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**Pancreatitis-imaging approach**

Busireddy KK *et al.*Pancreatitis-imaging approach

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**Abstract**

Pancreatitis is defined as the inflammation of the pancreas and considered the most common pancreatic disease in children and adults. Imaging plays a signiﬁcant role in the diagnosis, severity assessment, recognition of complications and guiding therapeutic interventions. In the setting of pancreatitis, wider availability and good image quality make multi-detector contrast-enhanced computed tomography (MD-CECT) the most used imaging technique. However, magnetic resonance imaging (MRI) offers diagnostic capabilities similar to those of CT, with additional intrinsic advantages including lack of ionizing radiation and exquisite soft tissue characterization. This article reviews the proposed definitions of revised Atlanta classification for acute pancreatitis, illustrates a wide range of morphologic pancreatic parenchymal and associated peripancreatic changes for different types of acute pancreatitis. It also describes the spectrum of early and late chronic pancreatitis imaging findings and illustrates some of the less common types of chronic pancreatitis, with special emphasis on the role of CT and MRI.

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**Key words:** Computed tomography; Magnetic resonance imaging; Acute pancreatitis; Chronic pancreatitis; Autoimmune pancreatitis; Chronic pancreatitis; Revised Atlanta classification; Motion-resistant imaging

**Core tip:** Imaging plays an important role in the diagnosis and staging of acute and chronic pancreatitis. Wider availability and good image quality makes computed tomography (CT) the mostly used imaging technique; however, magnetic resonance imaging (MRI) offers diagnostic capabilities similar to those of CT, with additional intrinsic advantages including lack of ionizing radiation and exquisite soft tissue characterization. This article reviews and illustrates the proposed definitions of the revised Atlanta classification for acute pancreatitis. It also describes the spectrum of early and late chronic pancreatitis imaging findings, with special emphasis on the role of CT and MRI.

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**INTRODUCTION**

Pancreatitis is defined as the inflammation of the pancreas and considered the most common pancreatic disease in children and adults. It can be acute; representing an acute inflammatory process of the pancreas, or chronic; progressing slowly with continued, permanent inflammatory injury to the pancreas.

The incidence of acute pancreatitis is increasing in the United States and worldwide contributing to be one of the major sources of hospitalization. Acute pancreatitis was the most common gastrointestinal diagnosis for hospitalization (with 274119 discharges) in the United States in 2009[[1](#_ENREF_1)], usually running a mild clinical course[[2](#_ENREF_2)]. However, a subset of patients develop severe disease independent of the degree of initial insult or etiology, with high morbidity and mortality up to 45%[[3](#_ENREF_3)]. Over one-half of cases of acute pancreatitis in adults are related to cholelithiasis or alcohol consumption; whereas trauma, viral infections and systemic diseases account for the majority of cases in children[[4](#_ENREF_4)].

The incidence of chronic pancreatitis is between five and twelve cases per 100000 persons per year; accounting for more than 120000 outpatient visits and 50000 hospitalizations annually[[5](#_ENREF_5)]. Alcohol consumption accounts for the majority (80%) of cases of chronic pancreatitis in adults in developed countries; whereas malnutrition is the most common cause worldwide[[4](#_ENREF_4)].

The purpose of our review is to illustrate the different imaging findings of pancreatitis on computed tomography (CT) and magnetic resonance imaging (MRI); with special emphasis on the revised terminology for acute pancreatitis and substantiate the increasing importance of imaging in the diagnosis, staging and follow-up of acute and chronic pancreatitis[[5](#_ENREF_5)].

***Acute pancreatitis***

Acute pancreatitis results from the exudation of fluid containing activated proteolytic enzymes into the interstitium of the pancreas and leakage of this fluid into surrounding tissue.

There is general acceptance that a diagnosis of acute pancreatitis requires two of the following three features: (1) Sudden onset abdominal pain suggestive of acute pancreatitis (epigastric pain radiating to the back); (2) Serum amylase and/or lipase levels at least 3 times greater than the upper limit of normal; and (3) Characteristic imaging findings of acute pancreatitis on contrast-enhanced computerized tomography (CECT), MRI, or transabdominal ultrasonography (US) studies.

If abdominal pain is strongly suggestive of acute pancreatitis but the serum amylase and/or lipase activity is less than 3 times the upper limit of normal, characteristic findings on a CECT or MRI are required to confirm the diagnosis[[6](#_ENREF_6)].

In order to assess and predict local or systemic effects of pancreatic injury, several disease severity-scoring systems were developed (*e.g.*, Ranson score, APACHE-II). In 1992, the Atlanta classification for acute pancreatitis was introduced to establish international standards of definitions of acute pancreatitis and its complications[[7](#_ENREF_7)]. This system was designed to facilitate understanding and correlation of findings seen by gastroenterologists, pathologists, radiologists and surgeons; aiding improved communication between clinicians.

This initial Atlanta classification system represented major progress; however, advancing knowledge of the disease process, improved imaging and ever-changing treatment options warranted a revision, which was undertaken in 2012.

The revision of the Atlanta classification focuses heavily on morphologic criteria for defining the various manifestations of acute pancreatitis as outlined principally by means of CT and MRI.

Two distinct phases of acute pancreatitis were introduced: a first, or early, phase that occurs within the 1st wk of onset of disease; and a second, or late, phase that takes place after the 1st wk of onset[[7](#_ENREF_7)].

***Early or first phase (less than 1 wk)***

During this phase, pancreatic or peripancreatic ischemia or edema may completely resolve, develop fluid collections or progress to permanent necrosis and liquefaction. Severity of the acute pancreatitis in the early phase is entirely based on clinical parameters; mainly determined by the presence and duration of organ failure, but not the morphologic characteristics and its extent in and around the pancreas[[8](#_ENREF_8)].

***Late or second phase (after 1 wk from onset)***

This phase occurs mostly in patients with moderate to severe acute pancreatitis and may extend for weeks to months. It is characterized by the presence of local complications, systemic manifestations (due to ongoing inflammation) and/or by transient or persistent organ failure. In this stage, the need for treatment is determined by presence of symptoms or complications, and the type of management is mainly based on the morphologic characteristics of pancreatic and peripancreatic region seen on cross sectional imaging.  The severity of acute pancreatitis in late phase is determined by both morphologic criteria and clinical criteria like persistence of organ failure.

***Updated terminology of acute pancreatitis***

The web based international consensus[[7](#_ENREF_7)] revised the original Atlanta classification of 1992 and proposed a new classification of acute pancreatitis to avoid the confusion in terminology seen over the last 2 decades. This consensus classiﬁcation deﬁnes criteria for the diagnosis of acute pancreatitis (see above), differentiates the two types of acute pancreatitis (interstitial edematous pancreatitis and necrotizing pancreatitis) classiﬁes the severity of acute pancreatitis into three categories and deﬁnes the morphology seen on imaging of pancreatic and peripancreatic collections that arise as complications of acute pancreatitis.

***Role of imaging in acute pancreatitis***

Imaging plays a significant role in the diagnosis of acute pancreatitis in clinically suspected cases or suggesting alternative diagnoses. It helps determine the causes of pancreatitis: gallstones, biliary duct obstruction or structural abnormalities. It also helps in grading the severity of the disease and identifying pancreatic or peripancreatic complications. Additionally, imaging can be utilized to guide therapeutic interventions.

The choice of appropriate imaging modality depends on the reason for investigation, clinical symptoms, time of onset of symptoms and lab findings. However, CECT is the most commonly used modality in the evaluation of acute pancreatitis. In 2010, the ACR committee on appropriateness criteria and its expert panels have developed guidelines for determining the most appropriate imaging examinations for the diagnosis and treatment of acute pancreatitis and have given high score ratings[8,9] to CECT in different clinical scenarios. This is based on it is wide availability and high degree of accuracy. They also stated that MRI appears to offer diagnostic capabilities similar to multi-detector computed tomography (MDCT) with intrinsic advantages including the lack of ionizing radiation and the exquisite soft tissue characterization unmatched by any other imaging modality; allowing better depiction of stones and evaluation of the pancreaticobiliary ductal system.

***Ultrasound***

Ultrasound is frequently the first investigation performed on admission; although it has little value in the diagnosis of pancreatitis or its complications. Ultrasound is usually reserved to confirm or exclude the presence of stones or biliary dilatation. Early identification and treatment of these calculi may have a significant positive impact on outcome. However, body habitus of patient, operator dependence pose a limitation in detection of distal common bile duct stones accurately compared to CECT or MR Imaging[[9](#_ENREF_9)].

Ultrasound is limited in evaluating the entire pancreatic parenchyma; which is often partially or completely obscured by overlying bowel gas. It can however be helpful in monitoring the evolution of fluid collections, which occur as a result of acute pancreatitis, and in guiding diagnostic and therapeutic interventions.

***CECT***

CECT can show morphologic characteristic findings that allow for establishing the diagnosis of acute pancreatitis and determining the extent of disease severity. The best time for performing CECT in acute pancreatitis not well established and if performed immediately after the onset of symptoms, the full extent of pancreatic damage and its severity can be easily underestimated[[10](#_ENREF_10),[11](#_ENREF_11)]. Conversely, a CECT obtained more than 5 days after onset of symptoms that reveals a normal aspect of the pancreas or only mild inﬂammatory changes (fat stranding) surrounding the pancreas virtually excludes a severe form of acute pancreatitis[[12](#_ENREF_12)].

Not all patients with acute pancreatitis need to undergo contrast-enhanced CT. In general, CT is not indicated in patients who are clinically classiﬁed as having mild pancreatitis (no clinical signs of severe pancreatitis) and show rapid improvement with appropriate medical management. CT should be used in patients who are classiﬁed as having severe pancreatitis or are at risk of developing severe pancreatitis; ideally after 72 hours, to best assess the full extent of the disease[[13](#_ENREF_13)].

CT should be repeated when the clinical picture drastically changes, such as with sudden onset of fever, decrease in hematocrit or sepsis. CT can also be useful to guide catheter placement for drainage and to assess success of treatment in patients who underwent percutaneous drainage or other interventions.

Furthermore, in patients with their first episode of pancreatitis who are over 40 years of age and have no identifiable cause for pancreatitis, contrast-enhanced CT should be used to exclude a possible neoplasm[[13](#_ENREF_13)] (Table 1).

The main limiting factors for CECT are ionizing radiation, use of iodinated contrast material; especially in patients in with renal failure or contrast allergy and moderate sensitivity in identifying gallstones and biliary stones[[14](#_ENREF_14),[15](#_ENREF_15)]. The above limiting factors can be over­come by using MRI; which does not use ionizing radiation; allowing it to be used during pregnancy, in patients with recurrent pancreatitis and for patients requiring multiple follow-up exams. Non-enhanced MRI seems to be more accurate and reliable for the early assessment of severity and prognosis of acute pancreatitis than is contrast-enhanced CT[[16-18](#_ENREF_16)]; thus proving beneficial in patients with renal failure and history of contrast allergy.

***MRI***

Recent technological developments have dramatically improved the quality of abdominal MRI. Respiration, bowel peristalsis and vascular pulsations are major sources for artifacts affecting the accuracy and reproducibility of MRI. Breathing-independent sequences and respiratory gating techniques form the foundation of high-quality abdominopelvic MRI. New motion-robust MRI techniques provide promising results even in detection and characterization of pancreatic disease in patients that are not able to cooperate with breath-hold instructions[[19](#_ENREF_19)].

A variety of pulse sequences are currently used for abdominal MRI including T1- and T2-weighted sequences with or without fat-suppression and post-gadolinium T1-weighted sequences (Figure 1). MR Cholangiopancreatography (MRCP) is routinely added to abdominal protocols to assess ductal obstruction, dilatation or course[[20-22](#_ENREF_20)] (Figure 2); providing comprehensive evaluation of full range of pancreatic diseases. Due to the increasing incidence of acute pancreatitis due to gallstones in the United States, it is more beneficial to consider MRCP as an initial diagnostic study.

MRI is sensitive for detection of subtle changes of acute pancreatitis; particularly minor peripancreatic inﬂammatory changes; even in the setting of a morphologically normal pancreas on CT imaging; which may appear normal in up to 15%–30% of patients with clinical features of acute pancreatitis[[23](#_ENREF_23)]. The sensitivity of MRI exceeds that of CT imaging, emphasizing its role in the evaluation of patients with clinically suspected acute pancreatitis and negative CT imaging findings.

It should be emphasized that MRI is a non-ionizing cross sectional imaging method and has a safer intravenous contrast profile in comparison to CT. This is particularly important in radiosensitive populations and those requiring repeated imaging followup. Additionally, patients who present with acute pancreatitis often have a degree of renal impairment.

The factors that make CECT the most frequently applied imaging approach in pancreatitis are related to its universal availability (especially near the emergency room), faster scanning times, and relatively easier interpretability of CT images by physicians and general radiologists. For early presentation of acute pancreatitis, CT might be the preferred method for the reasons stated above. However, the adequate diagnostic performance of MRI along with the mentioned additive advantages favors MRI as the preferred method.

***Endoscopic ultrasound***

Endoscopic ultrasound (EUS) has shown great utility in providing high-resolution images of the pancreatic duct and parenchyma as well as extra hepatic biliary system; as the probe can be positioned in close proximity to the pancreas. Furthermore, EUS has become an invaluable technique for its ability to obtain targeted biopsies of lesions in and around the pancreas; thus, playing a prominent role in evaluating patients with atypical findings on other imaging studies.

The disadvantages of EUS are the requirement of monitored anesthesia care, need for expert endo-sonographer, modality operator dependence, and interobserver variability.

According to ACR appropriateness criteria, the role of endoscopic US in the evaluation of acute pancreatitis is primarily reserved for assessing and/or confirming choledocolithiasis and subsequent stone removal, as well as for identifying anatomic abnormalities (*e.g.*, pancreas divisum or malignancy) that can lead to acute pancreatitis.  However, it has been recently proposed to use EUS in acute pancreatitis, as it is was found to contribute for the detection of causes like cancer, microlithiasis and chronic pancreatitis[[24](#_ENREF_24)].

**IMAGING-BASED MORPHOLOGIC TYPES OF ACUTE PANCREATITIS**

***Interstitial edematous pancreatitis***

Interstitial edematous pancreatitis (IEP) is a milder form of acute pancreatitis that usually resolves over the first week. IEP is characterized by diffuse or localized enlargement of the pancreas secondary to interstitial or inflammatory edema without necrosis.

On CECT, findings include enlarged pancreas with relatively normal enhancement. Peripancreatic fat may be normal or show mild stranding and ground glass opacity due to inflammation, with small to varying amounts of non-enhancing peripancreatic fluid (Figure 3). The characteristic CECT finding that distinguishes IEP is absence of pancreatic parenchymal and peripancreatic necrosis.

On MRI, the signal intensity characteristics of the pancreas in IEP resemble those of normal pancreatic tissue. Enlargement of the pancreas, parenchymal edema and fat stranding are well demonstrated on T1-weighted images (Figure 4). T1-weighted imaging with fat suppression improves the delineation of the pancreas and pancreatic borders[[25](#_ENREF_25)]. The pancreas demonstrates high signal intensity on pre-contrast fat suppressed T1-weighted images and enhances uniformly on immediate post-gadolinium images, reﬂecting a normal capillary blush. Fat suppressed T2-weighted sequences are very sensitive for detecting edema or minimal fluid and therefore have a role in detecting even milder forms of pancreatitis[[26](#_ENREF_26)](Figure 5).

***Necrotizing pancreatitis***

Necrotizing pancreatitis is the inflammation of the pancreas with obvious pancreatic and peripancreatic tissue necrosis. About 5–10% of patients develop necrosis; affecting the pancreatic parenchyma in 5%, peripancreatic tissue in 20% and both in 70%. Pancreatic parenchymal necrosis carries a worse prognosis than peripancreatic necrosis[[27](#_ENREF_27)].

Atlanta classiﬁcation deﬁnes necrotizing pancreatitis as being associated with more than 30% parenchymal necrosis. The presence of less than 30% necrosis demands follow-up scanning in 1 wk to confirm true necrosis *vs* IEP[[27](#_ENREF_27)].

On CECT, findings include areas of compromised pancreatic parenchymal enhancement on the post-Gadolinium images with or without peripancreatic inhomogeneous fluid collections (Figsure 6 and 7). The impairment of pancreatic perfusion and signs of peripancreatic necrosis evolve over several days[[28](#_ENREF_28)], which explains why an early CECT may underestimate the eventual extent of pancreatic and peripancreatic necrosis.

On MRI, necrosis shows appears as hypointense areas on T1-weighted images corresponding to areas of increased signal on fat-suppressed T2 weighted-images, associated with well­ defined areas of non-­enhancing pancreatic parenchyma on post-Gadolinium sequences[[29-31](#_ENREF_29)] (Figure 8).

For both CT and MRI, acquisition of an adequate arterial phase is of the utmost importance; as the maximum enhancement of pancreas is reached on the late arterial phase, and higher difference in signal between viable and necrotic is achieved on this phase.

Pancreatic duct disruption is an important prognostic factor. It is seen in 30% of the patients of necrotizing pancreatitis[[32](#_ENREF_32)] when necrosis involves the central gland[[33](#_ENREF_33),[34](#_ENREF_34)]. Drake *et al*[[35](#_ENREF_35)] study showed that MRCP, a noninvasive imaging method, achieved 95% accuracy in detecting pancreatic duct disruption; thus helping in identifying patients who might benefit from early treatment.

***Definition of pancreatic and peripancreatic collections***

An important distinction is made between collections that are composed of fluid alone and those that arise from necrosis and contain a solid component (and which may also contain varying amounts of fluid). Below, we define and illustrate the following terms: acute peripancreatic fluid collection; occurring in interstitial edematous pancreatitis, pancreatic pseudocyst as a delayed (usually after 4 wk) complication of interstitial edematous pancreatitis and necrosis; which may be an acute necrotic collection (in the early phase and before demarcation) or walled-off necrosis surrounded by an identifiable capsule on imaging (rarely develops before 4 wk).

***Acute peripancreatic fluid collections***

Fluid collections less than 4 wk in IEP lacking a discrete wall, with no internal solid components in the peripancreatic region are called acute peripancreatic fluid collections (APFC). Approximately 50% of APFC’s develop within 48 h following the onset of acute pancreatitis[[36](#_ENREF_36)].

On CT scan, they appear as homogenous collections with low attenuation. They do not have well-defined walls and are confined by normal fascial planes in the retroperitoneum (Figure 9). They can be single or multiple (Figure 10). Most acute fluid collections remain sterile and usually resolve spontaneously without intervention[[30](#_ENREF_30)].

On MRI, T2-weighted sequences are very sensitive in detecting peripancreatic fluid; which demonstrate high T2 signal intensity. On T1-weighted gradient echo images, APFC’s demonstrate low signal intensity in a background of high signal intensity fat. No perceptible enhancement is depicted on post-gadolinium fat-suppressed T1-weighted images. The majority of fluid collections are typically confined to the lesser sac and anterior pararenal space or may track down to the pelvis and superiorly into mediastinum[[29](#_ENREF_29)]. These collections are usually sterile and are spontaneously reabsorbed.

***Pancreatic pseudocysts***

Peripancreatic fluid collections that persist more than 4 wk in IEP, with a well-defined wall and no internal solid components in the peripancreatic region are called pseudocysts.

On CECT, they appear as homogenous collections of low-attenuation surrounded by a uniform enhancing capsule (Figure 11). Typically, an increase of enhancement is observed in the interstitial phase; reflecting the presence of granulation tissue.

On MRI, pseudocysts demonstrate low in signal intensity on and T1-weighted gradient-echo images and relatively homogeneous high signal intensity on T2-weighted images. Pseudocysts walls enhance minimally on early post-gadolinium images and show progressively intense enhancement on 5-min post-gadolinium images; due to the presence of ﬁbrous tissue (Figure 12).

Pseudocysts may sometime have communication with pancreatic duct and detecting this communication is helpful in the further patients’ management. MRCP; a noninvasive imaging modality has an advantage of demonstrating possible communication between pancreatic pseudocyst and pancreatic duct.

The majority of pseudocysts resolve spontaneously. Infection and hemorrhage may complicate simple pseudocysts. Infected pseudocyst may contain gas bubbles on CT. However, absence of these findings on CT may further require confirmation by fine needle aspiration, when there is a strong clinical suspicion.

***Acute necrotic collections***

During the first 4 wk, a collection containing variable amounts of fluid and necrotic tissue is termed an acute necrotic collection (ANC). Unlike APFCs, ANCs are present within the pancreas and peripancreatic regions. ANC’s may often maintain communication with the main pancreatic duct or one of its side-branches; for which, MRI can be useful in delineating this connection.

On CECT, ANC’s demonstrate heterogeneous attenuation variably higher that of thin fluid (Figure 7). Follow-up imaging may be useful to characterize acute collections. CECT often shows ANC’s as a homogenous non-enhancing area during the first week of necrotizing pancreatitis; making it difficult to be differentiated from APFC’s. MRI may be helpful to confirm the presence of solid content in the collection.

On MRI, the necrotic debris may appear as irregularly shaped regions of low signal intensity within the necrotic collections. Breathing-independent T2-weighted sequences such as single-shot echo-train spin echo are useful to evaluate these necrotic collections (Figure 13); not only because of their high sensitivity in demonstrating the complexity of ﬂuid, but also because many of these patients are very debilitated and are unable to cooperate with breath-holding instructions.

An advantage of MRI relative to MDCT in the evaluation of peripancreatic fluid collections is easier appreciation of solid debris with MRI[[37](#_ENREF_37)]. The sensitivity and specificity of MRI in detecting solid debris of necrosis is 100% when compared to CT; which has a sensitivity of 25% and a specificity of 100%[[38](#_ENREF_38)]. MRI can help in differentiating fluid collections secondary to pancreatitis from other cystic neoplasms.

***Walled-off necrosis***

After 4 wk, APFC’s mature and develop thick non-epithelialized wall; acquiring the term walled-off necrosis (WON). They commonly occur in the pancreatic body and tail. Management for WON is different from pseudocyst as it contains non-liquefied debris; which needs to be surgically removed. Previously suggested nomenclature for this entity includes: organized pancreatic necrosis, pancreatic sequestration, pseudocyst associated with necrosis and subacute pancreatic necrosis.

On CECT, walled-off necrosis demonstrates a heterogeneous fluid and non-fluid attenuation with varying degree of loculations surrounded by a well-defined and enhancing encapsulating wall; which may involve both the pancreatic and extrapancreatic tissue. CECT, however, may not readily distinguish solid from fluid contents; as a result, pancreatic and peripancreatic necrosis may be misdiagnosed as a pancreatic pseudocyst. For this purpose, MRI may be required for this distinction (Figure 14).

***Infected pancreatic necrosis***

Pancreatic and peripancreatic necrosis can remain sterile or become infected. The development of secondary infection in pancreatic necrosis is associated with increased morbidity and mortality[[3](#_ENREF_3)]. Most studies suggest that there is no absolute correlation between the extent of necrosis and the risk of infection and duration of symptoms[[7](#_ENREF_7)]. The early diagnosis of infected pancreatic necrosis is very important in the initiation of antibiotic therapy.

The diagnosis of infected ANC or WON can be suspected in the presence of extraluminal gas on CT or MRI. This extraluminal gas is present in areas of necrosis and may or may not form a gas/fluid level depending on the amount of fluid content present at that stage of the disease (Figure 15). The diagnosis may be confirmed by aspiration and analysis including microscopy and culture.

**SEVERITY OF ACUTE PANCREATITIS**

**Clinical *vs* MCTSI *vs* MRSI severity index:**

Several clinical scoring systems like Marshal, SOFA. APACHE or Ranson criteria were designed to accurately correlate the complications like organ failure and mortality in acute pancreatitis. In the last two decades, radiological scoring systems were developed to accurately diagnose and correlate complications in acute pancreatitis.

For the first time in 1990, Balthazar *et al*[[28](#_ENREF_28)] introduced the CT severity index for assessment of AP; which correlated well with morbidity, mortality and length of hospital stay. CTSI was widely adopted in clinical and research settings; however, a potential limitation was its inability to detect pancreatic necrosis. MCTSI introduced by Mortele KJ *et al*[[39](#_ENREF_39)] in 2004 to account for the limitations of CTSI (Table 2); which showed improved correlation with severity.

MCTSI incorporated extrapancreatic manifestations and simplified the evaluation of extent of parenchymal necrosis by categorizing­ into none, less than 30% or more than 30%; in addition to evaluating peripancreatic inflammation by detecting the presence or absence of peripancreatic fluid. Predictive accuracy of CT scoring systems for severity of AP and comparisons between CTSI and MCTSI were made[[40](#_ENREF_40)]. They reported that they could not detect any significant differences between CTSI and MCTSI in evaluating the severity of AP. Their study also demonstrated that compared with APACHE II, both CT indexes more accurately diagnosed clinically severe disease and correlated better with the need for intervention and pancreatic infection.

It has been reported that MR severity index (MRSI) significantly correlated with CTSI (Table 3), Ranson score, C-reactive protein levels, appearance of systemic complications, duration of hospitalization and clinical outcome[[17](#_ENREF_17),[41](#_ENREF_41)].

***Chronic pancreatitis***

Chronic pancreatitis is deﬁned pathologically by continuous or relapsing inﬂammation of the organ leading to irreversible morphologic injury and typically leading to permanent impairment of both exocrine and endocrine functions. The incidence of chronic pancreatitis ranges from 5-12 per 100000 people in industrialized countries[[1](#_ENREF_1)].

Chronic pancreatitis is a cause of abdominal pain, weight loss, steatorrhea and diabetes mellitus, which may occur as a consequence of multiple factors, including biliary stone disease, alcohol consumption, malignancy, metabolic disorders, and various genetic and environmental insults, including trauma[1].

The histopathological changes in chronic pancreatitis evolve from unevenly distributed fibrosis in early chronic pancreatitis to diffuse fibrosis involving the entire gland in late stages. In advanced disease, large areas of acinar parenchyma are replaced with sclerotic tissue causing atrophy. Ductal irregularities like strictures, dilatation and side branches ectasia occur due to surrounding fibrosis. Other characteristic findings of severe chronic pancreatitis are calcifications and presence of complications like pseudocyst, vascular aneurysms and venous thrombosis.

***Role of imaging***

Imaging plays a significant role in detecting parenchymal and ductal abnormalities in chronic pancreatitis and helps in differentiating early from advanced phases to a certain extent; which further guides the management of these patients.

Most commonly accepted CT- and MRI-based criteria for diagnosis of chronic pancreatitis are shown in Table 4.

***Early chronic pancreatitis***

Ultrasound and CT are insensitive in diagnosis of early chronic pancreatitis, as they often show no abnormalities.  A recent study showed that parenchymal changes might precede ductal changes in chronic pancreatitis; thus depicting the importance of MRI compared to MRCP in early diagnosis of disease[[42](#_ENREF_42)].

On MRI, normal pancreas is hyperintense on T1 weighted images and shows uniform enhancement on the late arterial phase (Figure 1). MRI detects not only morphologic characteristics, but also early fibrotic changes. Fibrosis is shown by diminished signal intensity on T1-weighted fat-suppressed images and diminished enhancement on immediate post-Gadolinium gradient-echo images[[43](#_ENREF_43)]. Low signal intensity on fat-suppressed T1-weighted images reﬂects loss of the aqueous protein in the acini of the pancreas. Diminished enhancement on capillary phase images reﬂects disruption of the normal capillary bed and increased chronic inﬂammation and ﬁbrosis.

MRCP findings in early chronic pancreatitis often demonstrate normal main pancreatic duct with dilated and irregular side duct branches. The limiting factor is the underestimation of ductal size. Some investigators reported that patients with abnormal MR imaging findings but normal MRCP might benefit from dynamic secretin-MRCP (S-MRCP) (Figure 16); which may reveal ductal abnormalities due to improved visualization otherwise not detected on MRCP[[42](#_ENREF_42)]. Secretin-MRCP has been reported to show ductal changes, like dilatations and strictures in early chronic pancreatitis.

EUS has a prominent role in chronic pancreatitis for its ability to detect early morphologic changes. Endoscopic retrograde cholangiopancreatography (ERCP) is considered to be gold standard test in detecting early changes, but unlike ERCP, EUS is relatively a non-invasive procedure, and also helps in the evaluation of both pancreatic duct and parenchymal changes compared to ERCP that has limitation in evaluating pancreatic side branches and parenchyma[[44](#_ENREF_44)]. Chong AK *et al*[[45](#_ENREF_45)] showed sensitivity of 83% and specificity of 80% of EUS for the diagnosis of chronic pancreatitis.

***Late chronic pancreatitis***

CT is reported to be 60% to 95% sensitive in diagnosing advanced disease as it can readily detect parenchymal changes associated with advanced chronic pancreatitis[[46](#_ENREF_46)]. Most common findings on CT include dilatation of main pancreatic duct and its side branches; which can be seen in 68% of patients. The ductal contour may be smooth, beaded or irregular[[47](#_ENREF_47)].

Other findings include intraductal calcifications, which is the most specific finding and is seen in nearly half of the patients with chronic pancreatitis and parenchymal atrophy (Figure 17). However, parenchymal atrophy is neither specific nor sensitive as it seen normally with aging. Intraductal or parenchymal calcifications are usually seen with alcohol related chronic pancreatitis but not on chronic pancreatitis resultant from other causes.

All patients with late or advanced chronic pancreatitis show diminished signal intensity of the pancreas on T1- weighted fat-suppressed images, an abnormally low percentage of contrast enhancement on immediate post-contrast images, and progressive parenchymal enhancement on the 5-min delayed post-contrast images; reﬂecting the pattern of enhancement of ﬁbrous tissue.

MRCP in advanced phase demonstrates dilatation of the main pancreatic duct with ectasia of the side branches (Figure 18); giving chain of lakes appearance manifested as pancreatic ductal strictures, irregularities and intraductal calculi, appearing as hypointense filling defects.

***Enlarged pancreatic head in chronic pancreatitis vs adenocarcinoma***

Chronic pancreatitis may involve only the pancreatic head in 30 % of patients, resulting in focally enlarged pancreatic head. In these cases, the focus of chronic pancreatitis can simulate the appearance of pancreatic ductal adenocarcinoma.

Both chronic pancreatitis and adenocarcinoma show similar imaging characteristics on CT and MRI due to abundant fibrosis and ductal obstruction; therefore, making the differentiation between these two entities very difficult. Both are generally seen as hypodense lesions on CT, mildly hypointense on T1-weighted images and heterogeneously mildly hyperintense signal on T2-weighted images. However, certain imaging characteristics are helpful in distinguishing enlarged pancreatic head in chronic pancreatitis from adenocarcinoma (Table 5).

Rarely, chronic pancreatitis may involve only the focally enlarged portion of the pancreas, with the reminder of the pancreas having no inﬂammatory changes. In these cases, the focus of chronic pancreatitis can also simulate the appearance of pancreatic ductal adenocarcinoma. The inﬂammatory process may also be sufﬁciently destructive that underlying stromal pattern is lost. In these rare cases, diagnosis can only be established by surgical resection and histopathological examination to conﬁrm the absence of malignancy.

Despite the high-resolution images produced by conventional EUS, there are no specific EUS imaging features that can differentiate pancreatic cancer from other common mimics, including lymphoma, focal pancreatitis, neuroendocrine tumors, metastases, and focal AIP[[48](#_ENREF_48)]. However, one of the strengths of EUS is its ability to allow guided fine needle aspiration (FNA); which may overcome this problem.

In a retrospective analysis by Agarwal *et al*[49], 110 patients with abnormal CT or MRI with an enlarged head of the pancreas or dilated pancreatic duct with or without dilation of the common bile duct underwent EUS or EUS-FNA. The study revealed an accuracy of 99.1% for EUS and/or EUS-FNA in diagnosing pancreatic neoplasm with a sensitivity of 88.8% and specificity of 100%[[49](#_ENREF_49)]. Given the high accuracy in the evaluation of pancreatic tumors, Eloubeidi *et al*[50] proposed routine EUS-FNA for the differential diagnosis of solid pancreatic masses. Other studies have shown that a negative EUS in ambiguous cases (where a mass is suspected) has a high negative predictive value[[51](#_ENREF_51), [52](#_ENREF_52)].

Positron emission tomography–computed tomography (PET-CT) has an established role in the diagnosis of pancreatic carcinoma, especially when cross sectional imaging or biopsies are equivocal or nondiagnostic.  In patients with a suspicion of pancreatic malignancy, a focal increase in 18F-fluorodeoxyglucose (FDG) uptake suggests the diagnosis of malignancy. Nonetheless, the cutoff value of maximum standardized uptake value (SUVmax) is not defined, as it overlaps in benign and malignant pancreatic disease processes[[53](#_ENREF_53),[54](#_ENREF_54)].

Furthermore, FDG-PET’s detectability of pancreatic cancer depends on lesion size and degree of FDG uptake and surrounding background uptake. In the setting of chronic pancreatitis, FDG-PET is shown to detect pancreatic adenocarcinoma with a sensitivity of 92% and with a negative predictive value of 87%. In the setting of acute pancreatitis, the specificity can be as low as 50%, as it is known that inflammatory tissue can also demonstrate FDG activity[[47](#_ENREF_47)].

***Complications of chronic pancreatitis***

The most common non-neoplastic complications of chronic pancreatitis include pseudocysts, pseudoaneurysms (due to erosion of the arterial wall), splenic vein thrombosis with subsequent development of collaterals, biliary obstruction (due to pseudocysts), and gastrointestinal complications like gastric outlet obstruction or bowel ischemia[[6](#_ENREF_6),[55](#_ENREF_55)]. These complications are well depicted with CT and MRI.

MRI with MRCP may be superior to CT in detecting specific complications like pseudocysts, fistula formation, distal common biliary dilatation and vascular complications associated with higher morbidity and mortality[[46](#_ENREF_46)].

***Special types of chronic pancreatitis******autoimmune pancreatitis***

Autoimmune pancreatitis is a distinct form of pancreatitis characterized clinically by obstructive jaundice (with or without pancreatic mass), histologically by a lymphoplasmacytic inﬁltrate and ﬁbrosis and therapeutically by a dramatic response to steroids[[56](#_ENREF_56)].

Autoimmune pancreatitis accounts for 2%-6% of chronic pancreatitis[[57](#_ENREF_57),[58](#_ENREF_58)]. It is associated with other autoimmune disorders like Sjogren’s syndrome, primary biliary cirrhosis, and primary sclerosing cholangitis[[59](#_ENREF_59),[60](#_ENREF_60)]. Early diagnosis of autoimmune pancreatitis is crucial as it often responds to steroid therapy; thus avoiding complications.

In AIP, affected areas appear enlarged and hypodense on CT. CECT demonstrates diminished enhancement of the involved parenchyma on the late arterial phase and delayed enhancement on the delayed phase (Figure 19). The MR appearance of autoimmune pancreatitis is similar and is characterized by enlarged pancreas with moderately decreased signal intensity on T1-weighted images, mildly high signal intensity on T2-weighted images and delayed post-gadolinium enhancement of the pancreatic parenchyma (Figure 20). Additional ﬁndings that may be observed in autoimmune pancreatitis include: (1) capsule like rim surrounding the diseased parenchyma, that is hypointense on T2-weighted images and may show delayed post-gadolinium enhancement[[59](#_ENREF_59)]; (2) absence of parenchymal atrophy; (3) ductal dilatation proximal to the site of stenosis; (4) absence of peripancreatic ﬂuid; and 5) clear demarcation of the abnormality[[60](#_ENREF_60)].

MRCP depicts diffuse or segmental narrowing and irregularity of the main pancreatic duct as characteristic findings. The most commonly involved segment is the intrapancreatic common bile duct, and less frequently multifocal intrahepatic biliary strictures are noted.

Autoimmune pancreatitis is has 3 types based on morphologic patterns: diffuse, focal, and multifocal. Diffuse disease is the most common type. CT and MRI commonly show a swollen, sausage-like pancreas with poorly demonstrated borders and a capsule-like rim of low-density/intensity[[61](#_ENREF_61)].

The diffuse form of AIP may mimic diffuse disorders like lymphoma, metastases or other diffuse infiltrative processes. In most of these disorders, unlike AIP, the parenchyma is heterogeneous and shows irregular contours.

Focal disease is less common and manifests as a well-defined hypodense mass, often involving the head and mimicking pancreatic adenocarcinoma. In patients who underwent pancreatic resection for suspected malignancy, 2.5%–8% were ultimately diagnosed with AIP without malignancy[[58](#_ENREF_58),[62](#_ENREF_62)]. However, the probability of AIP *vs* pancreatic cancer in patients with obstructive jaundice can be predicted based on CT/MRI ﬁndings.

Diffusely enlarged pancreas showing low density mass with enhancement on delayed phases on CT/MRI, especially with a capsule-like rim, and no pancreatic ductal cutoff is highly likely to have AIP. Low-density mass on CECT, pancreatic ductal cutoff in presence or absence of pancreatic atrophy mostly suggests pancreatic cancer.

***Groove/paraduodenal pancreatitis***

Groove pancreatitis is a rare form of focal chronic pancreatitis involving the anatomic groove between the pancreatic head, duodenum and common bile duct. Groove pancreatitis is categorized into 2 forms: pure, involving exclusively the groove; and segmental, involving the groove and extending in to the pancreatic head[[63](#_ENREF_63)] (Figure 21).

Pathogenesis remains controversial but may result from obstruction of the accessory pancreatic duct as it drains into the second portion of the duodenum through the minor ampulla[[64](#_ENREF_64)]. Presence of cystic changes, frequently located in the expected region of the pancreatic accessory duct, is considered a prominent feature of this process, likely related to accessory duct obstruction[[65](#_ENREF_65)]. It is commonly seen in patients with history of alcohol abuse[[64](#_ENREF_64)].

The classic MDCT features in the pure form can range from ill-defined fat stranding to frank soft tissue within the pancreaticoduodenal groove with increased delayed enhancement due to fibrosis. Thickening of medial duodenal wall on coronal images and presence of cysts can be appreciated sometimes[[66](#_ENREF_66)]. On MRI, groove pancreatitis is characterized by a sheet-like mass in the groove that shows low signal on T1-weighted images, slightly high signal on T2-weighted images relative to the pancreas and may show delayed enhancement. Cystic lesions are well shown on T2-weighted images in the groove or duodenal wall[[63](#_ENREF_63)].

It may be challenging to differentiate groove pancreatitis from pancreatic head duct adenocarcinoma. Recently, it was shown that by using three strict diagnostic criteria for groove pancreatitis: (1) focal thickening of the second portion of the duodenum; (2) abnormal increased enhancement of the second portion of the duodenum; and (3) cystic changes in the region of the pancreatic accessory duct, distinction from pancreatic duct adenocarcinoma could be achieved with high diagnostic accuracy (87.2% of patients), and a diagnosis of cancer could be excluded with a negative predictive value of 92.9%[[67](#_ENREF_67)].

***Hereditary pancreatitis***

Hereditary pancreatitis is an autosomal dominant disease presenting as multiple episodes of pancreatitis in the absence of any predisposing factors. Imaging findings include parenchymal and intraductal calcifications and parenchymal atrophy. However, in hereditary pancreatitis, imaging plays an important role to rule out structural causes of pancreatitis and to closely monitor the development of pancreatic cancer, the risk of which is increased by many folds in these patients.

**CONCLUSION**

In summary, imaging plays an important role in the diagnosis and staging of acute and chronic pancreatitis. Both CT and MRI are widely used and represent the best cross sectional techniques in the setting of pancreatitis. Wider availability and good image quality make CT the mostly used imaging technique; however, due to its nonionizing nature, unmatched soft tissue contrast and higher safety profile of intravascular contrast media make MRI particularly valuable in pregnant patients, patients with recurrent pancreatitis and patients requiring multiple follow up examinations. Also, early form of chronic pancreatitis and some specific types of chronic pancreatitis benefit from being imaged with MRI.

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**Table 1 Indications to perform contrast-enhanced computed tomography**[[58](#_ENREF_58)]

|  |  |
| --- | --- |
| Types | Indications |
| Initial Imaging | 1 When the diagnosis of acute pancreatitis is uncertain |
|  | 2 Patients with hyperamylasemia, severe clinical pancreatitis, abdominal distention and tenderness, fever > 102°, and leukocytosis for the detection of complications |
| ­ | 3 Ranson score > 3 or APACHE score > 8 |
|  | 4 Patients who fail to improve after 72 h of conservative medical therapy |
|  | 5 Acute change in clinical status, such as new fever, pain, and shock after successful initial medical therapy  1 Acute change in clinical status suggesting complication  2 7–10 d after presentation if CT severity score is 3–10 at presentation or grade |
| Followup Imaging |  |
|  | 3 To determine response to treatment after surgery or interventional radiologic procedures to document response to treatment. |
|  | 4 Before discharge of patients with severe acute pancreatitis |
|  |

**Table 2 MCTSI scoring ystem**[[38](#_ENREF_38)]

|  |  |  |
| --- | --- | --- |
| Prognostic Indicators | Characteristics | MCTSI1 |
| Pancreatic inﬂammation | Normal pancreas | 0 |
|  | Pancreatic ± peripancreatic inflammatory changes | 2 |
|  | One or more collection or peripancreatic fat necrosis | 4 |
| Pancreatic necrosis | No necrosis | 0 |
|  | < 30% | 2 |
|  | >30% | 4 |
| Extrapancreatic complications (pleural effusions, ascites, vascular, gastrointestinal, *etc.*) |  | 2 |

1Scores ≥5 are associated with higher morbidity and mortality.

**Table 3 MR severity index scoring system**[[59](#_ENREF_59)]

|  |  |  |
| --- | --- | --- |
| Prognostic Indicators | Characteristics | MRSI |
| Pancreatic inﬂammation | Normal pancreas | 0 |
|  | Focal or diffuse enlargement of the pancreas | 1 |
|  | Intrinsic pancreatic abnormalities with inﬂammatory Changes in the Peripancreatic fat | 2 |
|  | Single, poorly defined fluid collection | 3 |
|  | Two or more poorly deﬁned collection or presence of gas in or adjacent to the pancreas | 4 |
| Pancreatic necrosis | No necrosis | 0 |
|  | < 30% | 2 |
|  | 30%-50% | 4 |
|  | > 50% | 6 |

**Table 4 Imaging criteria for chronic pancreatitis**[[60](#_ENREF_60)]

|  |  |  |
| --- | --- | --- |
|  | CT Criteria | MRI/S-MRCP Criteria |
| Moderate chronic pancreatitis | ≥ 2 of the following:  Main duct enlarged (2–4 mm)  Slight gland enlargement (up to 2 × normal)  Heterogeneous parenchyma  Small cavities (< 10 mm)  Irregular ducts  Focal acute pancreatitis  Increased Density of the main pancreatic duct wall  Irregular head/body contour | Moderate pancreatogram changes  Main duct abnormal and  Abnormal side branches, > 3 |
| Marked chronic pancreatitis | with ≥ 1 of the following  Large cavities (>10 mm)  Gross gland enlargement (2 × normal)  Intraductal filling defects or pancreatic calculi  Duct obstruction, stricture, or gross irregularity  Contiguous organ invasion | Main duct abnormal and  Abnormal side branches, > 3  Plus one or more of the following:  Large cavity  Obstruction  Filling defects  Severe dilatation or irregularity |

**Table 5** **Differentiating imaging features between chronic pancreatitis and pancreatic adenocarcinoma**

|  |  |
| --- | --- |
| Chronic pancreatitis | Pancreatic adenocarcinoma |
| Preserved glandular, feathery or marbled texture similar to that of the remaining pancreas | Deﬁnable, circumscribed mass lesion is most often diagnostic for tumor, which disrupts the underlying architecture and results in loss of anatomic detail. |
| Heterogeneous pancreatic enhancement with presence of signal void (cysts and calciﬁcations) on immediate post-gadolinium images. | Irregular, heterogeneous, diminished enhancement on postgadolinium images compared to adjacent pancreatic parenchyma. |
| Irregular dilatation of main pancreatic duct with gradual narrowing. | Abrupt cut off of the pancreatic duct with significant proximal dilatation +/- presence of double duct sign. |
| Presence of multiple intraductal calcifications (the most specific finding). | Very few ductal calculi compared to chronic pancreatitis. |
| Dilatation of main pancreatic duct with and ectasia of the side branches, giving chain of lakes appearance. | Minimal dilatation of side branches. |
| No vascular encasement, significant lymphadenopathy or distant metastasis. | Vascular encasement, lymphadenopathy or distant metastasis. |

**Figure 1 Normal pancreatic appearance on magnetic resonance imaging.** **A:** Axial T2-weighted image with fat-suppression; B**:** Axial GRE out-of-phase T1-weighted image; **Cl** Axial post-contrast 3D-GRE T1-weighted image with fat-suppression during the late arterial phase. The pancreas demonstrates low T2 signal intensity (A) and high T1 signal intensity on pre-contrast images (B), reflecting high protein content of the exocrine gland. The pancreas demonstrates avid homogenous enhancement on immediate post-contrast images (C), reﬂecting a normal capillary blush.

**Figure 2 Normal pancreatic duct anatomy and pancreatic divisum.** (A and C) Coronal and (B and D) axial post-processed maximum intensity projection 3D-MRCP images from two different patients. In the first patient, the main pancreatic duct courses inferiorly (A) and posteriorly (B), joins the CBD and opens in the major papilla in keeping with normal pancreatic duct anatomy. In the second patient, the main pancreatic duct continues its course superiorly (C) and anteriorly (D), crosses the CBD and opens in the minor papilla in keeping with pancreatic divisum.

**Figure 3 Focal acute edematous pancreatic tail pancreatitis.** **A-C:** Axial CT scan of the pancreas during the late arterial phase. There is evidence of ill-definition and reduced enhancement of the pancreatic tail (A-B), associated with mild peripancreatic fatty stranding extending to the anterior left paranephric space in keeping with focal acute edematous pancreatitis.

**Figure 4 Gallstone acute edematous pancreatic tail pancreatitis.** **A:** Axial fast spin-echo (FSE) T2-weighted image with fat-suppression; **B-C:** Post-contrast 3D-GRE T1-weighted images with fat-suppression during the late arterial and portal venous phases. There is mild diffuse lace-like increased T2 signal involving the pancreatic parenchyma, associated with a small amount of peripancreatic fluid near the pancreatic tail (A). The pancreas demonstrates diffuse minimal decrease in T1 signal intensity (B) and minimally reduced enhancement on the late arterial phase (C) in keeping with diffuse edematous pancreatitis. There are also innumerable gallstones (A).

**Figure 5 Subtle focal acute edematous pancreatic tail pancreatitis.** **A:** Axial T2 weighted-image with fat-suppression; **B-C:** Axial post-contrast 3D-GRE T1- weighted images with fat-suppression during the late arterial and portal venous phases. There is a very subtle area of increased T2 signal seen around the pancreatic tail (arrow, A), with fairly normal enhancement of the pancreas on the post-contrast images (B-C) in keeping with subtle focal acute edematous pancreatic tail pancreatitis.

**Figure 6 Focal pancreatic head necrotizing pancreatitis confined to the pancreatic parenchyma.** A-B**:** Axial CT scan during the portal venous phase. There is evidence of significantly reduced enhancement of the pancreatic head (B), without peripancreatic extension or necrosis in keeping with focal acute necrotizing pancreatitis.

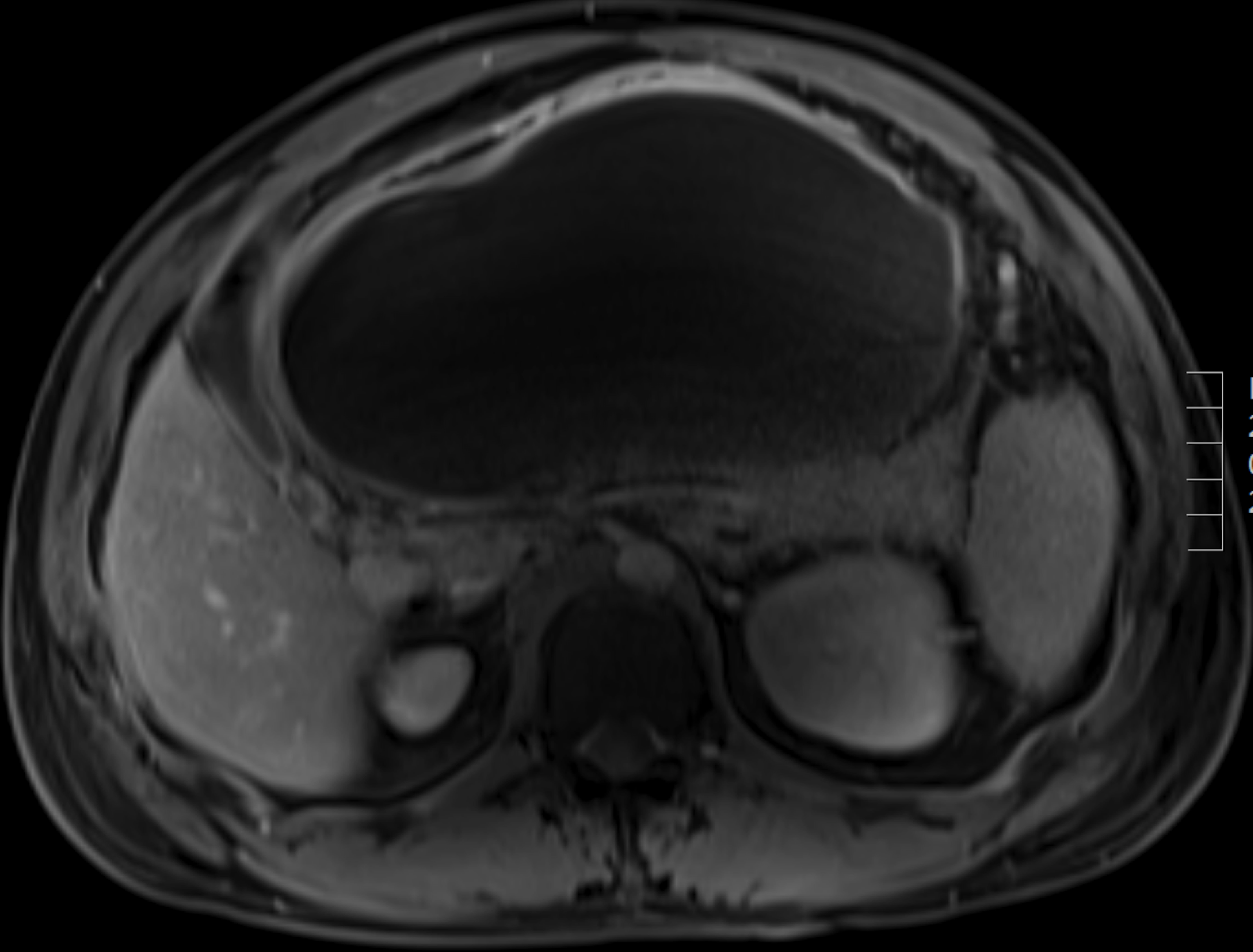
**Figure 7 Severe acute necrotizing pancreatitis and peripancreatitis.** A-B: Axial CT scan during the late arterial phase; C-D:Coronal reformatted CT images. There is evidence of lack of arterial enhancement involving the pancreatic body and tail, which are replaced by necrotic tissue, associated with heterogenous peripancreatic tissue inflammation and necrosis extending to left paranephric space (A-B) and paracolic gutter (C-D), in keeping with severe necrotizing pancreatitis and peripancreatitis. There is also evidence of splenic vein thrombosis (arrow, A, C), a known complication of acute pancreatitis.

**Figure 8 Focal acute necrotizing pancreatitis.** A: Axial fast spin-echo T2- weighted image with fat-suppression; B: Axial post-contrast 3D-GRE T1- weighted images with fat-suppression during the venous phase. There is a focal area of low T2 signal involving the proximal part of the pancreatic tail, associated with minimal peripancreatic fat stranding (A). This focal area demonstrates significantly reduced enhancement on the post-contrast images, in keeping with focal necrotizing pancreatitis.

**Figure 9 Acute interstitial edematous pancreatitis and acute peripancreatic fluid collections.** A-B: Axial CT scan during the portal venous phase. The pancreas is mildly thickened and demonstrates mildly heterogenous enhancement, reflective of edema, in keeping with acute interstitial edematous pancreatitis. There is a peripancreatic fluid with imperceptible wall in keeping with acute peripancreatic fluid collections.

**Figure 10 Peripancreatic fluid secondary to multifocal acute necrotizing pancreatitis.** **A-C:** Axial CT images during the late arterial phase. There are two areas of focal necrosis involving the pancreatic body (B) and pancreatic head/uncinate process (C), associated with loculated peripancreatic fluid collection (A).

**Figure 11 Large pancreatic pseudocyst.** A-B:Axial CT scan during the late arterial phase. There is a large oval shaped pancreatic pseudocyst located anterior to the pancreatic body and tail, associated with mass effect on the thinned out pancreatic tissue in keeping with a large pancreatic pseudocyst.



**Figure 12 Large pancreatic pseudocyst.** A**:** Axial fast spin-echo T2- weighted image with fat-suppression; B-C**:** Axial and coronal post-contrast 3D- GRE T1-weighted images with fat-suppression during the portal venous phase. There is a very large thin-walled cyst (A) within the lesser sac; which demonstrates mild uniform wall enhancement (B-C) in keeping with a large pancreatic pseudocyst. The central drop of signal on (A) is related to dielectric shading artifact.

**Figure 13 Acute necrotic collection.** Axial T2 single-shot turbo spin-echo image. There is a well-defined fluid collection involving the pancreatic neck with peripancreatic extension and communication with the main pancreatic duct. This collection demonstrates well-defined outlines and heterogenous low T2 signal intensity debris within it in keeping with acute necrotic collection. Multiple gallstones are also noted.

**Figure 14 Necrotizing pancreatitis, with peripancreatic walled-off necrosis.** A:Axial fast spin-echo T2-weighted image with fat-suppression; B-C:Axial post-contrast 3D-GRE T1-weighted images with fat-suppression during the late arterial and venous phase. There is a focal area of heterogeneous iso to slightly high T2 signal involving the pancreatic body-tail junction (A); which demonstrates lack of enhancement on the post-contrast images (B-C). There is associated sizable peripancreatic fluid collection; which demonstrates heterogeneous T2 signal intensity and thick enhancing wall post-contrast in keeping with walled-off necrosis

**Figure 15 Infected peripancreatic fluid in a patient with acute pancreatitis.** A-C:Axial CT scan during the portal venous phase. There are a few gas bubbles seen within a small peripancreatic fluid (arrows, A-C). In the absence of any intervention in keeping with infected peripancreatic fluid.

**Figure 16 Pancreatic divisum, with a small Santorinicele.** **A:** Coronal 3D- maximum intensity projection MRCP image before administration of secretin; B-F:Selected dynamic secretin thick-slab MRCP images obtained at 30 s (B), 60 s (C), 120 s (D), 4 mins (E) and 9 min (F). Prior to administration of secretin, it is difficult to identify the main pancreatic duct (A). After administration of secretin, there is better delineation of the main pancreatic duct (C), with demonstration of pancreatic divisum. There is also enlargement of the accessory pancreatic duct, with demonstration of a small santorinicele (B, F). S-MRCP allows qualitative and quantitative assessment of pancreatic exocrine secretions. In this case, the pancreatic flow output was considered within normal limits; excluding early chronic pancreatitis.

**Figure 17 Chronic pancreatitis with pancreatic parenchymal calcifications and pancreatic duct stones.** A, B:Axial CT scan during the late arterial phase; C, D:Axial T2 single-shot fast spin-echo images; E:Coronal 3D- Cholangiopancreatogram (MRCP) image. CT shows a markedly dilated and tortuous main pancreatic duct (MPD) (A, B), with foci of thick calcification involving the pancreatic head and uncinate process parenchyma (B). Large the proximal MPD stone was suspected on CT (arrow, B). MRCP shows gross pancreatic ductal dilatation with confirmation of the distal intraductal calculus (arrow, D), and shows an additional mid-pancreatic duct stone, not clearly seen on CT (arrow, C). No pancreatic masses or ductal anomalies are identified.

**Figure 18 Chronic pancreatitis.** A: Axial T1-weighted GRE MRI. B: Coronal-oblique thick-slab MRCP image. There is evidence of diffuse thinning of the pancreatic parenchyma with uniform dilatation of the pancreatic duct and prominence of the pancreatic duct side-branches (A-B), associated with multiple tiny stones at the proximal pancreatic duct (arrows, B) in keeping with chronic pancreatitis. There is also mild uniform dilatation of the CBD, which tapers down to the level of the pancreatic duct (B).

**Figure 19 Autoimmune pancreatitis.** A, B: Axial CT scan during the late arterial phase. There is evidence of diffuse pancreatic swelling with loss of the normal pancreatic lobulations, obliteration of the pancreatic duct and subtle low attenuating peripancreatic rim (A, B) in keeping with autoimmune pancreatitis. Patient had high IgG4 level (> 0.500 g/L).

**Figure 20 Autoimmune pancreatitis.** A: GRE T1–weighted image; B, C: Post- contrast 3D-GRE T1-weighted images with fat-suppression during the late arterial and portal venous phases. There is evidence of diffuse pancreatic swelling with reduced T1 signal, loss of the normal pancreatic lobulations and obliteration of the pancreatic duct, associated with a rim of low T1 signal (A). The pancreas demonstrates diffuse reduced enhancement on the late arterial phase and progression of enhancement on the portal venous phase in keeping with autoimmune pancreatitis. The patient had significant biliary tree irregularities in keeping with primary sclerosing cholangitis (not shown). Additionally, there are a few bilateral wedge-shaped areas of renal hypo- enhancement in keeping with segmental infarcts.

**Figure 21 Groove pancreatitis.** A: Axial T2-weighted single-shot fast spin-echo (SS-FSE) images with fat-suppression; (B) Pre- and (C, D) Post-contrast 3D-GRE T1- weighted images with fat-suppression during the late arterial and portal venous phases. There is a slightly low T2 signal sheet-like mass in the pancreaticoduodenal groove, with tiny cystic changes (arrow, A). The mass shows low T1 signal with extension into the pancreatic head (arrow, B). Imperceptible enhancement is depicted on the immediate post-contrast image (arrow, C), with progressive enhancement on the subsequent delayed images (D) in keeping with groove pancreatitis.