancreatitis).

**Tropical pancreatitis**
It is a form of **chronic relapsing juvenile pancreatitis**, associated with malnutrition. It is more frequent in tropical countries and has a **rapidly progressive course to severe pancreatitis, large intraductal lithiasis and increased risk of adenocarcinoma**. Clinically, it presents with abdominal pain, weight loss and diabetes.

Imaging examination shows multiple lithiasis in dilated pancreatic ducts (unlike the small calculi of chronic pancreatitis of alcoholic etiology) and in some cases atrophy of the pancreatic parenchyma can be seen.

**Hereditary pancreatitis**

It corresponds to less than 1% of the causes of chronic and recurrent pancreatitis. It is characterized by recurrent episodes of acute pancreatitis from childhood; **in adolescence, there are changes of chronic pancreatitis with parenchimal and ductal calcifications**, destruction of the ducts and endocrine/exocrine insufficiency. In most cases genetic mutations have been demonstrated.

**Imaging studies show similarities to tropical pancreatitis** (atrophy, calcifications and calculi). The diagnosis is suspected in patients with at least two episodes of acute pancreatitis without a triggering factor, in idiopathic chronic pancreatitis, family history of pancreatitis and children with episodes of severe idiopathic pancreatitis. There is significant increase in the risk of adenocarcinoma (which make necessary to start a screening with endoscopic ultrasound at age 30. The role of imaging study is to exclude underlying structural causes of pancreatitis.

**Pancreatitis in ectopic/heterotopic tissue**

Heterotopic pancreas consists in pancreatic tissue with its own ductal system in anomalous situation and without contact with the normal pancreas. Its location is more frequent at **duodenum, stomach (prepyloric region along the greater curvature) and jejunum**, especially at the submucosal layer.

Clinically, it can manifest as **abdominal pain simulating a peptic ulcer**, abdominal fullness and melena; there may even be biliary or intestinal obstruction. Pancreatitis tends to be subclinical (detected in histopathology examination).
In CT we may find nonspecific inflammatory extrapancreatic changes, with gastric or intestinal wall thickening, where a nodule with similar enhancement to the pancreas can be seen. Cystic dystrophy of the duodenal wall is related to pancreatic heterotopia in duodenum.

MR can identify ectopic pancreatic tissue by its characteristic signal, similar to the native pancreas. Adjacent inflammatory changes may be seen. Cholangiographic images show ectopic ducts in the ectopic tissue, being pathognomonic of heterotopic pancreas.

The differential diagnosis arises with submucosal lesions of gastrointestinal tract (such as GIST or carcinoid tumor) and inflammatory processes such as diverticulitis. If the patient is asymptomatic and the diagnosis is clear, the treatment is conservative. But if it is symptomatic or a tumor cannot be ruled out, resection is indicated.

**Associated with pancreas divisum**

It might develop either acute or chronic pancreatitis.

It is the most common congenital abnormality of the pancreatic ductal system (4-10% of population), usually asymptomatic. It consists in a failure in the fusion of the ventral and dorsal ducts. Pancreas divisum should be suspected in young or middle-aged patients without calculi or alcohol abuse, with recurrent acute pancreatitis or chronic relapsing pancreatitis.

There is a relative obstruction of the flow of pancreatic juice by the smaller dimensions of the minor papilla, where the dorsal duct drains (Santorini). There may also be pancreatitis caused by another variant consisting of small communication of the two ducts (dominant dorsal duct syndrome).

CT has a high sensitivity and specificity in detecting pancreatic divisum, although MR cholangiopancreatography is even more precise in its diagnosis. Heavily T2-weighted sequences have a very high sensitivity and specificity.

Associated findings of pancreatitis might be seen: enlarged pancreas, peripancreatic inflammatory changes, areas of necrosis and pseudocysts, as well as changes of chronic pancreatitis with atrophy and dilatation of the dorsal duct. Occasionally, identification of the ventral duct is not possible, but when we see the MPD with anterior course to the common bile duct before draining into the duodenum, it is highly suspicious of
pancreas divisum. Secretin stimulation tests may improve the visualization of the ductal anatomy. Fig. 16 on page 31.

Management is aimed at relieving functional obstruction of the minor papilla (sphincterotomy).

**Atypical obstructive causes**

Pancreatic tumors (intraductal papillary mucinous tumor, adenocarcinoma, metastasis) or duodenal lesions (duplication cysts, lipoma, diverticula, ampuloma) can manifest as recurrent acute or acute pancreatitis.

**Adenocarcinoma** may coexist with with chronic pancreatitis, but this combination is rare with acute pancreatitis. It is usually caused by obstruction, but there is also a non-obstructive theory (release of enzymes by the tumor). In these cases, the diagnosis of carcinoma is delayed by inflammatory changes which mask the tumor. Then, the lesion may only be detected when the swelling has decreased. Fig. 17 on page 32.

Typical imaging findings include significant dilatation of the MPD with associated changes of pancreatitis, disproportion of head/body size, and peripancreatic/upper abdomen lymphadenopathies. There may be metastases and vascular impairment.

**Duodenal diverticula** have been related with pancreatitis as they can cause sphincter of Oddi dysfunction or biliary stasis secondary to distension of the diverticulum, leading to sludge formation and compression of the pancreatic duct. The diagnosis is easily made by CT or MRI, appreciating a saccular image in the 2nd or 3rd duodenal portion, with fluid content or air-fluid level, which connects with the duodenal lumen.

**The sphincter of Oddi dysfunction**

It can be caused by papillary stenosis or sphincter dyskinesia. Papillary stenosis is a structural defect, caused by chronic inflammation. Dyskinesia of the sphincter of Oddi is a functional disorder. Both conditions can cause non-obstructive dilatation at the level of the papilla. CT and MR are useful to rule out neoplastic obstructive causes. Secretin stimulation studies are useful in the diagnosis.

**Post-Retrograde Cholangiopancreatography**
The incidence of post-ERCP pancreatitis is 2-9%, being most of them mild cases of pancreatitis. The factors acting as triggers of pancreatitis include dysfunction of the sphincter of Oddi, local edema by mechanical trauma or hydrostatic injury by overinjection in the pancreatic duct, among others. Fig. 18 on page 33.

**Traumatic pancreatitis**

Injury of the pancreas by blunt abdominal trauma is uncommon; however, its early diagnosis is important, in order to minimize serious complications such as fistulas, abscesses and bleeding.

The most common cause is the **compression of the pancreas against the vertebral bodies**. It can cause pancreatic contusion, partial laceration and even complete fracture. **The integrity of the pancreatic duct must be assessed** because its injury will be determinant in further complications, and a key factor to decide whether conservative or surgical management is indicated.

**MR is a more useful technique for assessing pancreatic duct** than CT, which will be the initial imaging modality in patients with significant abdominal trauma. CT findings associated with pancreatic injury are lacerations, intrapancreatic hematoma or pancreatic ruptures with surrounding fluid; other indirect signs are increased pancreatic size, blurring of fat planes or fluid in the lesser sac, in the peripancreatic region or between the splenic vein and the pancreas. Fig. 19 on page 34.

**Metabolic pancreatitis**

**Hypertriglyceridemia** and **hypercalcemia** are causes of acute pancreatitis, although the pathogenesis of inflammation is unclear. The hypertriglyceridemia pancreatitis should be suspected when alcoholic or bile cause have been discarded and when the patient is obese and presents poorly controlled diabetes.

**Miscellaneous**

**Drug induced pancreatitis** represents 2-5% of the cases of acute pancreatitis, which can be caused by a direct effect (hypersensitivity reaction) or indirect effects (generation of a toxic metabolite, ischemia or an increase in the viscosity of pancreatic fluid).
**Ischemic pancreatitis** is a rare entity, which may occur in cases of vasculitis, intraoperative hypotension, thromboembolism and hemorrhagic shock. CT findings include focal or diffuse hypoenhancement of the pancreas, with or without increase in size.

**Infectious pancreatitis** (viral, bacterial, fungal or parasitic) are very rare in our environment. It usually occurs as acute pancreatitis.

**COMPLICATIONS OF PANCREATITIS**

Pancreatic and peripancreatic collections are divided according to the time elapsed between the beginning of pancreatitis and its appearance. Fig. 20 on page 35, Fig. 21 on page 35.

Thus, if they appear in **less than 4 weeks**, they are classified as follows:

(a) acute peripancretic collection;
(b) post-necrotic peripancreatic collection;
(c) sterile necrosis;
(d) infected necrosis.

**After than 4 weeks**, we can find:

(a) pseudocyst (well confined, with a variable in thickness wall);
(b) abscess;
(c) pancreatic necrosis without wall or walled-off necrosis (irregular, large, with paracolic extension and deformity or discontinuity of pancreas).
Acute collections in MR will be hypointense on T1WI and homogeneously hyperintense on T2WI if there is serous content. If there are bleeding changes, they will be hyperintense on T1WI (more evident with fat-suppressed sequences).

**Simple pseudocysts are hypointense on T1WI and homogeneously hyperintense on T2WI.** Its wall enhances slightly in early phases and progressively increases in subsequent phases due to its fibrotic nature; multiplanar MR acquisition improves the visualization of its relationship with surrounding organs.

**Complicated pseudocysts are heterogeneous on T2WI** (due to the presence of necrotic debris, bleeding or infection), with proteinaceous fluid content arranged in layers (liquid-liquid level); areas of necrosis are displayed as hypointense irregular areas inside it. This is important in the management of these collections, because pseudocysts with necrotic material will not respond well to percutaneous drainage, requiring debridement. Respiratory-triggered T2-weighted images are the most effective in assessing these collections. *Fig. 22* on page 36.

**Wall-off pancreatic necrosis (WOPN)** is a partially liquefacted collection, which features solid content, developed as a late consequence of necrotizing pancreatitis. Its appearance in CT and MR is as an irregular fluid collection in the area of pancreatic necrosis, usually extended to the peripancreatic region. MR is more sensitive in detecting the solid content. It is usually possible to differentiate pseudocysts from WOPN when there is an obvious pancreatic necrosis in prior CT, although the wall-off necrosis may also develop from the peripancreatic tissue necrosis. *Fig. 23* on page 37.

It is sometimes difficult to differentiate the type of pancreatic collection, which is important for management; WOPN usually requires surgical approach or complex drainage techniques, while pseudocysts and abscesses are often resolved with simple percutaneous drainage techniques.

**The findings which make it possible to differentiate WOPN from pseudocyst** are the larger size of WOPN, a greater tendency to the septations, spread by the parietocolic gutters, worst definition of its edges, deformity and sometimes pancreatic discontinuity and presence of debris with fatty density/signal; on the other side, pseudocysts associate with dilatation of the MPD more frequently.

**Pancreatic abscesses** typically present with irregular and thick wall and septations but, as pseudocysts, they tend to show a smaller size and dilatation of the MPD. They usually respond better to the simple percutaneous drainage than the WOPN, as they have no solid content.
Complications of chronic pancreatitis include obstruction/stricture of bile ducts and duodenal obstruction, vascular stenosis (with portal hypertension), pseudocysts and pancreatic fistulas, among others. The pancreatic pseudocysts observed in patients with chronic pancreatitis usually have their origin as a sequel to episodes of acute inflammation.

Images for this section:

**Fig. 1**: MR in a patient with acute pancreatitis of biliary cause. T2WI with (a) and without (b) fat-suppression revealed biliary lithiasis (red arrows) and changes of acute pancreatitis (green arrows). The pancreas is enlarged and shows inhomogeneous signal, also seen in the peripancreatic tissue.

**Fig. 2**: Example of severe acute pancreatitis in axial (a) and coronal (b) contrast-enhanced CT, with pancreatic and peripancreatic fluid collections, showing gas inside
(blue arrow) and probably suggesting infection. Extensive necrosis of the gland was present (red arrows). The patient had concomitant intestinal ischemia; note the absence of enhancement of the descending colon wall (green arrow).

**Fig. 3:** Contrast-enhanced CT (a) of a patient with severe acute pancreatitis, with more than 50% of gland necrosis (arrow), showing lack of enhancement of the parenchyma. Images (b) and (c) correspond to the CT carried out a month later, where liquefaction of pancreatic necrosis can be seen and required fine-needle aspiration to rule out infection (c).
**Fig. 4:** Patient with mild acute pancreatitis. CT scan (a) shows absence of necrosis, with mild peripancreatic exudates (arrow). Signal changes in peripancreatic tissues are evident on fat-suppressed T2WI (b), showing hyperintensity as well as the presence of multiple cholelithiasis. On coronal T2WI (c), a pancreatic gland with featureless borders (typical of acute pancreatitis) is patent.
**Fig. 5:** MR of patient with severe acute pancreatitis. On axial in-phase T1WI the difference between the preserved signal of pancreatic head (a) and the hypointensity of the body and tail (b,c), corresponding to necrotic changes, is seen. Peripancreatic fluid collections (arrows) in left pararenal fascia and lesser sac are patent in T1WI (hypointense) and more evident in fat-suppressed T2WI (d) with high signal. Note a fluid-fluid level in the gallbladder, suggestive of biliary sludge. Contrast-enhanced MR (e) shows areas of pancreatic necrosis in body and tail with slight enhancement of the wall around the collection.
Fig. 6: Necrohemorrhagic acute pancreatitis. Fat-suppressed T1WI reflects a clear difference between non-necrotic tissue in uncinate process (green arrow in a) and necrotic tissue in isthmus and body (red arrow in b). MR can also detect bleeding changes as hyperintense areas (yellow arrow in c).
Fig. 7: Examples of patients with chronic pancreatitis and its findings in axial contrast-enhanced CT (a, b, c, d): pancreatic atrophy, dilatation of the common bile duct (red arrows), calcifications (green arrows) and occasionally, there may be pseudocysts (yellow arrow) with CBD dilatation.
**Fig. 8:** Axial (a, b) and coronal (c, d) CT in a patient with low attenuation density in the uncinate process (arrows), arising the differential diagnosis between adenocarcinoma or focal pancreatitis changes. There is dilatation of common bile duct with mild stenosis (which favors the diagnosis of inflammatory ethiology) and secondary duct branches dilatation in uncinate process. Note some small calcifications at this level.
Fig. 9: MR of the same patient. Axial fat-suppressed T1WI (a, b) exhibit a diffuse low signal in the uncinate process without a clear mass and with peripancreatic fatty tissues preserved. Cholangiographic image (c) reflects dilatation of intrahepatic bile ducts with mild intrapancreatic choledocal stenosis, sacular dilatation of MPD and dilatation of secondary duct branches. This patient underwent an ecoendoscopy with biopsy which was negative for adenocarcinoma. The final diagnosis was focal pancreatitis with branch duct type of intraductal papillary mucinous tumor located in the uncinate process.
Fig. 10: MR in a patient with exacerbated chronic pancreatitis. T1WI (a), T2WI (b) and cholangiographic images (c, d, e) contribute to reach a correct diagnosis. Signs of chronic pancreatitis such as atrophy of the gland (green arrow), dilatation of the MPD and intraductal lithiasis (yellow arrows), are seen. Peripancreatic exudates (red arrows), which have a high signal on T2WI and low signal on T1WI, are indicators of acute changes.
**Fig. 11:** Contrast-enhanced CT scan (a, b, c) shows findings suggestive of autoimmune pancreatitis, with an area of focal involvement in isthmus (red arrow). There is also loss of the lobulated contour of the pancreas (yellow arrows), typical of autoimmune pancreatitis. Axial T2WI (e, d) show similar findings as well as a stricture of the MPD in the pancreatic body with proximal dilatation (green arrow). Suspected autoimmune pancreatitis was confirmed with clinical and analytical data.
Fig. 12: CT scan (a, b, c) in patient with severe dilation of the intrapancreatic bile ducts; pancreas is slightly thickened, without evidence of focal damage or MPD dilatation. Note the loss of the lobulated pancreatic margins. Findings in T2WI (d, e) are virtually comparable. Final diagnosis was autoimmune pancreatitis. CT control after 6 weeks (f) shows an improvement of bile duct obstruction, highlighting a good response to corticosteroid therapy.

Fig. 13: Patient with paraduodenal pancreatitis. Note the hypointense thickening of pancreatoduodenal groove tissue (red arrow) on T1WI (a, b), while in T2WI (c) we can appreciate cysts along the medial wall of duodenum (green arrow). Contrast-enhanced MR (d) shows enhancement of fibrotic tissue in the pancreatoduodenal groove. In a second patient (e, f), there are cysts and duodenal wall thickening as well as signs of chronic pancreatitis (main and secondary duct dilation).
**Fig. 14:** Example of a patient with paraduodenal pancreatitis. Fat-supressed T2WI (a) and cholangiographic image (b) show cysts in the medial duodenal wall (red arrows), adjacent tissue signal alteration (green arrow) and associated changes of chronic pancreatitis. Note the dilatation of the MPD and secondary branches, as well as pancreatic calcifications, patent in CT MIP images (c).
**Fig. 15:** Example of cystic dystrophy of the duodenal wall. Both CT (a, c, d) and T2 fat-suppressed WI (b) are optimal imaging modalities for assessing this pathology. Note the cystic lesions in duodenal wall and slightly enhancing thickened tissue in delayed phases (arrows).
Fig. 16: Patient with a history of recurrent episodes of acute pancreatitis of uncertain origin (latest exacerbation two months before the exploration). MR shows on T2 fat-suppressed WI (a, b, c, d) mild signal changes in the peripancreatic tissue (arrow), consistent with residual inflammatory phenomena. Note also a MPD coursing anterior to the common bile duct before draining into the minor papilla. Cholangiographic images thin-slab 3D reconstruction (c) and 2D thick-slab acquisition (d) show the unusual route of the dorsal duct. Diagnosis of pancreas divisum was established.
Fig. 17: Study in patient with jaundice and abdominal pain. CT scan (a) shows dilatation of CBD and MPD, with a hypodense image (green arrow) located in uncinate process. T2WI (b, c) show a significant dilatation of the MPD with sudden obstruction and obvious peripancreatic signal changes, suggestive of being exudates (red arrows). It was diagnosed as a likely pancreatic neoplasm (adenocarcinoma) with acute pancreatitis changes associated. This diagnosis was surgically confirmed.
Fig. 18: Patient who underwent ERCP and later developed abdominal pain clinically compatible with pancreatitis. CT scan (a, b) shows significant right perirrenal exudates as well as aerobilia and air inside Wirsung duct (arrows). Pancreatitis is a rare complication of ERCP.

Fig. 19: Patient with history of abdominal trauma. The contrast-enhanced CT scan (a, b) shows peripancreatic exudates (red arrows) and faint low attenuation of the pancreatic body with some ectasia of the MPD (green arrow). MR performed within 30 days of
income (c), revealed a fluid collection attached to the greater curvature of stomach and a smaller one adjacent to the uncinate process. Coronal T2WI (d, e) showed the same collection in gastric wall and an tubular image with liquid signal (red arrows) in the pancreatic body, which was different from MPD, (green arrows), suggestive of being a pancreatic fistula due to injury of the MPD; which communicates with the fluid collection(f).

**Fig. 20:** Contrast-enhanced CT scan of a patient with acute pancreatitis with signs of pancreatic necrosis(a). CT performed 4 weeks later (b, c) reveals a voluminous fluid collection in prior location of pancreatic necrosis. Note the indirect signs of thrombosis of the splenic vein, with collateral circulation through gastroepiploic veins(arrows).
**Fig. 21:** CT scan (a) showing changes of pancreatitis with acute peripancreatic fluid collections consistent with exudates; CT scan three weeks later (b) reveals more organized peripancreatic collections, having thick and enhancing wall. In a different patient (c), a peripancreatic collection with thick wall and heterogeneous content is seen, likely corresponding to solid debris (arrow) and suggestive of walled-off necrosis.
Fig. 22: Axial and coronal (a, b) T2WI showing a fluid collection in the pancreatic tail (red arrow) which has a thick and hypointense wall as well as hyperintense content, being findings consistent with pseudocyst. The inferior images show a fluid collection in pancreatic tail in a different patient. MR highlights its hyperintense content (yellow arrow) on in-phase T1WI (c) and slightly hypointense on T2WI (d, e), thus reflecting a blood content. Pseudocysts may have bleeding changes in their evolution.
**Fig. 23:** CT scan (a, b) in a patient with acute pancreatitis, showing a voluminous fluid collection with extension to lesser sac and towards the parietocolic area. It has irregular margins, with heterogeneous content and including areas of fat density (arrows). Coronal and axial T2WI (c,d,e) show the extension of the collection and a heterogeneous content. The diagnosis that should be considered with these findings is pancreatic necrosis without wall (walled off necrosis).

<table>
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<tr>
<th>Balthazar grade</th>
<th>Appearance on CT</th>
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<th>Necrosis percentage</th>
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<tr>
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<tr>
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<td>&gt;50%</td>
<td>6</td>
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<tr>
<td>E</td>
<td>Two or more fluid collections and/or gas bubbles in or adjacent to pancreas</td>
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**Table 1**
Conclusion

MR is useful in the management of many patients with pancreatitis, in determining the etiology and evaluating the possible associated complications, often being an essential and a complementary modality besides ultrasound and CT.

Any radiologist dedicated to abdominal pathology must know indications of MR in these patients and the different image findings of the usual and unusual pancreatitis in order to adapt the management to each case and minimize unnecessary interventions.

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