

MDCT Imaging Features of Acute and Chronic Pancreatitis with its Complications: A Pictorial Review

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ABSTRACT

The incidence of Acute Pancreatitis (AP) was 30.0 per 100,000 population overall. The two most common aetiological factors of AP are gallstones and alcohol abuse, while other causes such as metabolic, iatrogenic, vascular, infections, and toxic factors are less important and less common. Two laboratory tests, serum amylase and lipase levels, which have relatively good sensitivity and specificity, complement imaging in the diagnosis and treatment of some cases of AP. Imaging helps confirm the clinical diagnosis, determine the cause, grade the extent of AP, and assess its severity, in addition to clinical symptoms and laboratory studies. Chronic Pancreatitis (CP) is characterised by continuous inflammatory and fibrotic changes in the pancreas, eventually resulting in exocrine and endocrine dysfunction. Alcoholism is a factor in 70-90% of CP cases. Other causes include chronic ulcerative colitis, Sjogren's syndrome, primary sclerosing cholangitis, etc. Early diagnosis of CP is challenging, as biochemical studies do not provide definitive diagnosis in the early stages. Imaging, mainly Contrast Enhanced Computed Tomography (CECT) and Magnetic Resonance Imaging (MRI), aids in the definitive diagnosis. The present pictorial article aimed to provide an image-rich overview of the morphological features associated with the early-stage and late-stage local consequences of acute and chronic pancreatitis.

Keywords: Aneurysm, Emphysematous, Fat stranding, Fluid collection, Multidetector computed tomography, Necrosis, Oedema, Pancreas, Thrombosis

INTRODUCTION

Pancreatitis is an inflammatory condition that refers to the autodigestion of the pancreas, where pancreatic enzymes damage pancreatic tissue, leading to dysfunction of the gland and potential damage to distant organs and systems. Pancreatitis can be either acute or chronic.

ACUTE PANCREATITIS (AP)

Acute Pancreatitis is an acute inflammatory condition that develops when fluid containing activated proteolytic enzymes exudes from the pancreatic interstitium and leaks into the surrounding tissue [1].

The AP is classified as mild or severe, based on the presence of local complications and organ failure. Morphologically, AP can be of two types, including acute oedematous or interstitial pancreatitis and acute Necrotising Pancreatitis (NP). Acute oedematous or interstitial pancreatitis comprises pancreatic oedema and inflammation, often having a modest clinical course and a self-limiting process. It has fewer local complications. Its pathological hallmarks are interstitial oedema and sporadic tiny regions of parenchymal necrosis [2]. A much more severe form of pancreatitis known as NP is marked by significant fat necrosis, bleeding, and necrotic liquefaction of the pancreas [3]. It is associated with local complications such as pseudocysts, Walled-off Necrosis (WON), and organ failure. Overall, the mortality associated with severe AP ranges from 10% to 30% [3].

According to the updated Atlanta classification, to diagnose AP, two or more of the following criteria must be satisfied [4]:

- Abdominal pain suggestive of pancreatitis;
- A serum amylase or lipase level that is three times higher than normal; or
- Distinctive imaging findings, for which the most common modality used is CECT.

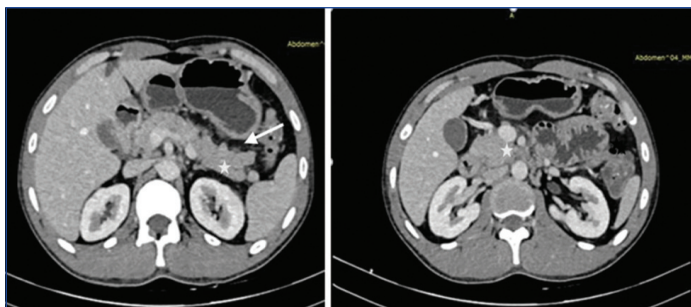
Imaging Technique-Computed Tomography

The CECT is the most commonly used modality in the evaluation of AP. Guidelines for selecting the most appropriate criteria were created by the American College of Radiology (ACR) committee on appropriateness criteria and its expert panels in 2010 [5]. CECT has received high score ratings in a variety of clinical settings when compared to imaging exams for the diagnosis and treatment of AP [5,6]. The CT identifies peripancreatic involvement and determines the presence and degree of necrosis. Additionally, using CT to guide percutaneous aspiration and drainage treatments related to pancreatic necrosis is a great concept. An ideal predictive approach should be accurate, simple to use, widely accessible, and have minimal interobserver variability to distinguish between patients with mild and severe pancreatitis.

Hence, the CT severity index was introduced, focusing on the presence and degree of pancreatic inflammation and necrosis. However, this index had a few drawbacks, including the lack of a discernible relationship between the index score and the later occurrence of organ failure, extra pancreatic parenchymal problems, or peripancreatic vascular complications [7]. As a result, the modified CT severity index was introduced to predict clinical outcomes more accurately.

Interstitial Oedematous Pancreatitis versus Acute Necrotising Pancreatitis (NP)

Interstitial oedematous pancreatitis is more common and represents the non necrotising form of AP. On contrast study, it shows diffuse or focal involvement, with focal involvement most commonly seen in the head or tail of the pancreas, occurring in 20% of cases [Table/Fig-1,2]. The pancreas appears mildly heterogeneous with a loss of lobulated outline, and non enhancing areas are not seen in the parenchyma. Inflammatory changes are noted in the form of fat stranding or mesenteric haziness and hypodensity in the



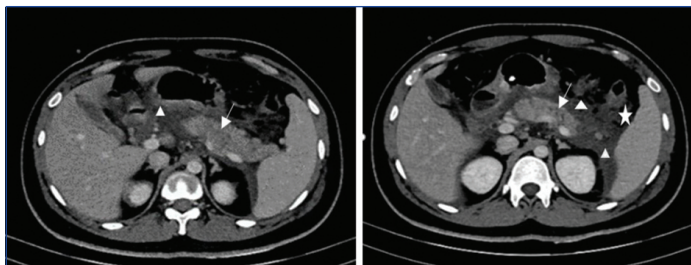
[Table/Fig-1,2]: A case of mild acute interstitial pancreatitis of 40-year-old male. Contrast enhanced axial CT images shows bulky head and tail of pancreas (white star), with homogenous enhancement of pancreas and mild hypodense areas within the peripancreatic fat which is suggestive of peripancreatic fat stranding noted (white arrow). (Images from left to right)

peripancreatic fat. There may be associated fluid, shown as ill-defined homogeneous low-attenuating areas [8]. Most patients usually resolve on their own without complications.

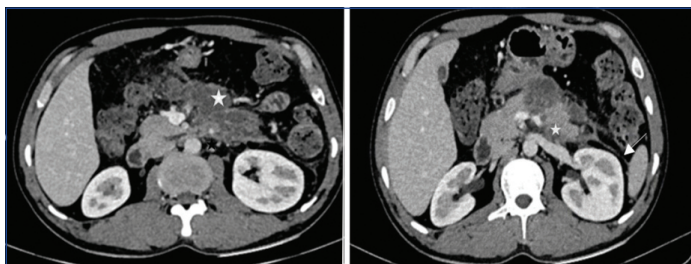
About 20% to 30% of instances of AP are caused by NP [9]. It's critical to realise that necrosis can occur in either the peripancreatic tissues or the pancreatic parenchyma. The scan should ideally be done five to seven days after the onset of symptoms.

The NP is subdivided into three subtypes based on CECT according to the new Atlanta classification [4]:

- Combined pancreatic necrosis and peripancreatic necrosis, which is the most common, seen in around 75% of all NP [Table/Fig-3,4];
- Peripancreatic tissue necrosis alone, which has a peripancreatic necrotic collection with normal homogeneous enhancement of the pancreas (less common, with an incidence of approximately 20%) [Table/Fig-5,6];
- Pancreatic parenchymal necrosis alone (rare, with an incidence of only 5%).

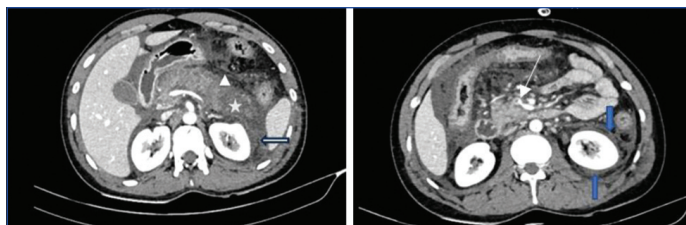


[Table/Fig-3,4]: A case of severe acute NP with acute necrotic collection. Contrast enhanced axial CT images shows bulky body and tail of pancreas with few non enhancing areas (white arrows) less than 30% necrosis in body and tail of pancreas. Significant peripancreatic fat stranding (white star) with ill-defined heterogeneous hypodense fluid noted in peripancreatic, splenic hilar regions and along greater curvature of stomach (white arrow heads) suggestive of acute necrotic collection. (Images from left to right)



[Table/Fig-5,6]: A case of acute moderate pancreatitis with acute necrotic collection. Contrast enhanced axial CT images shows pancreatic head and bulky body with significant peripancreatic fat stranding and an ill-defined heterogeneous hypodense collection was noted adjacent to body of pancreas (white stars) with thickening of anterior conal fascia (white arrow). (Images from left to right)

The extent of necrosis can be stratified as <30% [Table/Fig-3,4], 30-50% [Table/Fig-7,8], and >50% to grade the severity of pancreatitis. As the extent of necrosis increases, the chance of extra pancreatic complications increases.

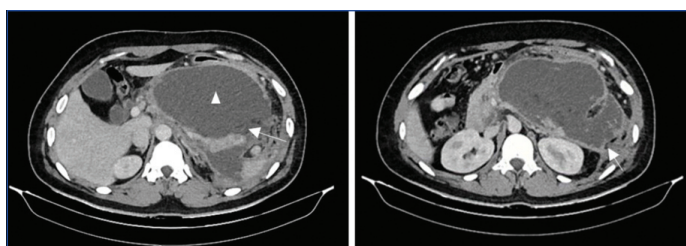


[Table/Fig-7,8]: A case of severe AP with acute necrotic collection and vascular complications. Contrast enhanced axial CT images shows bulky pancreas with heterogeneous enhancement and multiple non enhancing areas noted in body and tail of pancreas (s/o necrosis >50%) (white star) significant peripancreatic fat (white arrow heads) stranding with moderate peripancreatic fluid noted tracking along left perisplenic region (short white arrow with blue outline) and left pararenal space with thickening of anterior and posterior pararenal fascia (blue arrows). Focal eccentric thrombosis of Superior Mesenteric Vein (SMV) noted (white arrow) however splenic vein was normal. (Images from left to right)

Acute Peripancreatic Fluid Collections (APFCs) and Pseudocyst

The APFCs and acute necrotic collections are the current diagnostic names for local complications in the early stages of Acute Pancreatitis (ANCs). APFCs only appear in patients with Interstitial Oedematous Pancreatitis (IEP) during the first four weeks of treatment. APFCs solely contain fluid and are seen as homogeneous fluid-attenuation collections without a wall that tends to conform to the retroperitoneal spaces since the pathophysiology involves inflammation without necrosis. The most common sites are the lesser sac and anterior pararenal space. APFCs are always found in the peripancreatic region. Since most APFCs resolve on their own, drainage should not be done to avoid the danger of contaminating an otherwise sterile collection [9].

If an APFC does not resolve within four weeks, it organises further and develops a capsule, which appears as an enhancing wall at CECT and contains only fluid without necrosis. At this time, the mass is referred to as a pseudocyst, which is a well-defined peripancreatic fluid accumulation surrounded by fibrous or granulation tissue [Table/Fig-9,10]. About 50% of persistent pseudocysts develop clinical symptoms or consequences, which might include secondary infection, discomfort, haemorrhage due to erosion into nearby arteries, decompression or rupture, or localised mass effect. They typically resolve on their own. The diagnosis is not pseudocyst but WON, if there is even a modest area of fat or soft tissue attenuation in an otherwise fluid-attenuation collection [4].



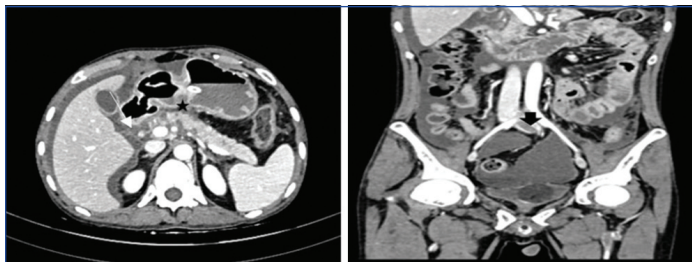
[Table/Fig-9,10]: A 48-year-old male who was a known case of AP, now presented with intrapancreatic pseudocyst. Contrast enhanced axial CT image shows a large well-defined, thick-walled, peripherally enhancing hypodense homogeneous cystic lesion in lesser sac (white arrow head), replacing most of the body and tail of pancreas- suggestive of pseudocyst. A focal breach was noted in its posterior wall with the collection noted tracking into the anterior pararenal space on left-side (white arrows). (Images from left to right)

Acute Necrotic Collection and Walled-off Necrosis (WON)

Acute necrotic collections, on the other hand, have the following characteristics: they occur only in NP [Table/Fig-3,4]. They can be located in the peripancreatic and pancreatic regions, surrounding the pancreas or with intrapancreatic extension or both. They have a heterogeneous appearance due to containing variable amounts of necrotic material [3].

Acute necrotic collections often extend beyond the boundaries of interfascial planes and can affect retroperitoneal spaces, interfascial planes, subperitoneal spaces, and other abdominal spaces [10]. They are most commonly found in the lesser sac and the anterior

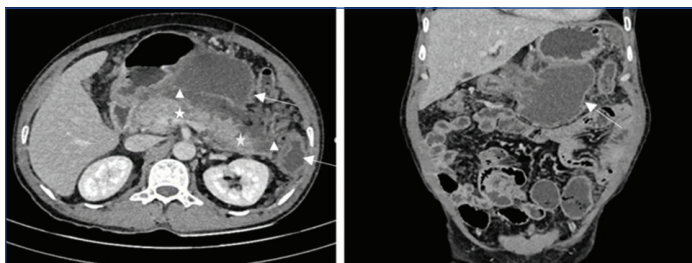
pararenal spaces, followed by the transverse mesocolon, mesenteric root, and then the gastrohepatic [Table/Fig-11,12], gastrosplenic, and gastrocolic ligaments. These collections may also involve remote areas, such as the mediastinum and sidewalls of the pelvis.



[Table/Fig-11]: A case of moderate severe pancreatitis with vascular complication. Contrast-enhanced axial and coronal CT images shows inhomogeneous enhancement of the pancreas, with multiple small non enhancing areas within (s/o necrosis- <30%) the head and body of pancreas (black star) with moderate peripancreatic fluid tracking along right subhepatic space along gastrohepatic ligament, extending into right paracolic gutter and pelvis (white arrows).

[Table/Fig-12]: Shows partial non opacification of left common iliac vein proximal to joining of Inferior Vena Cava (IVC) (black arrow). (Images from left to right)

The WON occurs more than four weeks after acute NP. WON appears inhomogeneous and contains non liquefied components encapsulated with a well-defined wall. The WON can be intrapancreatic or extra-pancreatic [Table/Fig-13,14].

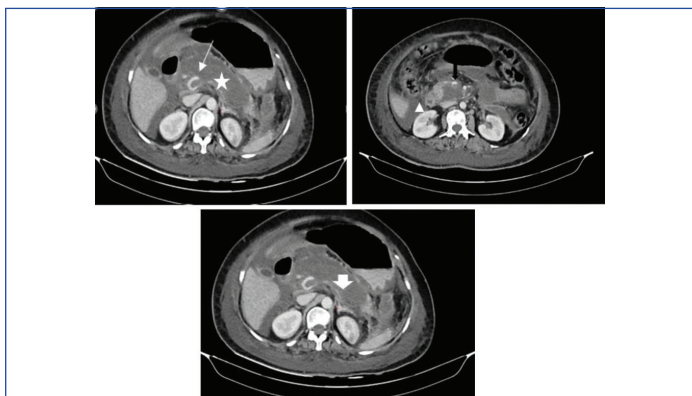


[Table/Fig-13,14]: A case of severe acute NP with WON shows heterogeneous enhancement of pancreas within in head and body of pancreas and shows few non enhancing areas within (white star) (s/o necrosis <30%) and mild peripancreatic fluid noted along body and tail region of pancreas (white arrow heads). A well-defined thin walled peripherally enhancing heterogeneous cystic lesion noted in lesser sac and left pararenal space- s/o WON (white arrows). (Images from left to right)

Complications

In addition to collections, complications such as vascular issues may also occur. Patients most frequently experience splenic vein thrombosis, which is a vascular issue in patients with AP. Vascular complications occur in a quarter of patients with AP and can cause substantial morbidity and mortality [11].

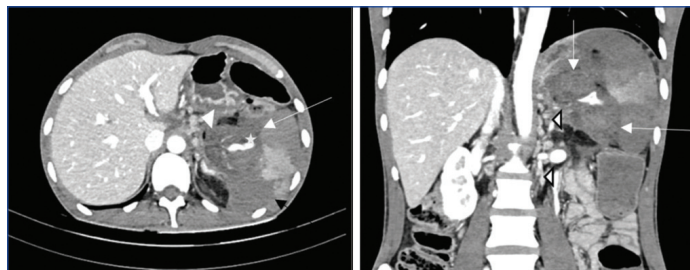
Venous thrombosis: The most typical vascular consequence is splanchnic vein thrombosis, which may be caused by inflammatory responses, reduced venous flow, and mass effect by adjacent necrotic tissue on vascular structures [Table/Fig-15-17]. In later stages, it may lead to splenomegaly and the formation of collaterals [12]. Isolated



[Table/Fig-15-17]: Shows extensive NP (white star) with acute necrotic collection involving the peripancreatic region, pararenal space (white arrow head) with concentric thrombosis of portal (long white arrow) and superior mesenteric vein (thick black arrow), in [Table/Fig-17] splenic vein is not visualised-likely thrombosed (short thick white arrow). (Images from left to right)

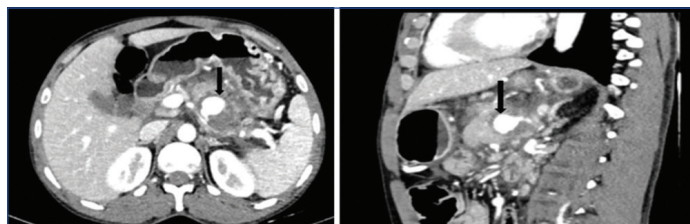
superior mesenteric vein involvement is very uncommon and usually occurs together with splenic vein thrombosis.

Haemorrhage: Secondly, the splenic artery, portal vein, and other peripancreatic veins are the most frequent sources of bleeding in AP because the release of pancreatic enzymes causes erosion of the surrounding vasculature, which may result in pseudoaneurysm formation and spontaneous haemorrhage [Table/Fig-18,19] [12].



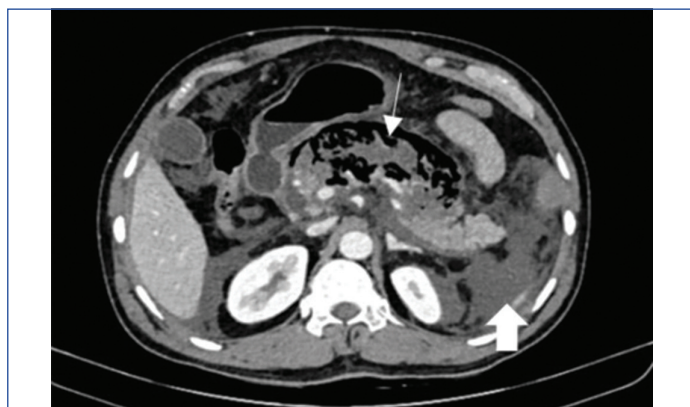
[Table/Fig-18,19]: A 37-year-old male who was a known case of acute NP contrast-enhanced axial and coronal images show few fairly defined non enhancing cystic lesions with areas of blood attenuation in peripancreatic and perisplenic space along lesser sac (white arrows) with a segment of the splenic artery (white star) encased by the mass-like erosion of splenic artery into pseudocysts. Multiple collaterals noted in peripancreatic region (white arrow heads). (Images from left to right)

Pseudoaneurysm: The splenic artery is the most common artery involved in pseudoaneurysm formation [Table/Fig-20,21]. It is a late and potentially fatal complication. Pseudoaneurysms can breach into the pancreatic parenchyma, the peritoneum, the gastrointestinal system, or the necrotic collection [13].



[Table/Fig-20,21]: A case of acute NP, contrast enhanced axial and sagittal CT images showing focal saccular aneurysmal outpouching noted arising from mid part of splenic artery- s/o Pseudoaneurysm (black arrow). (Images from left to right)

Infection: About 20% of patients with NP experience infection as a complication, which is thought to be caused by bacterial translocation from the gut to nearby necrotic pancreatic tissue. Although gas is found in only a small percentage of cases of proven infection, the presence of gas during imaging indicates infection [Table/Fig-22] [12].



[Table/Fig-22]: A case of severe acute NP. Contrast-enhanced CT image shows a bulky pancreas with extensive ill-defined non enhancing area and multiple pockets of air (thin white arrow) noted involving the head, uncinate process and the body regions (>60%). Significant peripancreatic fat stranding with mild necrotic fluid collection was noted (thick white arrow). Thickening of anterior and posterior pararenal fascia's noted bilaterally with reactive perinephric fat stranding.

Inflammation and mass effect on surrounding organs: The NP can displace and compress nearby organs due to the inflammatory changes and collections it causes. Complications are noted in the form of bowel obstruction, hydronephrosis, and inflammatory

changes in the form of inflammatory thickening and adjacent fat stranding [12].

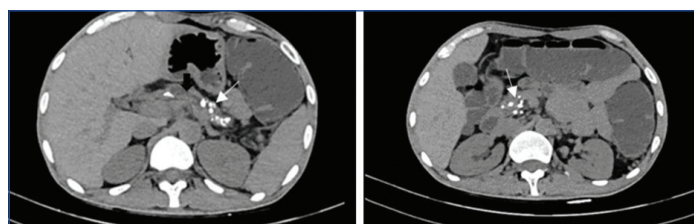
CHRONIC PANCREATITIS (CP)

The CP is a progressive, irreversible inflammatory disease of the pancreas with clinical manifestations of chronic abdominal pain, weight loss, and permanent pancreatic exocrine and endocrine insufficiency [2]. The histologic hallmarks of CP include fibrosis, chronic inflammation, and loss of acinar cells. Malnutrition and diabetes mellitus are the results of advanced-stage destruction of exocrine and endocrine pancreas tissue [14]. In Western nations, 70-90% of cases of CP are caused by alcoholism. Additional significant risk factors include smoking, a high-protein diet, hypercalcaemia, hyperparathyroidism, pancreatic divisum, pancreatic or periampullary neoplasms, and ampullary stricture. Less common causes of CP include hereditary pancreatitis, autoimmune pancreatitis, malnutrition, and chronic renal insufficiency [15]. Idiopathic CP is the term for the 30-40% of CP patients who have no known underlying aetiology [2].

Computed Tomography

The sensitivity of CT in detecting late stages of CP is 60-95% [16]. However, it is limited in identifying early CP. The most common CT findings include ductal dilatation (68%), parenchymal atrophy (54%), and parenchymal and ductal calcification, which is the most specific feature [15].

Depending on the degree of fibrosis or atrophy and the level of enzyme activity, the overall size of the pancreatic gland in CP may be normal, enlarged, or diminished. The presence and number of parenchymal calcifications are independent predictors of the degree of pancreatic fibrosis. Other less common complications include collections, bile duct dilatation, and heterogeneous glandular enhancement [16]. Calcium buildup is a defining feature of chronic inflammation [Table/Fig-23,24]. Pancreatic necrosis and inflammation cause local chemical changes that result in deposits of calcium carbonate and calcium phosphate. The location and volume of the calcium salt deposits can vary. Calcification most frequently starts in the head, but with persistent inflammation, it can spread to the body and tail as well [Table/Fig-23] [17,18].



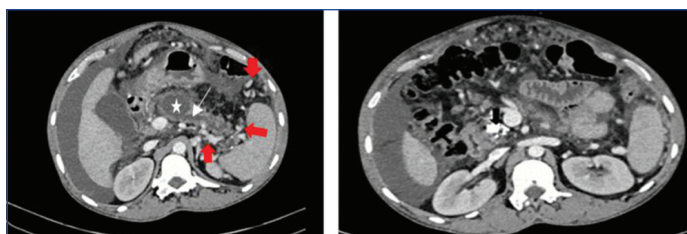
[Table/Fig-23,24]: A case of chronic calcific pancreatitis. Non enhanced axial CT images shows Pancreas is atrophic. Main pancreatic duct is smoothly dilated (black arrow) (maximum calibre is 8 mm in body region). Multiple specks of intraductal and intrapancreatic calcifications noted (white arrows). (Images from left to right)

Compared to calcifications, parenchymal atrophy and ductal dilatation are less distinctive signs because they can also be present in older people. The dilated duct may be smooth, crooked, or beaded [Table/Fig-24,25] [17]. Pleomorphic ductal calculi may appear as sporadic regions of strictures and dilatation in the primary pancreatic duct.

Complications

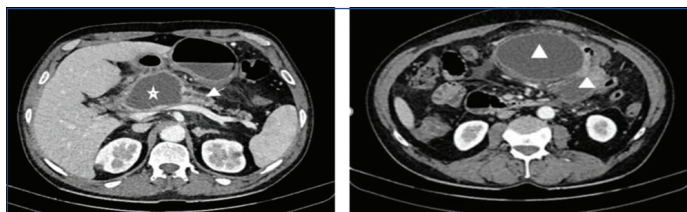
Complications of chronic pancreatitis include pseudoaneurysm, biliary obstruction, venous thrombosis, and pancreatic pleural and peritoneal fistulae [19]. CT is a great tool for locating problems associated with chronic pancreatitis. Fluid collection is one such problem that 30% of people experience. Although distant sites are seldom reported, fluid collections are typically seen inside [Table/Fig-25,26] or close to the pancreas.

Pseudocyst/ Walled-off Necrosis (WON): The majority of fluid accumulations linked to chronic pancreatitis are tightly encapsulated, presenting as pseudocysts or WON. Free fluid near the pancreas is



[Table/Fig-25,26]: A case of acute on CP. Contrast-enhanced axial CT images shows heterogeneous enhancement of pancreas. A well-defined thin walled (maximum wall thickness- 3 mm) non enhancing hypodense lesion (white star) in body region of pancreas communicating with MPD likely pseudocyst. MPD-5 mm in body region, irregularly dilated (white arrow) which is noted communicating with pseudocyst. Few intrapancreatic calcification noted in uncinata process (short black arrow). No e/o intraductal calcifications. Splenic vein is not visualised which is replaced by multiple perisplenic, peripancreatic vascular collaterals (short red arrows). (Images from left to right)

a sign of underlying acute pancreatitis [Table/Fig-25]. Pseudocysts in chronic pancreatitis can communicate with the duct [Table/Fig-25,27,28] or be associated with ductal stricture. These pseudocysts are difficult to treat [14].



[Table/Fig-27,28]: A case of acute on Chronic Pancreatitis (CP) with intrapancreatic pseudocyst contrast enhanced axial CT images shows body and tail of pancreas is atrophic with irregularly dilated MPD noted (short white arrow in [Table/Fig-27]). On post contrast study, mild peripancreatic fluid (short white arrow) with adjacent fat stranding noted with a well-defined peripherally enhancing hypodense thin-walled cystic lesion noted involving head and body of pancreas and lesser sac- s/o pseudocyst (white star) which is noted communicating with MPD. Few pseudo cysts are abutting greater curvature of stomach superiorly and small bowel loops inferiorly and laterally (white arrow heads). (Images from left to right)

(Courtesy Krishna Rajendra Hospital -MMCRI) (Courtesy Krishna Rajendra Hospital -MMCRI)

Venous thrombosis: In vascular complications s/o chronic venous thrombosis, the most common is splenic vein thrombosis, which causes non visualisation of the same and is replaced by collaterals [Table/Fig-29-31]. Gastric and mesenteric varices may form in the presence of left-sided portal hypertension when the splenic vein is affected [2].

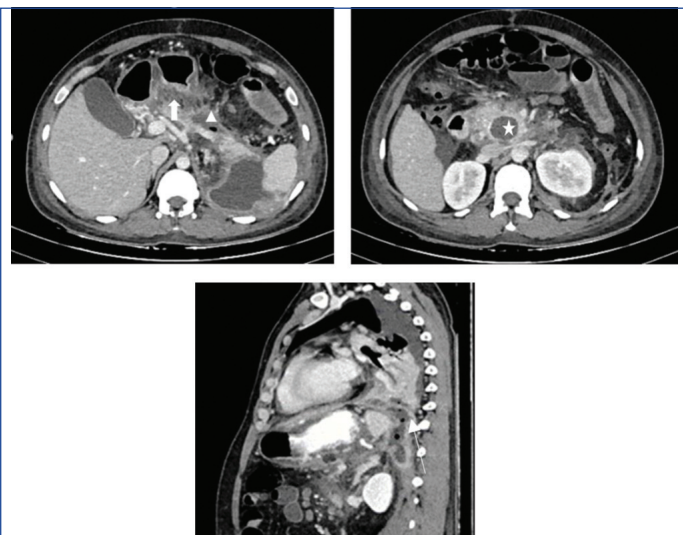


[Table/Fig-29]: A case of chronic calcific pancreatitis with chronic splenic vein thrombosis. Contrast enhanced axial CT image shows multiple specks of intra pancreatic calcification (long white arrows) throughout the pancreas with non visualisation of splenic vein from its origin (s/o thrombosis).

[Table/Fig-30,31] Contrast-enhanced axial and coronal CT image shows multiple non enhancing cystic lesion in pancreas (s/o pseudocyst) (short white arrows). Splenomegaly noted. Splenic vein is not visualised which is replaced by multiple dilated venous collaterals (white stars) noted along perisplenic region periportal, peripancreatic, and along lesser curvature. (Images from left to right)

Pancreatic pleural fistula: Pancreatic pleural fistula is an uncommon complication in chronic pancreatitis. Typically, when the posterior pancreatic duct is disrupted or a pancreatic pseudocyst extends into

the pleural cavity, there is an aberrant communication to the pleural space [Table/Fig-32-34] [20].



[Table/Fig-32,33,34]: A case of acute on Chronic Pancreatitis (CP) with complications. [Table/Fig-32,33]: Contrast-enhanced axial CT images shows atrophic body and tail of pancreas with heterogeneous contrast enhancement with few non enhancing areas (>30%) (white arrow heads) and significant peripancreatic fat stranding (short white arrow) noted. [Table/Fig-33] shows A well-defined hypodense non enhancing cystic lesion in head of pancreas (white star)-likely WON. [Table/Fig-34]: Contrast-enhanced sagittal image shows an ill-defined peripherally enhancing heterogeneous hypodense collection with pockets of air (measuring ~100 cc) (acute necrotic collection) noted in tail region of pancreas and splenic hilum. The collection is noted communicating with the left pleural cavity through a defect (long white arrow) in posteromedial aspect of left hemidiaphragm- s/o pancreatic pleural fistula. (Courtesy Krishna Rajendra Hospital -MMCRI). (Images from left to right)

CONCLUSION(S)

One of the most frequent pancreatic pathologies and medical/surgical emergencies seen in daily clinical practice is pancreatitis. The most challenging aspect of managing pancreatitis is identifying and managing its (systemic or local) consequences, rather than the pancreatitis itself. Local problems can be identified using imaging tests, and CT has long been considered one of the most effective imaging methods for this.

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