INTRODUCTION

Despite the continuum of acute and chronic pancreatitis (AP and CP), we will discuss both entities separately. AP is nearly three times as common as pancreatic cancer, and is triggered by alcohol abuse, particularly binge-drinking, or obstructing gallstones in 80% of cases. Moreover, its incidence has more than doubled in the past 10 years. Whether this is due to the increasing prevalence of obesity or simply higher detection rates is unclear. AP occurred in 1.04% patients following bariatric surgery, e.g. a rate significantly higher than in the general population.

Although pancreatic and periglandular inflammation, as well as systemic inflammatory response (SIR) are the predominant sequela of AP, the underlying etiology of inflammation includes mechanical obstacles, such as biliary or pancreatic duct (PD) stones, strictures, or tumour masses, such as cystadenomas or early pancreatic adenocarcinoma (PCa).}

Intermittent mechanical obstruction, such as Sphincter of Oddi dysfunction, where the duodenal sphincter fails to relax normally, along with anatomic variations in the union of the PD and common bile duct (CBD), are rare causes of recurrent AP. Up to 10% of AP is due to infection, e.g. mumps or salmonella. AP may follow surgery or endoscopic retrograde cholangiopancreatography (ERCP). Trauma-induced AP, after penetrating or blunt injury, is far more frequent than recognised. According to the International Consensus Guidelines on Early Chronic Pancreatitis, pancreatic inflammation must last at least 6 months before it can be labelled CP. The distinction is important because, unlike AP, CP can destroy endocrine and exocrine pancreatic function, emphasising the importance of early diagnosis. As typical AP can be diagnosed by clinical symptoms plus laboratory tests, imaging is usually reserved for those with recurrent, complicated or CP. Imaging typically starts with ultrasound and more frequently with contrast-enhanced computed tomography (CECT). MRI and/or MR cholangiopancreatography can be used as a problem-solving tool to confirm indirect signs of pancreatic mass, differentiate between solid and cystic lesions, and to exclude pancreatic duct anomalies, as may occur with recurrent AP, or to visualise early signs of CP. MR cholangiopancreatography has replaced diagnostic endoscopic retrograde cholangiopancreatography (ERCP). However, ERCP, and/or endoscopic ultrasound (EUS) remain necessary for transpapillary biliary or pancreatic duct stenting and transgastric cystic fluid drainage or pancreatic tissue sampling, respectively. Finally, positron emission tomography-MRI or positron emission tomography-CT are usually reserved for complicated cases and/or to search for extra pancreatic systemic manifestations. In this article, we discuss a broad spectrum of inflammatory pancreatic disorders and the utility of various modalities in diagnosing acute and chronic pancreatitis.
Nonetheless, nowadays, pancreatitis is considered a spectrum of disease spanning from acute to recurrent to chronic pancreatitis,14 (Table 1).

In Western societies, chronic alcoholism accounts for 80% of CP in adults.15 Nicotine consumption also increases the risk of CP and malignancy, mainly PCa.16 Otherwise, patient demographics, family history, and geography can help in determining the aetiology of CP, e.g., hereditary CP or cystic fibrosis. In young to middle-aged adults, autoimmune pancreatitis (AIP), pancreas divisum, and hyperlipidaemia should be leading considerations. Obstructive pancreatitis, due to pancreatic or duodenal neoplasms, or groove pancreatitis should be considered in middle-aged to elderly patients, especially if there is a history of weight loss.17 CP, like recurrent AP, may also occur as a sequelae of AP.18

Diagnosis of acute pancreatitis

The 2012 Revised Atlanta Criteria provide a standardised framework for classifying AP. Clinically, these criteria divide AP into mild, moderate, and severe forms, depending on the absence or presence of additional organ involvement. Morphologically, AP is classified as either interstitial oedematous (IEP) or necrotising acute pancreatitis (NAP). Because the pancreas synthesises digestive enzymes, in AP, pathophysiologically, the parenchyma “self-digests” due to the release of prematurely-activated pancreatic enzymes.11

Clinically, early-phase AP, usually lasting 1 week, is accompanied by a systemic inflammatory response (SIR) indicated by elevated C-reactive protein (CRP), leucocyte count,19 and fibrinogen.19 The late phase begins in the second week and can last months, in moderate or severe pancreatitis, as local complications arise or systemic inflammation persists.20 Periumbilical or flank ecchymoses, referred to as the Cullen and Grey Turner signs, respectively, suggest NAP,21 which, on cross-sectional imaging, appear as inflammation along the gastrohepatic and/or falciform ligaments and other peritoneal reflections, respectively.22,23 These signs are neither sensitive nor specific for necrotising pancreatitis. However, they indicate life-threatening disease, the extent of which can be seen on cross-sectional imaging, with mortality approaching 40%.24

The 2012 Revised Atlanta Criteria require two of three criteria to establish the diagnosis of AP: (1) characteristic epigastric pain; (2) ≥ threefold increase in lipase and/or amylase concentration in the blood serum; and/or (3) pathognomonic CT or MRI features of AP.20 Although imaging is not a prerequisite for diagnosis, portal venous phase imaging on contrast-enhanced CT (CECT) especially, may diagnose when labs lag or identify complications precluding clinical improvement after 48–72 h of therapy, e.g., 20,25 splenic and/or portal venous thrombosis or pseudoaneurysm of the splenic or less commonly gastroduodenal artery.26

Because AP is of biliary origin in approximately 40% of cases,27 ultrasound is an ideal radiation-free starting point to search for stones within the gallbladder, cystic duct or PD. However, ultrasound is unsatisfactory for assessing the pancreas due to anatomic and echotexture variations in the healthy pancreas. Furthermore, because the pancreas lacks a capsule, extrapancreatic structures, such as bowel, lymph nodes or vessels, may be mistaken for pancreatic lesions.28

Should there be a suspicion of glandular and/or peripancreatic fluid collections, cross-sectional imaging is indicated to determine management.25 CT is usually the modality of choice in AP, principally because it enables a rapid examination of the entire abdomen/pelvis, exclusion of symptoms-mimickers of AP, such as aortic dissection, shedding light on other causes of an acute abdomen or the aetiology and/or extent of pancreatic

---

Table 1. Overview of known risk factors of acute and chronic pancreatitis, age-dependent and age-independent

| Age of onset       | Acute pancreatitis                                                                 | Chronic pancreatitis                  |
|--------------------|------------------------------------------------------------------------------------|---------------------------------------|
| Youth              | Pancreas divisum, Mumps, Cystic fibrosis                                           | Hereditary pancreatitis, Tropical CP  |
| Middle-Aged        | Stones* (GB, PD, CBD, Cystic duct), Obesity, S/P bariatric surgery, Ectopic tissue, Sphincter dysfunction | Chronic alcoholism, Hyperlipidaemia, Groove pancreatitis, Autoimmune pancreatitis, Pancreatic or duodenal neoplasm |
| Older              | Binge-drinking, Polytrauma involving pancreas, Salmonella, Drug-induced, Iatrogenic, e.g. ERCP | Pancreatic or duodenal neoplasm       |

---
Balthazar CT Severity Index (CTSI) correlates most closely with pancreatic inflammation, high signal intensity. Furthermore, (DWI) sequences may show subtle pancreatic and/or peripancreatic stranding (arrow) is present. On the HASTE image, the pancreatic duct is minimally dilated. Increased signal (arrowheads) is seen in the pancreatic tail and the affected part of the body on T2 and DWI. Low signal intensity on T1 and inhomogeneous but preserved enhancement in the pancreatic tail and body represent oedema and no necrosis (arrowheads). DWI, diffusion-weighted image.

In the trauma setting, even with state-of-the-art CT, 20–40% of CTs done within 12 h of presentation will appear normal. Follow-up CT should be done at 12 to 24 h. If “hard” signs of pancreatic trauma, i.e. laceration, contusion or hematoma are found, therapy can begin. But if fluid is present between the pancreas and splenic vein or there is an abrupt cut-off of the superior mesenteric vein, i.e. “soft” signs, further CT or MRI is warranted, especially if serial amylase/lipase levels, which are not specific for pancreatitis, are trending higher. A high index of suspicion is necessary to prevent the high morbidity and mortality associated with delayed or missed diagnosis of main pancreatic duct (MPD) injury especially, most common at the body–tail junction. MRI, and if necessary and available, secretin-enhanced MR cholangiopancreatography (MRCP), can reliably exclude MPD rupture.

Clinical categorisation using the Revised Atlanta Criteria

These criteria define three stages: (a) mild AP, most common, where involvement is limited to the pancreas. There are no complications. Most patients are discharged after 1 week; (b) moderate pancreatitis applies to those who experience transient organ involvement ≤ 48 h after onset of AP. Local or systemic complications may also occur; and (c) severe AP where organ involvement occurs > 48 h after the onset. Usually the cardiovascular, respiratory, and renal systems are involved.

Morphological categorisation using imaging findings

The Revised Atlanta Criteria separate AP, histologically, into IEP and NAP, which correspond to the mild and severe clinical forms defined above, respectively. On cross-sectional imaging, IEP, accounting for 85% of all hospitalised AP cases, appears as diffuse or focal parenchymal swelling, with fluid-like density/ signal intensity on CT/MRI, respectively, within the pancreas (Figure 1). On CE-CT, pancreatic lobules may be less distinct and contrast uptake is heterogeneous. If contrast uptake in the pancreas is preserved, by definition, there is no necrosis. The absence of contrast uptake raises the possibility of NAP. IEP usually subsides within a week, and its mortality is only 3–4%). IEP rather than the four- and more than 10-fold greater mortality as with NAP and NAP complicated by infection, respectively.

NAP can affect parenchymal and/or peripancreatic tissue and accounts for 5–10% of all AP cases. On CT, three distinct patterns have been identified: (a) necrosis of pancreas and peri-pancreatic tissues (75%); (b) necrosis limited to the peri-pancreatic tissues (20%); and (c) necrosis involving only the pancreas (5%) (Figure 2). Since necrosis requires several days to occur, CECT done during the first week may be inconclusive. Follow-up CECT is recommended, if suspected.

Pancreatic and peripancreatic fluid collections

The Revised Atlanta Criteria further subdivides IEP and NAP fluid collections according to their age, and content, i.e. purely liquid or partly solid/necrotising into four types (Table 2).

(1) Acute peripancreatic fluid collection (APFC) occurs before 4 weeks of IEP, appearing as a homogeneous, extra pancreatic fluid accumulation without a wall (Figure 3). Over half of APFC regress spontaneously. Rarely, APFC evolves into a pseudocyst.
(2) Pancreatic pseudocyst, a late complication of IEP, is an encapsulated, non-enhancing, purely liquid collection (Figure 4). It appears homogeneously hypodense on CE-CT and hyperintense on $T_2$ weighted MRI. Occasionally, these cysts may be connected to the PD and are easily identified on MRCP.\(^{4,20}\)

(3) Acute necrotising collection (ANC), like APFC, appears before 4 weeks of NAP. ANCs contain solid or fatty tissue surrounded by fluid, pancreatic and/or peripancreatic. Often, ANC spreads to the omental bursa and perirenal space (Figure 5). Cross-sectional imaging after the first week distinguishes ANC from APFC.\(^{4,20}\)

(4) Walled-off necrosis (WON) is a late consequence of NAP, since the thick enhancing rim takes time to organise. But, unlike the pseudocyst, it contains necrotising/solid tissue (Figure 6). WON is frequently localised peripancreatically rather than in the organ.\(^{4}\) MRI is superior to CT in that it characterises lesions as solid, semi-solid or liquid, identifying targets for drainage. PD interruption, which may cause parenchymal necrosis, is usually better discerned by MRI,\(^{44}\) especially secretin-enhanced MRCP (S-MRCP).\(^{45}\)

Local and systemic complications
These include retroperitoneal bleeding, pseudoaneurysm, pancreatic fistula formation, extrahepatic portal hypertension, gastric/bowel perforation, renal obstruction, and “gastric-outlet syndrome” due to extrinsic luminal compression. Additional CT (or MRI) findings predictive of multiorgan failure, including pleural effusions, ascites, pulmonary oedema, renal, and perinephric oedema, were integrated into Balthazar’s modified CTSI.
which is occasionally used by radiologists.\textsuperscript{21,46} When gas is seen within the pancreatic or peripancreatic tissues, a fistula with bowel should be ruled out. Rarely, gas is seen within a walled-off retroperitoneal collection implying super-infection with a gas-forming organism. This complication of NAP is associated with very high mortality, especially in the setting of extra pancreatic...

Figure 7. A 63-year-old male who developed infected necrosis following acute pancreatitis a, Axial CECT, arterial phase images; and b, coronal CE-CT, portal venous phase images, show a large, thick-walled, rim-enhancing collection in the pancreatic bed (arrows). Several air bubbles within the fluid collection and a large air-fluid level (arrowhead) are suspicious for an infected necrosis replacing the entire pancreatic gland. Fine-needle aspiration confirmed the diagnosis. Minimal peri-pancreatic stranding is present. Note the stomach ventral to the infected necrosis (asterisk).
Figure 8. Signs of advanced chronic pancreatitis in two different patients. a, Axial; and b, coronal non-enhanced CT in a 17-year-old male. Diffuse parenchymal atrophy and calcification consistent with severe chronic pancreatitis. MRI of a 35-year-old male patient with advanced chronic pancreatitis. c, Axial turbo spin-echo T2 weighted (HASTE) image with fat-suppression shows moderate dilatation and diffuse irregularity of the MPD with a few visible side-ducts (arrows) and atrophy of the pancreatic gland. d, Coronal oblique maximal intensity projection image of a 3D MR cholangiopancreatogram shows generalised irregularity and marked dilatation of the MPD with multiple massively dilated side-branches (arrows), classified as Cambridge 4. e, Pre-contrast axial 3D- GRE T1-weighted image with fat-suppression shows markedly decreased signal intensity of the pancreatic body compared to that of the tail (asterisk). f, Contrast-enhanced, portal venous phase, axial, T1-weighted images show the atrophied pancreas with lobular disappearance and mild MPD dilatation, as well as diminished contrast enhancement of the body more than the tail.

organ failure47 (Figure 7). A positive bacterial culture on fine needle aspiration confirms the diagnosis prior to antibiotics.20 Furthermore, sterile NAP must be distinguished from superinfected NAP since the latter often requires drainage, while necrosis can be managed expectantly and rarely requires endoscopic or surgical necrosectomy.36,48

Diagnosis of chronic pancreatitis
CP will develop in 4–24% of patients with recurrent AP. CP refers to episodic flareups of acute inflammation that result in irreversible fibrosis of the pancreatic gland, which causes first exocrine, and, ultimately, endocrine insufficiency.55 End-stage CP is a straightforward imaging diagnosis, typically characterised by atrophy,54 calcifications (parenchymal much more frequent than intraductal), calibre, and/or contour alterations of the main and side-branch PD (Figure 8).2 Because the degree of fibrosis seen on conventional imaging does not directly correlate with the severity of glandular dysfunction, imaging does not help determine CP severity. It only excludes end-stage disease.52

The diagnosis of early CP is very challenging because the symptoms are nonspecific. Ninety percent reduction of pancreatic lipase, the first enzyme impaired in pancreatic insufficiency, must occur before malabsorption occurs. S-MRCP is by far the best test to estimate exocrine function, with 75% sensitivity for early- and up to 97% sensitivity for late-stage CP.53 However, Secretin (Secrelux®) is no longer commercially available in the EU. Although it may be purchased in the US under the generic name human secretin (trade name ChiRhoStim®), it is rather expensive.

Alternatively, indirect tests, such as fecal chymotrypsin since the fecal concentration of elastase is directly proportional to that excreted by the pancreas. Furthermore, if the patient is on exogenous enzyme, the fecal elastase test can be done without stopping the oral preparation. However, it has very low sensitivity in mild CP and circa 75% in moderate and severe CP.54 Therefore, MRCP has become the diagnostic exam for CP in many radiology centres with a sensitivity of 75% for advanced disease and 25% for small-duct, i.e., early CP.53

Because debilitating pain, malabsorption and malnutrition impair quality of life in end-stage CP and predispose to PCa,17 the goal of imaging is to identify CP as early as possible. This usually means assessing for anatomical variations, early changes along the main and side-branch PDs that might progress to strictures/stenosis, and morphologic pancreatic changes. With early diagnosis, using S-MRCP55 oral pancreas lipase (Creon) can be started, and if necessary insulin, to replace exocrine and endocrine pancreatic enzymes, respectively. Additionally, chronic epigastric pain can be managed by alcohol cessation, analgesics or narcotic drugs, endoscopic drainage, stenting or stone removal from the PD, extracorporeal shock-wave lithotripsy for PD calculi, surgical PD drainage or resection of pancreatic tissue and/ neuroablation.55 For early detection of PCa, the Cancer of the Pancreas Screening consortium recommends periodic EUS or MRI in these high-risk individuals.56

Although CP is more likely to present as atrophy, due to fibrosis in advanced cases, the gland may also be enlarged early on and/or exhibit focal inflammation.51 This mass-forming appearance of AP or CP can be very difficult to distinguish from PCa on cross-sectional imaging. Both are hypo- or isodense on CE-CT, have a predilection for the pancreatic head, and can dilate the PD. Biopsy can be equivocal and may not help as atrophy, fibrosis, and leukocytic infiltration may occur in both entities.56 S-MRCP can demonstrate the duct penetrating sign which can be helpful in such cases (Figure 9).35

Classification of chronic pancreatitis
In 1988, the Marseilles-Rome Classification57 was redefined, based upon aetiology of CP, and pancreatic morphology and function.58 With ERCP, the 1984 Cambridge Classification79
was devised to clinically quantify CP according to the extent and severity of main and side branch PD involvement.60

PD pathologies include strictures, dilatations, and cysts. Side-branch pathologies additionally include a reduction in the number or length of side branches. However, with the advent of non-invasive S-MRCP, ERCP has largely been relegated to therapeutic interventions, thus reducing the risk for ERCP-related AP.61 The American Pancreatic Association recommends adapting the Cambridge Classification to coronal-oblique MRCP images. Since only PD morphology is quantified, the pancreatic exocrine functional reserve cannot be estimated by this method.45

In contrast, S-MRCP can detect an obstruction and estimate pancreatic exocrine function, respectively, in response to secretin provocation. The healthy MPD distends about 66% after secretin, returning to its original calibre within 10 min.45 Signs of PD pathology include loss of MPD tapering within the pancreatic tail, rigid MPD post secretin (Figure 10); visualization of ≥3 side-branch ducts, strictures, and/or sacculations of the MPD, and reduced and/or delayed duodenal filling (Table 3). Pancreatic exocrine function, is considered impaired if <Grade 345 according to the Matos classification (Table 3). But, it is limited because it cannot distinguish between patients with early versus more advanced CP.45

Complications of CP
The foremost complication of CP is PCa, which is 15 to 25 times more likely to occur in this group than in the general population. Two types of CP deserve special mention in this regard: tropical pancreatitis, where PCa favours the body or tail rather than the pancreatic head; and hereditary pancreatitis in which not only...
Figure 11. Biopsy-proven groove pancreatitis in a 44-year-old female a, Axial non-contrast; and b, axial contrast-enhanced arterial-phase; and c, portal venous-phase 3D GRE T1 weighted images with fat-suppression; and d, axial DWI, b-value = 50; and E, axial turbo spin-echo T2 weighted (HASTE) image with fat-suppression. There is mild duodenal thickening and oedema associated with a sheet-like mass in the pancreaticoduodenal groove that extends to the pancreatic head (arrowheads), plus multiple tiny bright cystic lesions in the pancreatic-duodenal groove (arrows) (a). Decreased pancreatic parenchymal T1 signal and diminished enhancement on post-contrast images (d). Coronal, oblique, thick-slab MR cholangiopancreatogram image after administration of secretin (S-MRCP) shows normal calibre of the entire MPD (thick arrow) as it goes through the mass (duct penetrating sign), excluding malignancy. MRCP, MRcholangiopancreatography.

is the risk of PCa still greater (50- to 70-fold above the general population), but it can occur within 7 years from diagnosis of CP. 62

Other complications include those seen with AP, namely pseudocysts, bile duct compression due to pseudocysts, pseudoneurysms, and splenic vein thrombosis with variceal collaterals. Gastrointestinal complications, such as intestinal ischaemia or gastric outlet stenosis due to pancreatic head enlargement may also occur. 63

Specific cross-sectional imaging patterns of chronic pancreatitis

Typical CP
This is most frequently assessed with CT rather than MRI. Transabdominal ultrasound is not recommended. 61 Although calcifications and atrophy, i.e., end-stage features, are more easily detected on CT (Figure 8), MRI is superior for detecting the subtle findings of early CP, including subtle duct irregularities, signal intensity alterations, and loss of lobulations (Figure 10). 61

Furthermore, both T1 weighted sequences and T1-mapping techniques have shown promise. Tirkes et al 64,65 found a positive correlation between T1 relaxometry and exocrine dysfunction, even in persons without ductal changes. Also, MR elastography (MRE) has been reported to be diagnostically helpful. 66 Therefore, MRI and, in particular, S-MRCP, is particularly very helpful in diagnosing early CP (Figure 10). 65 However, CT remains the first-line exam because of its wide field of view that can help exclude other entities that mimic CP, as well as detect the above-mentioned complications of end-stage disease easily. 61

Groove pancreatitis
Also known as paraduodenal pancreatitis or cystic duodenal dystrophy, groove pancreatitis is a focal form of CP, between the duodenum, pancreatic head, and CBD, mainly found in alcoholics. The CBD may also become fibrosed. 67 If fibrosis spreads to the pancreatic head, it can mimic carcinoma. 68 Anatomic variants, such as pancreas divisum or stenosis of the papilla duodeni minor, are believed to cause the inflammatory changes. 67 Excess alcohol or nicotine use, by changing pancreatic secretion viscosity, causes PD calcification, enzyme flow impairment, and Brunner gland hyperplasia and duodenal wall cysts, which can be seen at histology. 66

Small duodenal cysts seen on endoscopic ultrasound (EUS) suggest the diagnosis. Low attenuation in the pancreatic head on late-phase CE-MR images, and/or CBD or gastroduodenal artery displacement without invasion or encasement, as can be seen with groove pancreatitis, may be helpful to avoid the misdiagnosis of PCa.

Unlike the moderately hyperintense, non-pathologic pancreas seen on fat-saturated T1 weighted MRI, paraduodenal pancreatitis, like any pancreatitis, appears iso- to hypointense. On T2 weighted images, it is iso- to hypointense. The “double duct sign,” thought to be pathognomonic for rigid malignancy, e.g. PCa, compressing the ampullary PD and CBD, and causing upstream dilatation of the CBD and PD, can be seen with duodenal wall fibrosis. 68 S-MRCP can also display the “duct-penetrating sign,” indicating that the main PD can pass unhindered through non-carcinomatous tissue (Figure 11). 45

Autoimmune pancreatitis
AIP, accounting for 2–10% of all CP, 70 frequently affects males. 71 Although incompletely understood, immunological and genetic factors are suspected as its cause. 72 Unlike alcohol-induced or biliary-associated pancreatitis, AIP is characterised by the expression of various autoantibodies, as with autoimmune hepatitis (AIH) or primary biliary cholangitis (PBC). 73 Although AIP has two distinct subgroups, expressing different pathological and clinical features, 74 a common denominator is that many lack increased inflammatory parameters 75 or fever 71 and all respond well to corticosteroids, which can be used both therapeutically and diagnostically. 76 On CT and MR imaging, a capsule-like rim, which is thought to correspond to an inflammatory process...
involving peripancreatic tissues, appears to be a characteristic finding of AIP.77

The Type 1 AIP IgG4-positive patients have systemic manifestations of autoimmune diseases, which can pre-or post-date AIP.77 In some cases, multiple organ involvement occurs, sparing the pancreas.78 Histology shows lymphoplasmocytic infiltration and fibrosis. In contrast, Type 2 AIP has neutrophil infiltrates and epithelioid cell granulomas on histology and no increase in serum IgG4.79 Unlike Type 1, which predominates in Asian countries, Type 2 AIP occurs mainly in Europe and America.80

On cross-sectional imaging, any of the three patterns can be seen: diffuse “sausage-like” (Figure 12); focal swelling (30–40%) usually limited to the pancreatic head; and multifocal pancreatic involvement. PD dilatation, as well as pancreatic atrophy, are absent.72,81,82 In challenging cases, DWI may help, showing significantly lower apparent diffusion coefficient (ADC) values in a pseudotumour than PCa.83 F-18 fluordeoxyglucose (FDG) positron emission tomography (PET) may help diagnose AIP, showing uptake beyond the pancreas.71 Conversely, FDG-PET/CT may exclude a pseudotumour when the standard uptake value (SUV) is raised.84

It is estimated that 3–9% of all patients with AIP undergo unnecessary pancreatic resection for suspected carcinoma,71 particularly if repeated biopsies remain equivocal. Serology, i.e. IgG4, and CA 19–9 levels, as well as a trial of steroids, should be recommended under such circumstances. Because the CA 19–9 tumour marker is not specific to pancreatic cancer and can even be found with some benign entities, performing all three of these recommendations yields higher certainty than any single one.82,85 Imaging findings that support PCa over focal AIP include persistent hypodensity/hypointensity of a cancer on the late phase of CECT and CE-MR relative to a non-pathologic pancreas.76 The presence of intraluminal enhancement of the MPD wall (“enhanced duct sign”) on CECT indicates pancreatitis.86

And, finally, the degree of MPD dilatation suggests the aetiology. While in focal AIP the MPD is only slightly dilated (≤4 mm), more pronounced PD dilatation indicates malignant obstruction. In addition, as with colon cancer versus diverticulitis,87 long-segment PD narrowing argues for AIP rather than the abrupt calibre changes in cancer. Likewise, multiple, segmental stenosis along the MPD favours AIP.82 The key is that confirmed AIP does not exclude synchronous PCa, which was found at histology in nearly 5% of Type 1 AIP patients.88

In summary, the spectrum of pancreatitis includes acute, recurrent and chronic pancreatitis. The aetiologies are multifactorial. However, two of the leading causes remain alcohol abuse and gallstones. Clinical and lab findings are often adequate for diagnosing AP. Cross-sectional imaging is warranted when the clinical course is severe or atypical. Generally, CE-CT is preferable to MRI because it allows coverage of an extended area. But, MRCP is the preferred technique for the diagnosis of early CP, and the estimation of exocrine pancreatic function. In the near future, DWI, T1 mapping, and MRE may become routine imaging adjuncts.

REFERENCES

1. Weiss FU, Laemmerhirt F, Lerch MM. Etiology and risk factors of acute and chronic pancreatitis. Visc Med 2019; 35: 73–81. doi: https://doi.org/10.1159/000499138
2. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer.
Gastroenterology 2013; 144: 1252–61. doi: https://doi.org/10.1053/j.gastro.2013.01.068
3. Schima W, Ba-Salalah A, Plank C, Kulina-CoSentini C, Püspök A, Pankreas — Teil I. Radiologe 2006; 46: 321–38. doi: https://doi.org/10.1007/s00117-006-1340-4
4. Foster BR, Jensen KK, Bakis G, Shaaban AM, Cokley FV. Revised Atlanta classification for acute pancreatitis: a pictorial essay. RadiologyGraphics 2016; 36: 675–87. doi: https://doi.org/10.1148/rg.2016150097
5. Kumaravel A, Zelisko A, Schauer P, Lopez R, Kroh M, Stevens T. Acute pancreatitis in patients after bariatric surgery: incidence, outcomes, and risk factors. Obesity Surgery 2014; 24: 2025–30. doi: https://doi.org/10. 1007/s11695-014-1337-4
6. Li S, Tian B. Acute pancreatitis in patients with pancreatic cancer. Medicine 2017; 96: e5908. doi: https://doi.org/10.1097/MD. 0000000000055908
7. Sherifi F, Bexheti S, Gashi Z, Bajraktari I, Shatri J, Lahu A. Anatomic variations of pancreaticobiliary Union. Open Access Maced J Med Sci 2018; 6: 988–91. doi: https://doi. org/10.3889/ojams.2018.196
8. Tagajdiz MR. Acute pancreatitis caused by mumps infection in an adult. Infectious Diseases 2018; 2: 3.
9. Hamaguchi H, Okabayashi Y, Yoneda R, Ueno H, Yoon S, Sakaue M, et al. A case of acute pancreatitis complicating Salmonella enteritis. Int J Gastrinol Cancer 1999; 26: 189–92. doi: https://doi.org/10.1385/IJGC:26:3:189
10. Schreyer AG, Grenacher L, Iuchems M. Pankreatitis. Radiologie 2016; 56: 355–62. doi: https://doi.org/10.1007/s00117-016-0088-8
11. Xiao B, Xu H-B, Jiang Z-Q, Zhang J, Zhang X-M. Current concepts for the diagnosis of acute pancreatitis by multiparametric magnetic resonance imaging. Quantitative Imaging in Medicine and Surgery 2019; 9: 1973–85. doi: https://doi.org/10.21037/qims.2019.11.10
12. Kumar A, Panda A, Gamanagatti S. Blunt pancreatic trauma: a persistent diagnostic conundrum? World J Radiol 2016; 8: 159–73. doi: https://doi.org/10.4329/wjr.v8.i2.159
13. Whitcomb DC, Shimosegawa T, Charli ST, Forsmark CE, Fruoloni L, Garg P, et al. International consensus statements on early chronic pancreatitis. recommendations from the Working group for the International consensus guidelines for chronic pancreatitis in collaboration with the International association of Pancreatology, American pancreatic association, Japan pancreas Society, PancreasFest Working group and European pancreatic Club. Pancreatology 2018; 18: 516–27. doi: https://doi.org/10.1016/j.pan.2018.05.008
14. Kamisawa T, Shimosegawa T. Definition and classification of chronic pancreatitis. Pancreas 2010; 39: 701. doi: https://doi.org/10.1097/MPA.0b013e32833f5807
15. Elshirly SB, Virarakar M, Javadi S, Ibarra-Rovira JJ, Tamp EP, Bhosal PR. Pancreatitis and PDAC: association and differentiation. Abdominal Radiology 2019.
16. Talamin G, Bassi C, Falconi M, Sartori N, Salvia R, Rigo L. Alcohol and smoking as risk factors in chronic pancreatitis and pancreatic cancer. Digestive Diseases and Sciences 1999; 44: 1303–11. doi: https://doi.org/10.1023/A:1026670911955
17. Pham A, Forsmark C. Chronic pancreatitis: review and update of etiology, risk factors, and management. F1000Research 2018; 7: 607. doi: https://doi.org/10.12688/ f1000research.12852.1
18. Ahmed Ali U, Issa Y, Hagenarsa JC, Bakker OJ, van Goor H, Nieuwenhuijs VB, et al. Risk of recurrent pancreatitis and progression to chronic pancreatitis after a first episode of acute pancreatitis. Clinical Gastroenterology and Hepatology 2016; 14: 738–46. doi: https://doi.org/10.1016/j.cgh.2015.04.040
19. Ransons JHC, Lackner H, Bermann IR, Schinella R. The relationship of coagulation factors to clinical complications of acute pancreatitis. Surgery 1977; 81: 502–11.
20. Banks PA, Bolten TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. Gut 2013; 62: 102–11. doi: https://doi.org/10.1136/gutjnl-2012-302779
21. Morgan DE. Imaging of acute pancreatitis and its complications. Clin Gastroenterol Hepatol 2008; 6: 1077–85. doi: https://doi. org/10.1016/j.cgh.2008.07.012
22. Ba-Salalah A, Bastati N, Uffmann M, Pretterklieber M, Schima W. Peritoneum and Mesentery. Radiology 2009; 49: 543–56. doi: https://doi.org/10.1007/s00117-008-1769-8
23. Wright WF. Cullen sign and grey Turner sign revisited. J Am Osteopath Assoc 2010; 110: 583–9. doi: https://doi.org/10.14210/jaoa.2010.0018
24. Guldmann G, Magee EM. Grey Turner sign. Treasure Island (FL: StatPearls; 2020.
25. Reynolds PT, Brady EK, Chawla S. The utility of early cross-sectional imaging to evaluate suspected acute mild pancreatitis. Annals of Gastroenterology 2018; 31: 628–32. doi: https://doi.org/10.20524/aog.2018.0291
26. O’Connor OJ, Buckley JM, Maher MM. Imaging of the complications of acute pancreatitis. AJR Am of Roentgenol 2011; 197: W357–81. doi: https://doi.org/10.2214/AJR.10.4339
27. Gullo L, Migliori M, Oláh A, Farkas G, Levy P, Arvanitakis C, et al. Acute pancreatitis in five European countries: etiology and mortality. Pancreas 2002; 24: 223–7. doi: https://doi.org/10.1097/00006676-200204000-00003
28. Čwik G, Gierblitski IW. Errors and mistakes in the ultrasound diagnosis of the pancreas. J Ultrasound 2013; 13: 178–91. doi: https://doi.org/10. 15557/JoU.2013.0018
29. Heyn C, Sue-Chue-Lam D, Jhaveri K, Hader MA. MRI of the pancreas: problem solving tool. J Magn Reson Imaging 2012; 36: 1037–51. doi: https://doi.org/10.1002/jmri.23708
30. Hao L, Liu L, Meng X, Yu G, Li E, Gu H. Evaluation of different b-values in DWI and 1H MRS for pancreatic cancer and pancreatitis: a rabbit model. Bioscience Reports 2019; 39: doi: https://doi.org/10. 1042/BSR20181933
31. Kim YK, Ko SW, Kim CS, Hwang SB. Effectiveness of Mr imaging for diagnosing the mild forms of acute pancreatitis: comparison with MDCT. J Magn Reson Imaging 2006; 24: 1342–9. doi: https://doi.org/10.1002/jmri.20801
32. Shinya S, Sasaki T, Nakagawa Y, Gaiquing Z, Yamamoto F, Yamashita Y. The efficacy of diffusion-weighted imaging for the detection and evaluation of acute pancreatitis. Hepato-Gastroenterology 2009; 56(94–95): 1407–10.
33. Tokunaga A, Arizono S, Shimizu H, Fujimoto K, Kurata M, Minamiguchi S, et al. Optimizing b-values for accurate depiction of pancreatic cancer with tumor-associated pancreatitis on computed diffusion-weighted imaging. Clinical Imaging 2020; 61: 20–6. doi: https://doi.org/10.1016/j.clinimag.2020. 01.007
34. Martin DR, Karabulut N, Yang M, McFadden DW. High signal peripancreatic fat on fat-suppressed spoiled gradient echo imaging in acute pancreatitis: preliminary evaluation of the prognostic significance. J Magn Reson Imaging 2003; 18: 49–58. doi: https://doi.org/10.1002/jmri.10333
35. Gallix BP, Bret PM, Atri M, Lecesne R, Reinhold C. Comparison of qualitative and quantitative measurements on unenhanced T1-weighted fat saturation MR images in predicting pancreatic pathology. J Magn Reson Imaging 2005; 21: 583–9. doi: https://doi.org/10.1002/jmri.20310
36. Schwarze V, Marschner C, Schulz C, Streitparth F. Akutes abdomen – gastrointestinale Ursachen. Der Radiologie
Prophylactic infected necrosis: morbidity and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. Gastroenterology 2010; 139: 813–20. doi: https://doi.org/10.1053/j.gastro.2010.06.010

Radulova-Mauersberger O, Belyaev O, Birgin E, Bosch F, Brunner M, Müller-Debus CE, et al. Indications for surgical and interventional therapy of acute pancreatitis. Zentralbl Chir 2020; 145: 374–82. doi: https://doi.org/10.5555/z.1164-7099

Ke L, Li J, Hu P, Wang L, Chen H, Zhu Y. Percutaneous catheter drainage in infected pancreatic necrosis: a systematic review. Indian J Surg 2016; 78: 221–8. doi: https://doi.org/10.1007/s12262-016-1495-9

Shi J, Xue J. Inflammation and development of pancreatic ductal adenocarcinoma. Chin Clin Oncol 2019; 8: 19. doi: https://doi.org/10.21037/ccov.2019.04.02

Kim T, Murakami T, Takamura M, Hori M, Takahashi S, Nakamori S, et al. Pancreatic mass due to chronic pancreatitis: correlation of CT and MR imaging features with pathologic findings. AJR Am J Roentgenol 2001; 177: 367–71. doi: https://doi.org/10.2214/ajr.177.2.1770367

An azi A, Hart PA, Conwell DL. Diagnosing chronic pancreatitis. Dig Dis Sci 2017; 62: 1713–20. doi: https://doi.org/10.1007/s10620-017-4493-2

Lieb JG, Dragano N, Vlckova A, Boscovich D, Marko T, et al. Prognostic factors for the severity of interstitial pancreatitis. Marseilles-Rome 1988. HEPATOGASTROENTEROL 1989; 13: 857–9.

Beyer G, Mahajan UM, Budde C, Bulla TJ, Kohlmann T, Kuhlmann L, et al. Development and validation of a Chronic Pancreatitis Prognosis Score in 2 Independent Cohorts. Gastroenterology 2017; 153: 1544–54. doi: https://doi.org/10.1053/j.gastro.2017.08.073

Sarner M, Cotton PB. Classification of pancreatitis. Gut 1984; 25: 756–9. doi: https://doi.org/10.1136/gut.25.7.756

Tirkes T, Shah ZK, Takahashi N, Grajo JR, Chang ST, Venkatesh SK, et al. Reporting standards for chronic pancreatitis by using CT, MRI, and MR cholangiopancreatography: the Consortium for the study of chronic pancreatitis, diabetes, and pancreatic cancer. Radiology 2019; 290: 207–15. doi: https://doi.org/10.1148/radiol.2018181353

Conwell DL, Lee LS, Yadav D, Longnecker DS, Müller FH, Mortejl KJ, et al. American pancreatic association practice guidelines in chronic pancreatitis: evidence-based report on diagnostic guidelines. Pancreas 2014; 43: 1143–62. doi: https://doi.org/10.1097/MPA.0000000000000237

Shanbhogue AKP, Fasih N, Surabhi VR, Doherty GP, Shanbhogue DKP, Sethi SK. A clinical and radiologic review of uncommon types and causes of pancreatitis. Radiographics 2009; 29: 1003–26. doi: https://doi.org/10.1148/rg.290487548

Busireddy KK, ALObady M, Ramalho M, Kalubowila J, Baodong L, Santostagino I, et al. Pancreatitis-imaging approach. World J Gastrointest Pathophysiol 2014; 5: 252–70. doi: https://doi.org/10.4291/wjip.v5.i3.152

Tirkes T, Fogel EL, Sherman S, Lin C, Swenson J, Akiki F, et al. Detection of exocrine dysfunction by MRI in patients with early chronic pancreatitis. Abdominal Radiology 2017; 42: 544–51. doi: https://doi.org/10.1007/s00261-016-0917-2

Tirkes T, Lin C, Fogel EL, Sherman SS, Wang Q, Sandrasegaran K, Tj, mapping for diagnosis of mild chronic pancreatitis. J Magn Reson Imaging 2017; 45: 1171–6. doi: https://doi.org/10.1002/jmri.25428

Shi Y, Cang L, Zhang X, Cai X, Wang X, Ji R, et al. The use of magnetic resonance elastography in differentiating autoimmune pancreatitis from pancreatic ductal adenocarcinoma: a preliminary study. J Magn Reson Imaging 2018; 47: 1309–13. doi: https://doi.org/10.1002/jmri.25428

Triantopoulou C, Dervenis C, Giannakou JG, Papadopoulos D, Groove pancreaticitis: a diagnostic challenge. European Radiology 2009; 19: 1736–43. doi: https://doi.org/10.1007/s00330-009-1332-7

Wolske KM, Ponnatapura J, Kolokythas O, Burke LMB, Tappouni R, Lalwani N. Chronic pancreatitis or pancreatic tumor? A problem-solving approach. RadioGraphics 2019; 39:
