

Imaging Assessment of Etiology and Severity of Acute Pancreatitis

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Abstract

Acute pancreatitis is a polymorphic disease with dynamic imaging characteristics and a multitude of possible complications established on cross-sectional imaging. Effective utilization of imaging in patients with acute pancreatitis requires a profound knowledge of the natural course of disease and familiarity with the subtypes and complications of acute pancreatitis. Imaging, primarily computed tomography (CT), in acute pancreatitis has various aims. CT can confirm the diagnosis or provide an alternative diagnosis, identify the etiology of pancreatitis, detect local pancreatic and extrapancreatic complications, offer prognostic information, and guide therapeutic interventions. Conventional radiography, ultrasound (US), endoscopic retrograde cholangiopancreatography (ERCP), and magnetic resonance imaging (MRI) have important complementary roles in the assessment and management of patients with acute pancreatitis. This chapter will discuss the role of imaging in the assessment of etiology of acute pancreatitis. Also, imaging characteristics of those with predicted severe disease and those with pancreatic and extrapancreatic complications directly influencing individual patient management will be addressed.

1. Introduction

The incidence of acute pancreatitis continues to increase worldwide, in parallel with an increasing demand on imaging resources to

evaluate the severity of disease. Imaging modalities available for assessment of acute pancreatitis include conventional radiography, abdominal ultrasound (US), multidetector computed tomography (CT), and magnetic resonance imaging (MRI). Of these, CT has become the standard of choice and worldwide the most commonly used imaging modality for the initial evaluation of acute pancreatitis and its sequelae (12, 14, 15, 104, 105). This chapter reviews the role of imaging in the evaluation of patients with acute pancreatitis. Emphasis will be on the use of imaging to assess the etiology and stage the severity of acute pancreatitis. This review applies only to cases of acute pancreatitis, not to chronic pancreatitis, flare-ups of chronic pancreatitis (i.e. acute-on-chronic pancreatitis), groove pancreatitis, auto-immune pancreatitis and other forms of pancreatitis (e.g. tuberculous, hereditary pancreatitis), which all differ considerably in clinical presentation, imaging findings, prognosis, therapy, and clinical outcome.

2. Imaging Modalities

The need for imaging in patients suspected of having acute pancreatitis largely depends on the severity of disease and clinical presentation. In patients with mild acute pancreatitis, imaging is rarely necessary for patient management, except for identifying the cause of acute pancreatitis. Conversely, those with severe

Table 1. Indications for cross-sectional imaging in acute pancreatitis

Early phase (< 1 week) <ul style="list-style-type: none">- To establish the correct diagnosis or provide an alternative diagnosis- To elucidate the etiology- To stage the morphologic severity- To assess for complications for those who deteriorate clinically or fail to improve
Late phase (> 1 week) <ul style="list-style-type: none">- To monitor established pancreatic collections- To delineate the presence of symptomatic and asymptomatic complications- To guide interventional procedures

acute pancreatitis often demand imaging for reasons stated in **Table 1**. Of all imaging modalities available, contrast-enhanced CT (CECT) is the standard technique for overall assessment of acute pancreatitis and its sequelae (35, 46, 53, 101, 116, 120). Other adjunctive imaging modalities include US, MRI, and angiography (92, 120). Angiography primarily is used to help diagnose the vascular complications of acute pancreatitis. This section will review the imaging techniques of US, CT, and MRI along with their advantages and disadvantages.

Role of US in Acute Pancreatitis

In the initial phase of acute pancreatitis, abdominal US is the primary imaging technique for assessment of biliary stones as the cause of acute pancreatitis and for assessment of the biliary tract (101, 115). Abdominal US is about 95% sensitive for the detection of cholecystolithiasis but only 50% sensitive for the detection of choledocholithiasis (99). At this stage, US enables allocation of patients that may benefit from a cholecystectomy (to prevent future attacks) and those requiring an endoscopic retrograde cholangiopancreatography (ERCP). US may also be used for detecting and monitoring pancreatic collections. Furthermore, US is useful for characterization of pancreatic collections by demonstrating necrotic debris within pancreatic collections, and thus, differentiating fluid from nonliquid material (120) With Doppler techniques vascular structures can be evaluated, particularly the presence of arterial

pseudoaneurysms. US can serve as an imaging guide during diagnostic or therapeutic interventions. Finally, US is the imaging technique of choice in children. US has various advantages: it is inexpensive, widely available, quick and easy to perform at the bedside or in an intensive care environment, and able to examine the pancreas in a variety of anatomical planes. US does not expose the patient to ionizing radiation and requires no potential hazardous intravenous contrast agents. Despite these advantages, there are several significant disadvantages that preclude US from being the primary imaging modality. The major disadvantage of US remains the limited visibility of the pancreas and peripancreatic region in a large proportion of patients with severe acute pancreatitis because of the presence of overlying bowel gas, particularly in case of ileus. The body habitus may also limit the penetration of acoustic waves in obese patients. Additionally, abdominal US is less accurate in delineating extrapancreatic inflammatory spread within retroperitoneal spaces and in detecting intrapancreatic necrosis. Finally, US is operator dependent and displayed on a limited number of images which are not easy to comprehend and convey to practicing clinicians.

Role of CT in Acute Pancreatitis

CT is at present the best imaging technique for the initial assessment and follow-up of patients with acute pancreatitis (**Table 1**)(12, 14, 15, 104, 105). Advantageous features of currently available multi-slice CT scanners are the high speed of acquisition with narrow collimation,

high image resolution, possibility of multi-planar imaging and reformats using volume data. Even in severely ill patients, CT will yield data of diagnostic quality that can be acquired during quiet respiration. Furthermore, CT is widely available, easily accessible in most institutions, less costly (than MRI), highly sensitive for the detection of gas bubbles and calcification, highly accurate, reproducible, and relatively easy to read by both radiologists and clinicians (**Figures 1, 2**). Indications to perform a CT varies considerably among different institutions in different geographic areas and is largely dictated by local preferences and cost factors. Some advocate performing CT on admission for staging purposes and triaging patients to different levels of care (82, 114). Others defer CT for the first week for several legitimate reasons (14, 46, 101, 116). First, early CT may underestimate the final morphologic severity of disease, as parenchymal necrosis may not be visible on CECT within 24-48 h after symptom onset (**Figure 3**) (6, 10, 16). On the other hand, a small number of patients will have a false-positive diagnosis for parenchymal necrosis due to interstitial edema and vasoconstriction of the vascular arcades. Repeat CT within a few days may show normal pancreatic enhancement. Second, CT at this stage will not have an impact on patient decision-making, unless the diagnosis is unclear. Third, only one out of four to five patients with acute pancreatitis will develop parenchymal necrosis, i.e. the majority will have morphologically mild findings (46, 53). Finally, the presence and extent of parenchymal necrosis shows no linear correlation with the development of systemic complications, such as organ failure (24, 43, 54, 80). However, urgent CT is indicated if an early complication of pancreatitis is suspected, primarily bowel ischemia or perforation. Conversely, at a later stage (after 3-7 days of hospitalization) patients who present with severe acute pancreatitis or who present initially with mild to moderate acute pancreatitis but fail to respond to supportive treatment should undergo abdominal CT (20). Serial CT enables following the evolution of

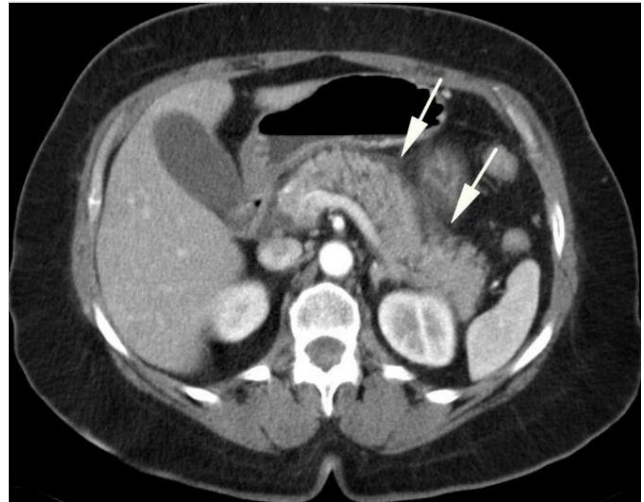


Figure 1. Acute interstitial pancreatitis. Normal enhancing pancreas with swelling and little peripancreatic fat stranding (arrows).

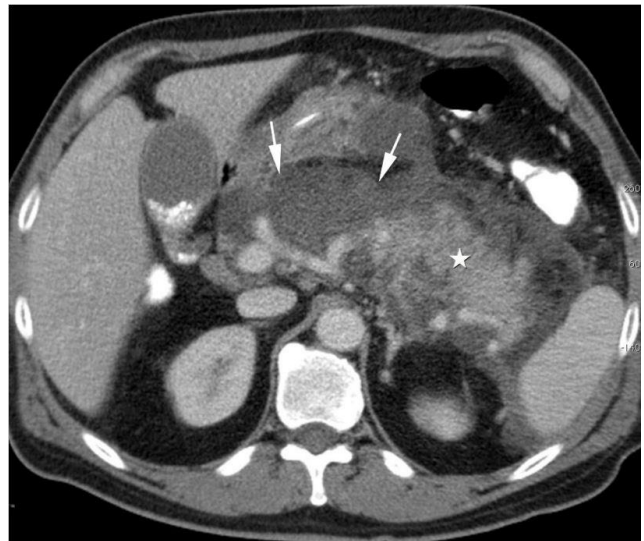


Figure 2. Acute necrotizing pancreatitis. CT shows nonenhancing parts of pancreatic head, neck, and body (arrows) with normal enhancing tail (asterisk). Note, stones in the gallbladder.

pancreatic collections and will delineate the extent of extrapancreatic inflammatory changes that will serve as a roadmap for interventional procedures like endoscopic, transabdominal, or minimal invasive surgical approaches. Imaging protocols vary in practice worldwide, but the common opinion is to obtain thin section images during the pancreatic (delay of 40-50 seconds) or portal venous phase (delay 60-70 seconds) (15, 92, 104, 105, 120). The use of intravenous contrast material is essential for detecting parenchymal necrosis and vascular complications. Yet, noncontrast CT still allows

for ascertaining the diagnosis and depicting pancreatic collections. Typically, the entire abdomen and pelvis is scanned to fully evaluate the extent of pancreatic collections and extrapancreatic abnormalities. A monophasic CT protocol after intravenous contrast administration is usually sufficient for the diagnosis, severity assessment, and for monitoring the progression of acute pancreatitis. Dual-phase studies are recommended in case of hemorrhage, mesenteric ischemia or suspicion of an arterial pseudoaneurysm or underlying pancreatic mass. CT has some important limitations. CECT is contraindicated in patients

who have intravenous contrast allergy or renal insufficiency. In addition, CECT compared with US, is less sensitive in identifying gallstones or biliary duct stones, a common cause of acute pancreatitis. Therefore, US is required if gallstones are not depicted on CT. The radiation dose may be significant in those requiring multiple CT examinations. Finally, although CT elegantly documents the extent of the pancreatic inflammatory process, it has limited capability of differentiating fluid from nonliquid material within peripancreatic collections (64). However, the aforementioned advantages of CECT clearly outweigh its limitations.

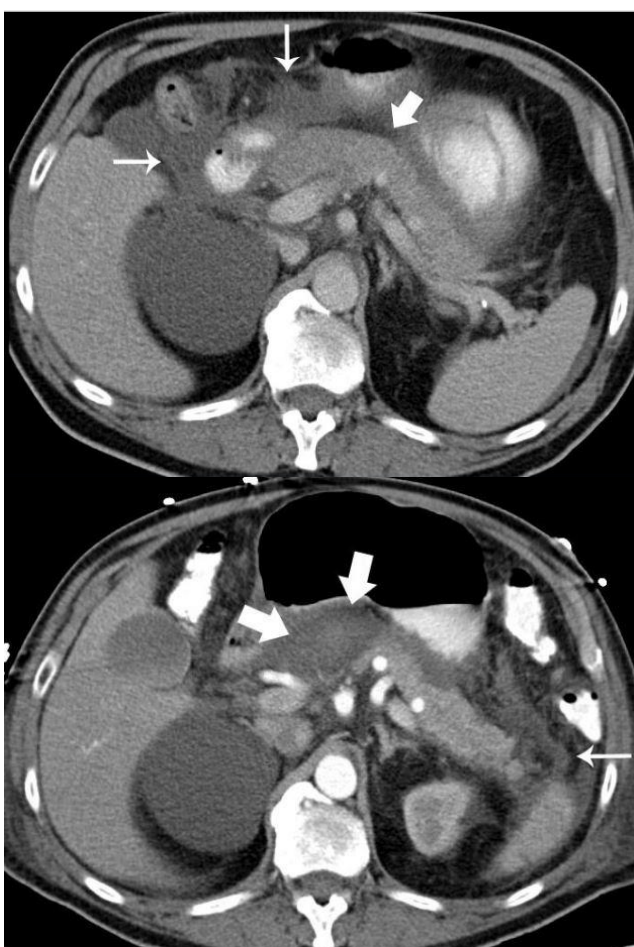


Figure 3. Pancreatic necrosis on day 1 (top) and day 5 (bottom). CT (top) performed on the day of admission shows a normal enhancing pancreatic parenchyma (thick arrow) with little peripancreatic fluid (thin arrows). Follow-up CT on day 5 (bottom) shows necrosis of pancreatic head and neck (thick arrows) and an acute necrotic collection in the left retroperitoneal space (thin arrow).

Role of MRI in Acute Pancreatitis

Over the years, MRI has gained a more prominent role in the assessment of acute pancreatitis. The presence and extent of pancreatic necrosis and peripancreatic collections can be evaluated with equal accuracy compared with CECT. In fact, MRI is better in detecting mild acute pancreatitis and elucidating the cause of acute pancreatitis with high sensitivity and specificity for choledocholithiasis and congenital pancreatic anomalies (**Figure 4**) (2, 59, 98, 117, 118). Due to its inherent tissue contrast resolution capability, MRI is superior to CECT in internal characterization of pancreatic collections (i.e.

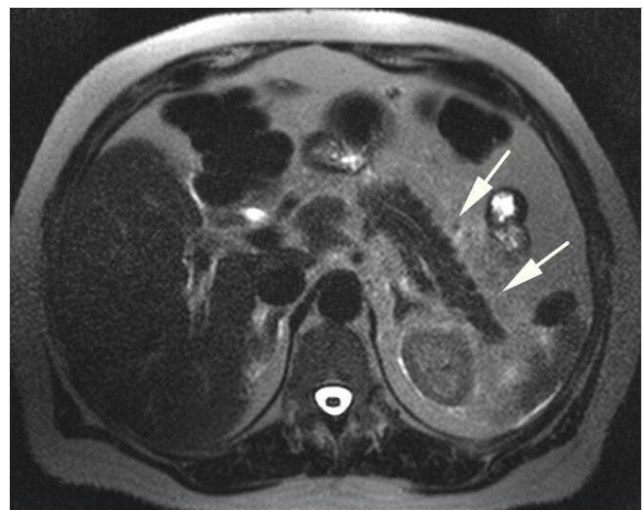


Figure 4. MRI of interstitial pancreatitis. T2-weighted sequence depicts little peripancreatic edema (arrows) around the pancreatic body and tail.

delineating the presence and extent of necrotic material) (64). Indeed, findings on MRI have been shown to accurately predict drainability of collections (**Figure 5, 6**). In addition, MRI is capable of detecting pancreatic duct disruption by using MR cholangiopancreatography (MRCP) (88). In approximately 30% of patients with severe acute pancreatitis, disruption of the pancreatic duct is observed, which heralds important prognostic and therapeutic information (34, 77). Finally, MRI is an excellent alternative

imaging modality in the setting of renal failure, young patients, and pregnant women. The major disadvantages of MRI include the longer scanning time (which can pose a problem for very ill patients), motion artefacts, the need for specialized MRI-compatible monitoring equipment in critically ill patients, lack of general availability (especially in urgent settings), and high costs if routinely used. Moreover, sensitivity of MRI in detecting gas bubbles is inferior to CECT, whereas image-guided percutaneous intervention is easier to perform with CT. Finally, MRI is more difficult to read and understand for



Figure 5. CT versus MRI in acute pancreatitis. CT (top) shows a heterogeneous collection in the transverse mesocolon with predominantly fluid density and fat density (arrowheads pointing at the borders). MRI (bottom) depicts more accurately the contents which consists of T2-weighted hypointense necrotic material without any significant amount of fluid (arrowheads pointing at the borders)



Figure 6. CT versus MRI of walled-off necrosis. CT (top) shows walled-off necrosis replacing a large part of the pancreatic parenchyma. Corresponding T2-weighted MRI (bottom) accurately depicts necrotic material (arrowheads) within the collection.

non-radiologists (compared with CT) given the multitude of sequences generally required for full evaluation. Therefore, at present, MRI is mainly used as problem solving tool in acute pancreatitis.

3. Imaging & Etiology

Determining the cause is essential in the assessment of all patients presenting with acute pancreatitis. First, elucidation of the cause may affect patient management significantly. An etiologic diagnosis may result in removal of the provocative factor and prevention of repeated insults; i.e. discontinuation of medication causing drug-induced pancreatitis. Second, some causes of acute pancreatitis have long-term consequences; i.e. acute alcoholic pancreatitis may result in recurrent and chronic pancreatitis with increased risk of pancreatic cancer, especially in those with a smoking history (73). Third, different etiologies have different natural courses with different complications; i.e. acute biliary pancreatitis requires a cholecystectomy or endoscopic intervention (5, 52, 108).

Despite a wide variety of etiologies of acute pancreatitis, gallstones and alcohol abuse account for about 75-80% of all causes (11, 46, 53). The relative rate of gallstones versus alcoholism as the cause of pancreatitis highly depends on patient's age and the geographic area. Other causes include hypercalcemic states (of which the most commonly recognized condition is hyperparathyroidism), hypertriglyceridemia, hereditary pancreatitis, trauma including post-procedural trauma (i.e. ERCP) or surgery, drug induced pancreatitis (i.e. thiazide diuretics, steroids, and azathioprine), and rare causes like scorpion venom. With thorough evaluation the cause of acute pancreatitis can be identified in 85-90% of cases, leaving about 10-15% of cases as idiopathic applying to patients with confirmed pancreatitis in whom a causative agent cannot be identified (11).

While many causes of acute pancreatitis require a detailed assessment of clinical history and biochemical evaluation, some causes are suggested or identified by imaging. In the following section, causes of acute pancreatitis depicted by imaging will be outlined.

Biliary

The diagnosis of biliary lithiasis is straightforward when gallstones are seen at abdominal US; gallstones appear as intraluminal, echogenic, mobile foci that are gravity-dependent and create a clean acoustic shadow. US has a sensitivity and specificity of around 95% for depicting gallstones and is the preferred imaging modality as CT shows significant lower sensitivity (of around 75%) (99). A repeat abdominal US is advised in those with "idiopathic" acute pancreatitis as gallstones may be missed on the initial evaluation (93). Because of the superior sensitivity an abdominal US should be performed in every patient presenting with acute pancreatitis early in the disease course to rule out gallstones as possible etiology. However, acute biliary pancreatitis may also be due to microlithiasis or biliary sludge (defined as stones smaller than 2 mm), which can be difficult to diagnose by abdominal US, but may be responsible for recurrent episodes of acute pancreatitis (99, 112). Biliary sludge is a viscous suspension of bile fluid that includes small stones, cholesterol monohydrate crystals, or calcium bilirubinate particles. Most patients who have biliary sludge are asymptomatic. Yet, biliary sludge is detected with increasing frequency in patients who have acute, otherwise idiopathic, pancreatitis (113). Although controversial, many institutions perform cholecystectomy for repeated episodes of otherwise idiopathic pancreatitis associated with biliary sludge. On CT gallstones appear as single or multiple filling defects within the gallbladder. Gallstones may have varying densities on CT depending on the composition (**Figures 7, 8**). Stones may be densely calcified, rim calcified or laminated or have a central nidus of calcification. Stones also may present as a

soft-tissue density or a lucent filling defect within the bile. Some stones may contain gas. In about 25% of cases, stones are isodense to fluid and therefore not identifiable on CT (99). MRI is an excellent, but costly alternative for US for depicting stones (larger than 4-5 mm) in the gallbladder or common bile duct (**Figure 9**). If a biliary etiology of acute pancreatitis is not diagnosed, the risk of pancreatitis recurrence is

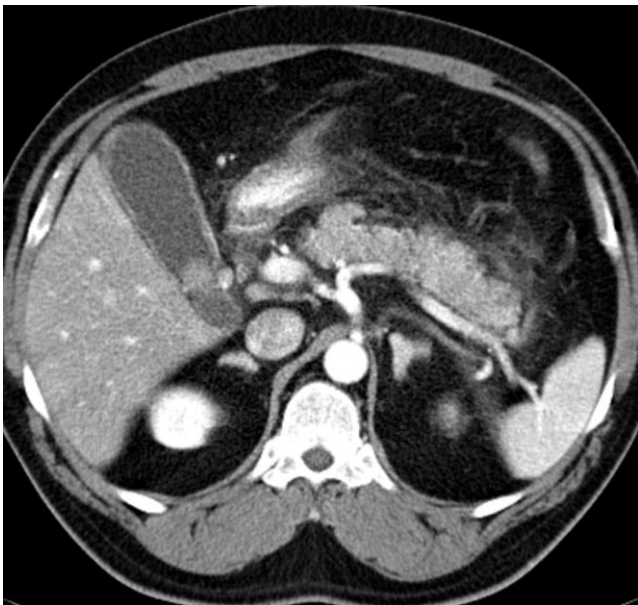


Figure 7. Biliary pancreatitis. Hyperdense stone is present in the gallbladder in a patient with interstitial pancreatitis.



Figure 8. CT of choledocholithiasis. Unenhanced CT depicts a calcified stone in the common bile duct (arrow) at the level of pancreatic head (asterisk) with little peripancreatic fat stranding (arrowheads) compatible with interstitial pancreatitis.

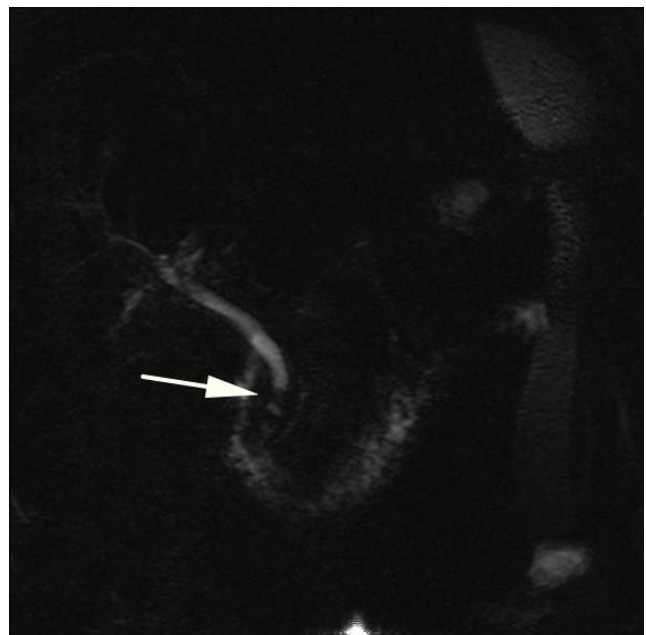


Figure 9. MRCP of choledocholithiasis. Heavily T2-weighted 3D sequence depicts 2 filling defects in the distal part of the common bile duct (arrow) representing stones.

about 30% after 6 months follow-up with variable severity (102). Hence, current guidelines advocate performing cholecystectomy during hospitalization in those with mild acute pancreatitis (115).

Cross-sectional imaging may show secondary findings suggesting a biliary cause of pancreatitis. The ‘choledochal ring’ sign, defined as hyperenhancement of the common bile duct wall relative to the pancreatic parenchyma (difference of more than 15 HU), has been reported to be indicative for a biliary cause of acute pancreatitis (29). However, the sensitivity of this finding was not significant in the study by Yie et al (119) and needs to be validated in large-scale studies. In this study, some other CT features were significantly associated with biliary pancreatitis, including pericholecystic fluid or fat stranding, pericholecystic increased attenuation of the liver, increased gallbladder wall enhancement, and gallbladder wall thickening (45, 119). Further study is needed to validate these results.

Traumatic

Pancreatic injury is more commonly seen in children than in adults and occurs in less than 2% of all abdominal injuries with associated mortality ranging from 9-34% (19, 27, 47, 57, 61). Early mortality is caused by massive hemorrhage (often due to concomitant organ injuries) and late mortality by multi-organ dysfunction and/or sepsis (19, 47). The low rate of pancreatic injury after abdominal trauma is related to its retroperitoneal location. Isolated pancreatic injury is less commonly seen than concomitant duodenal and pancreatic injury. Coexisting injuries are often present owing to the central location of the pancreas and the close relationship with surrounding organs and vessels. Injury to the pancreas can cause acute pancreatitis (posttraumatic pancreatitis) that may present with equivocal clinical symptoms and laboratory findings, often masked by other organ injuries (19, 27, 47). Posttraumatic pancreatitis should be considered when patients present with abdominal pain, nausea, and vomiting associated with increased serum amylase levels after blunt abdominal trauma. Contrast-enhanced CT is the primary imaging modality in abdominal trauma as it may diagnose posttraumatic pancreatitis and readily depicts accompanying traumatic injuries to other parenchymal organs, vessels, and bony structures (57, 61). Posttraumatic pancreatitis is likely in the right clinical setting combined with imaging features of pancreatitis. CT features of posttraumatic pancreatitis vary with the impact and severity of abdominal trauma and ranges from normal findings, mild pancreatic swelling, and exudate or soft tissue infiltration in the retroperitoneal spaces and mesenteries to hypo-enhancement of pancreatic parenchyma (representing contusion) or frank pancreatic transection with associated hemorrhage, fluid exudate, and duct disruption. Most CT findings in posttraumatic pancreatitis lack specificity and are often indistinguishable from pancreatitis of other etiologies, except for transection or laceration (depicted as a hypoattenuating linear density perpendicular oriented to the long axis of

the pancreas) and fracture of the pancreas (clear separation of pancreatic fragments). Similar to findings of non-traumatic pancreatitis, CT findings of traumatic pancreatitis are time dependent: CT may show near normal findings in 20-40% of cases during the first 12 hours after trauma with progressive changes on serial CT (57, 61). These subtle findings may be overlooked initially especially when coexistent organ injuries are present. Therefore, repeated imaging (CT or MRI) is warranted in those with sustained abdominal pain despite normal findings at index CT (57, 61). A diligent search for ductal injury should be undertaken in every patient with blunt abdominal trauma and posttraumatic pancreatitis as its integrity dictates clinical management: when intact, a conservative management is maintained, whereas a disrupted duct necessitates urgent surgical intervention. Delays in diagnosis and treatment of ductal injury results in subsequent increases in morbidity and mortality (19, 27, 47, 57, 61). The main pancreatic duct is most prone to injury from blunt trauma at the pancreatic neck or body as it traverses the vertebral column. Minor or major pancreatic duct rupture can cause pancreatic ascites from leakage of pancreatic fluid into the lesser and greater peritoneal compartments. Ductal injury can be diagnosed non-invasively by CT or MRCP and semi-invasively by ERCP. On CT, ductal injury can be inferred when a pancreatic laceration of more than one-half the pancreatic diameter is observed or in case of a complete transection or pancreatic fracture along the expected course of the pancreatic duct. A characteristic telltale sign of ductal injury is the presence of a posttraumatic pancreatic collection or pseudocyst. Occasionally, MRCP may be a helpful non-invasive adjunct to emergency abdominal CT to better assess pancreatic duct integrity. A long-term complication of posttraumatic pancreatitis is ductal scarring and stenosis, which may cause obstructive pancreatitis proximal to the stricture.

Pancreatic Neoplasms

Obstructive causes of acute pancreatitis due to pancreatic neoplasms involve periampullary tumors, cystic and solid pancreatic tumors, of which pancreatic adenocarcinoma is the most frequent and challenging diagnosis given the narrow therapeutic window for curative surgery. The incidence of solitary or recurrent attacks of acute pancreatitis associated with pancreatic adenocarcinoma is estimated to be 3-5% (7, 30, 33, 72, 106). Pancreatic cancer may cause pancreatitis because of pancreatic duct obstruction. Yet, the triggering mechanism of acute inflammation is incompletely understood as a minority of patients with pancreatic adenocarcinoma develop pancreatitis. Fortunately, pancreatitis resulting from underlying malignancy is usually mild (interstitial pancreatitis) such that curative resection is still possible (**Figure 10**). Necrotizing pancreatitis caused by pancreatic adenocarcinoma is rarely reported and notoriously difficult to diagnose and treat, as the extensive peripancreatic changes associated with necrotizing pancreatitis would likely render curative resection impossible in the majority of cases (121). Pancreatic adenocarcinoma as the cause of pancreatitis is surrounded by pitfalls in clinical presentation and diagnostic imaging features leading to delays in correct diagnosis and appropriate treatment (7, 30, 72, 106). Often, the diagnosis of an occult pancreatic adenocarcinoma is masked by the clinical presentation of signs and symptoms of acute pancreatitis. Also, on imaging, features of the inflammatory process may hamper the visualization of a pancreatic mass. On CT, primary diagnostic signs for pancreatic adenocarcinoma are an infiltrating irregular hypovascular mass, signs of invasion of surrounding organs and vascular structures, necrotic regional lymphnodes, and metastases in liver or peritoneum (7). Suspicious secondary imaging findings are an abrupt stop of the pancreatic duct with upstream duct dilation (whether or not with associated atrophy of pancreatic parenchyma), as this is rarely, if at all, seen in acute pancreatitis of benign cause. In most published reports, pancreatic



Figure 10. Interstitial pancreatitis due to pancreatic adenocarcinoma.

A slightly dilated pancreatic duct (top) is noted which ends abruptly due to a hypovascular mass in the body of the pancreas (bottom). Mild exudate is present in the left retroperitoneal space. Patient underwent surgery and pancreatic adenocarcinoma was confirmed at pathology.

adenocarcinoma has not been suspected clinically with a delay of diagnosis up to 12-24 months (7, 30, 72, 106). In patients with worrisome clinical symptoms such as new-onset of diabetes, jaundice, high bilirubin levels, recurrent attacks of 'idiopathic' pancreatitis (unknown or uncertain etiologies), and weight loss, complimentary tests are warranted to rule out pancreatic cancer (30, 106). Also, in patients with suspicious findings on regular CT, a short interval (2-3 weeks) follow-up study is needed to ascertain the right diagnosis. Complimentary imaging by means of EUS and/or MRI (depending on availability and expertise) is excellent in defining the morphology of

pancreatic duct, the nature of obstructive lesion, and depicting the presence of a pancreatic mass in case of equivocal CT findings.

Congenital Pancreatic Anomalies

The following two etiologies (pancreas divisum and annular pancreas) occasionally cause acute pancreatitis. The association between these congenital pancreatic anomalies and acute pancreatitis remains, however, controversial.

Pancreas divisum is the most common congenital pancreatic duct anomaly with a reported prevalence of 2-14% in the normal population (18, 23, 55, 69, 74, 107). Pancreas divisum represents a fusion anomaly in which the dorsal (containing the Santorini duct) and ventral (containing the Wirsung duct) pancreatic anlagen fail to fuse. Accordingly, the ventral (Wirsung) duct drains only the pancreatic head via the major papilla, whereas the majority of the pancreas drains via the minor papilla through the dorsal (Santorini) duct. It is assumed that drainage via the smaller calibre minor papilla into the duodenum may result in structural and functional outflow obstruction leading to pain and/or pancreatitis. Pancreas divisum is a definite cause of acute pancreatitis only when associated with ductal hypertension from increased resistance to flow through a proximally narrowed pancreatic duct and delayed clearance of injected contrast during ERCP (23, 55). Pancreas divisum is usually asymptomatic and the clinical relevance has been the subject of considerable debate. However, it is undoubtedly more frequently diagnosed in patients with repeated episodes of acute pancreatitis and chronic pancreatitis than in the general population. Yet, the incidence of pancreatitis in patients with pancreas divisum is low (about 5%) as ductal narrowing at the papillary origin is infrequently observed (23, 55). Pancreatic divisum can be confidently diagnosed semi-invasively by ERCP and non-invasively by MRCP. MRCP with secretin stimulation may depict inadequate outflow of pancreatic secretions through the minor papilla.

In the normal population, multidetector CT (with its high spatial resolution and thin collimation) also allows for accurate assessment of pancreas divisum when the dorsal (Santorini) duct courses directly from the tail and body of the pancreas through the anterior part of the pancreatic head draining into the minor papilla without evident connection with the ventral duct. However, inflammatory changes of the pancreas (such as pancreatic oedema, swelling, and necrosis) often preclude accurate CT assessment of ductal anatomy in patients with acute pancreatitis (3). Recognition of cross-sectional findings suggestive for pancreas divisum can guide patient management by recommending ERCP evaluation and assessment of minor papilla function. Possible treatments include stent placement in the minor papilla or minor papillotomy.

Annular pancreas is an uncommon congenital migration anomaly (1/20,000) where a ring of pancreatic tissue most commonly encircles the second part of the duodenum (87). Annular pancreas is usually diagnosed during infancy (with severe duodenal obstruction requiring urgent surgery), but clinical manifestations may develop at any age. Pancreatitis due to annular pancreas is often focal, confined to the pancreatic head and likely relates to the obstruction of pancreatic secretions through the annular duct (Santorini duct). In infants, the diagnosis is usually made by upper gastrointestinal double-contrast studies (with the classic 'double-bubble' sign, i.e. proximal dilation of both duodenum and stomach) or gastroduodenoscopy (with concentric narrowing and prestenotic duodenal dilatation). In adults presenting with pancreatitis, annular pancreas can be depicted on CT as a ring of inflammatory tissue (isodense with pancreatic parenchyma) surrounding the descending duodenum. Sometimes CT may show an annular duct (Santorini) also encircling the duodenum. EUS and MRI can be valuable for the diagnosis too.

Ischemic and Postoperative

Ischemic and postoperative pancreatitis are rare etiologies of acute pancreatitis (11, 23). Although their mechanisms in inducing acute pancreatitis are intimately intermingled, independently they may account for an acute episode of pancreatitis. The common denominator in the pathogenesis of both etiologies is the disturbance of pancreatic microcirculation; i.e. the decrease of capillary perfusion and hemoglobin desaturation, which relate to the duration of both ischemia and reperfusion. The pancreas is highly susceptible to ischemia/reperfusion injury as established by experimental studies and in clinical settings such as cardiopulmonary bypass surgery and hemorrhagic shock (38, 60, 81, 86). Important components in the pathophysiology of ischemia/reperfusion-induced acute pancreatitis include release of oxygen free radicals, activation of polymorphonuclear leukocytes, cellular acidosis, disturbance of intracellular homeostasis, and compromised pancreatic microvascular perfusion. These factors both induce and propagate premature intracellular activation of autodigestive pancreatic proteases and the resultant inflammatory response. Pancreatic ischemia may occur as a secondary event and, as such, may aggravate acute pancreatitis severity caused by other etiologies, but may also be the primary initiator of acute pancreatitis (38, 60, 81, 86).

Postoperative pancreatitis may occur after a variety of surgical procedures, among these are intra-abdominal procedures (such as common bile duct exploration, sphincteroplasty, distal gastrectomy, splenectomy, and organ transplantation) and operations distant from the gastrointestinal tract; both after major surgery like cardiovascular surgery, spinal, vascular, and esophageal surgery, but also after relatively minor procedures that do not involve manipulations near the pancreas, such as thyroidectomy, parathyroidectomy, and inguinal hernia repair (13, 22, 79).

Possible factors linking these surgical procedures with acute pancreatitis include drugs (medication during cardiopulmonary bypass surgery, immunosuppressive drugs in organ transplantation), intraoperative or postoperative periods of low flow or hypotension resulting in reduced splanchnic flow and impaired pancreatic vascularization, thromboembolic events, mechanical factors (direct pancreatic, duodenal or biliary manipulation), and metabolic factors.

The spectrum of symptoms associated with ischemia-induced acute pancreatitis may vary from asymptomatic hyperamylasemia (e.g. after cardiopulmonary bypass) to clinically severe disease as in hemorrhagic shock. The definition and diagnosis of ischemia-induced acute pancreatitis are difficult to determine and often delayed (13, 22, 38, 60, 79, 81, 86). Clinical symptoms of acute pancreatitis may be masked after major surgery in patients who are mechanically ventilated, sedated, and/or receive narcotic analgesics. Ischemic acute pancreatitis should be considered in patients who develop abdominal pain and signs of sepsis after an episode of prolonged hypotension and/or visceral hypoperfusion, especially in those after cardiac or major surgery or who unexpectedly deteriorate rapidly postoperatively (13, 22, 38, 79). Imaging studies are necessary when the diagnosis of acute pancreatitis is uncertain. CT is a valuable objective imaging modality for the evaluation of patients with suspected ischemic or postoperative pancreatitis. In postoperative patients, CT may show findings of acute pancreatitis (with or without parenchymal necrosis) with peripancreatic collections that show varying degrees of encapsulation due to the often delayed diagnosis. Also, it is important to bear in mind that in patients with ischemic acute pancreatitis, a possible coexistence of intestinal ischemia may occur, in particular of the right hemicolon, the transverse colon, or the gallbladder. Furthermore, special attention should be paid to the patency of the portomesenteric venous structures as well as

the celiac trunk and superior mesenteric artery (i.e. high grade stenosis, occlusion, or emboli) (13, 22, 38, 79).

In conclusion, the diagnosis of ischemic or postoperative pancreatitis requires a high index of suspicion. Increased clinical awareness perioperatively appears to be the most effective strategy for early diagnosis and timely treatment of acute ischemic pancreatitis following cardiac or major vascular surgery. Liberal use of diagnostic imaging modalities, primarily CT, to establish an early diagnosis and institution of appropriate therapy is, therefore, warranted.

Miscellaneous Findings

Steatosis of the liver may be seen in patients with an alcoholic etiology or metabolic disturbances such as hypertriglyceridemia, but may also be a pre-existent condition (in case of obesity or usage of medication) and, therefore, lacks specificity (**Figure 11**). The presence of liver abnormalities characteristic for cirrhosis (caudate lobe hypertrophy, lobularity of liver contour, venous collaterals, splenomegaly) may, however, suggest an alcoholic etiology.

4. Diagnostic Algorithm for Assessing Etiology

The standard work-up of the cause of acute pancreatitis may vary significantly among different centers based on personal experience and acquired skills, available equipment, and institutional strengths and weaknesses. Timing and the individual contribution of available imaging tests (US, EUS, CT, MRI/MRCP, and ERCP) are subject to debate and mainly driven by individual preferences. However, based on current available evidence and recommendations according to established guidelines, an abdominal US is advised in all patients presenting with acute pancreatitis, both at first presentation and in recurrent episodes of otherwise idiopathic pancreatitis (23, 55, 101, 116). Depending on expertise, availability, and

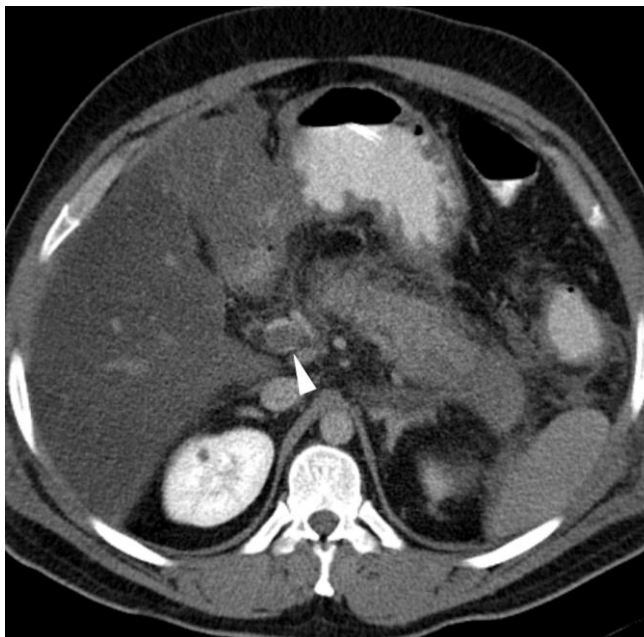


Figure 11. Hepatic steatosis in drug-induced pancreatitis. Markedly hypodense liver parenchyma is seen representing severe hepatic steatosis in a patient with necrotizing pancreatitis and a thrombus in the portal vein (arrowhead).

local practices, further testing by means of EUS or MRCP is indicated as a next step if US is negative but the clinical suspicion for a biliary etiology is high. Additional imaging (i.e. state-of-the-art multidetector CT, EUS, and/or MRI/MRCP) is especially warranted in patients over 40-50 years of age with “idiopathic” acute pancreatitis or repeated episodes of acute pancreatitis to exclude a pancreatic neoplasm as possible cause of the pancreatitis.

5. Imaging & Severity

Acute pancreatitis is a serious disease with varying severity. The recently revised Atlanta Classification 2012 on acute pancreatitis (RAC) classified the severity of acute pancreatitis clinically (on the basis of presence or absence of organ failure) and morphologically (on the basis of presence or absence of tissue necrosis) (12). Morphologically (i.e. on imaging), two types of pancreatitis are discriminated; interstitial pancreatitis (no tissue necrosis) and necrotizing pancreatitis (tissue necrosis).

Interstitial pancreatitis

Interstitial pancreatitis is usually a self-limiting disease with a short hospitalization stay and represents the most common form of acute pancreatitis (46, 53). These patients typically recover uneventfully without complications. On imaging, interstitial pancreatitis may reveal a minimal increase in size of the pancreas, focally or diffusely (**Figure 12**). The pancreatic contour becomes irregular with inflammatory changes; the peripancreatic fat planes become blurred with increased attenuation values. Peripancreatic extension of the inflammatory process is relatively common because the pancreas lacks a well-defined capsule. Thickening of the small bowel mesentery, renal



Figure 12. Interstitial pancreatitis
CT (top) depicts a swollen and slightly heterogeneous enhancing pancreatic parenchyma with fluid in the peripancreatic and retroperitoneal spaces (asterisks).
Follow-up CT 9 days (bottom). later shows resolution of fluid and normalization of pancreatic parenchyma.

fascia, and lateroconal fascia is common. More severe forms of interstitial pancreatitis can result in moderate amounts of peripancreatic fluid (15, 104, 105). Morbidity from interstitial disease ranges about 10% with mortality less than 3%, primarily due to co-morbid disease (95).

Necrotizing pancreatitis

Necrotizing pancreatitis is associated with a protracted clinical course, long hospital stay with a high morbidity (30-80%), and a mortality rate up to 20-30% (111). The 2012 revised Atlanta Classification distinguishes three subtypes of necrosis depending on involvement of pancreatic parenchyma alone (rare), peripancreatic tissues (extrapancreatic necrosis or EXPN, more common), or the combination of both (combined necrosis, most common) (12). Pancreatic parenchymal necrosis tends to occur early in the course of the disease, within the first 48-72 h after symptom onset. CT criteria for the diagnosis of pancreatic parenchymal necrosis are dependent on the detection of areas lacking enhancement, which may be focal or diffuse (**Figure 13**). Lack of pancreatic enhancement corresponds with decreased blood perfusion of the pancreatic gland and correlates well with necrosis. Accuracy for depicting areas of pancreatic parenchymal necrosis is excellent when the region measures at least 3 cm or larger in diameter or involves more than one-third of the gland. Caution in defining pancreatic



Figure 13. Acute necrotizing pancreatitis. CT shows extensive necrosis involving more than 90% of pancreatic parenchyma with associated acute necrotic collections.

parenchymal necrosis is important as areas of intrapancreatic fluid or reversible ischemia can simulate areas of necrosis. Pancreatic parenchymal necrosis is ideally detected on scans performed >72 h after the onset of an attack of acute pancreatitis (14, 15, 104, 105). Scans done within this timeframe may be falsely negative or equivocal. EXPN is a relatively new subtype of necrotizing pancreatitis which has received increasing attention in the literature over the past years (4, 84, 85). Its diagnosis hinges on the detection of heterogeneous peripancreatic collections with preserved pancreatic parenchyma perfusion. On CT, EXPN is determined when a normally perfused pancreatic parenchyma is noted surrounded by collections composed of various densities (fat, fluid, and non-liquid Hounsfield units) (**Figure 14**). In general, EXPN heralds a better prognosis than combined necrosis when sterile, but similar prognosis when infection of necrotic tissue develops (4, 84).

Scoring Systems for Predicting Severity

The clinical course of acute pancreatitis is highly variable ranging from mild self-limiting symptoms to rapidly progressive organ dysfunction potentially culminating in death if not treated appropriately. Proper initial management includes transfer of patients to specialized centers or admission to intensive care units for supportive treatment or for targeted therapy (i.e. institution of tailored fluid resuscitation, endoscopic intervention, enteral nutrition, or new therapies as they become available). Besides the need from a clinical management perspective, there are other potential benefits for early severity prediction of acute pancreatitis. Accurate stratification is essential for reliable comparison of clinical outcomes among institutions, for evaluation of novel therapeutic strategies, and for inclusion of patients in randomized controlled clinical trials (12). Hence, considerable efforts have been targeted over the past decades to the early identification of those who will develop persistent organ failure in the

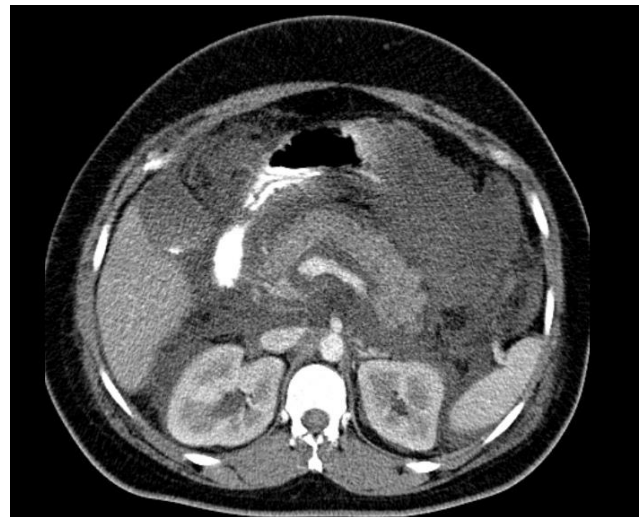


Figure 14. Extrapaneacreatic necrosis. CT depicts a normal enhancing pancreatic parenchyma surrounded by acute necrotic collections. Note, calcified stone in the gallbladder.

early stages and infected necrosis and sepsis in the later phase.

Prediction of disease severity can be done using thorough clinical evaluation including detailed assessment of established risk factors (such as age, obesity, and comorbid disease). However, based on clinical evaluation alone, even experienced physicians fail to diagnose those with severe acute pancreatitis in 30-50% of cases. Other means of determining the severity include the use of single prognostic indicators (e.g. serum blood urea nitrogen, creatinine, hematocrit, levels of C-reactive protein, procalcitonin) and the utilisation of multiple clinical scoring systems that incorporate physiologic and laboratory parameters (among these are the Ranson score, Systemic Inflammatory Response Syndrome (SIRS), Bedside Index of Severity in AP (BISAP), and Acute Physiology and Chronic Health Evaluation (APACHE)-II score). In a large dual-center study, the accuracy of all available clinical scoring systems in predicting the development of persistent organ failure (signifying severe acute pancreatitis) on the day of admission was prospectively studied using comparative effectiveness analysis. This study found that all clinical scoring systems failed to perform with

high performance characteristics and revealed only modest and comparable predictive accuracy (71). Finally, since the introduction of CT for diagnosis and assessment of acute pancreatitis some four decades ago, several imaging-based scoring systems have been proposed to predict the severity of acute pancreatitis.

Imaging-based scoring systems related to CT are the most studied and widely used because CT is regarded the frontline imaging modality for the overall assessment of acute pancreatitis. Determinants of most CT-based scoring systems include pancreatic, peripancreatic and extrapancreatic features. Pancreatic changes include the subjective or objective enlargement of the pancreatic gland and presence and extent of parenchymal necrosis. Peripancreatic features include fat stranding or oedema, (fluid) collection(s) (presence, number, and volume), perirenal oedema, mesenteric inflammation and retroperitoneal extension. Extrapancreatic features include the presence of ascites, pleural effusion, vascular, gastrointestinal, and/or extrapancreatic parenchymal organ complications. Over the past four decades, at

least 10 different radiographic scoring systems have been developed (**Table 2**) using incremental numerical scores or grades with higher scores or grades correlating with increasing morbidity and mortality (9, 10, 26, 41, 44, 49, 58, 62, 70, 89). Two of these evaluate the presence and extent of parenchymal necrosis (i.e. CT Severity Index (CTSI) and Modified CT Severity Index (MCTSI)) for which the use of intravenous contrast material is indispensable (10, 70). The remainder of scoring systems can be assessed on unenhanced CT scans. In **Table 2**, an overview of existing imaging-based scoring systems in order of year of development with the parameters evaluated and their respective advantages and limitations is depicted.

Among all radiographic scoring systems available, the CTSI is the most commonly used and studied (10). The CTSI combines the Balthazar grade (0-4 points) with the extent of pancreatic necrosis (0-6 points) on a 10-point severity scale (**Figure 15, Table 3**). The calculated CTSI can then be subdivided in three categories

Table 2. Radiographic scoring systems in acute pancreatitis

Radiographic scoring system	Year of development	CECT	CT parameters	Advantage(s)	Disadvantage(s)
Extrapancreatic score (EP or Schroeder index, range 0-7)	1985	-	Edema in part or entire pancreas, ascites, pleural effusion, perirenal fat oedema, mesenteric fat oedema, and bowel paralysis.	Relatively easy to assess Does not require intravenous contrast	Not validated for early use* Presence of ascites and perirenal oedema can be a normal finding Not extensively studied [†]
Balthazar Grade (A-E)	1985	-	Pancreatic swelling, peripancreatic fat stranding, presence and number of associated pancreatic collections	Relatively easy to assess Does not require intravenous contrast	Variable interobserver agreement, i.e. counting the number of collections
Pancreatic size index (PSI, cut-off 10 cm ²)	1989	-	Multiplying the maximum anteroposterior measurement of the head and body of the pancreas	Measurement of single parameter Does not require intravenous contrast	Normal size may vary depending on age and previous attacks Not extensively

					studied [¶]
CT Severity Index (CTSI, range 0-10)	1990	+	Balthazar grade + presence and extent of parenchymal necrosis	Most used and studied Depicts the order of morphologic severity in acute pancreatitis	Variable interobserver agreement for counting pancreatic collections and assessing % of necrosis
MOP score (range 0-2)	2003	-	Mesenteric oedema and peritoneal fluid (ascites)	Measurement of two parameters only Simple and easy to assess Does not require intravenous contrast	Not validated for early use* Ascites can be physiologic in female and elderly Not extensively studied [¶]
Modified CTSI (MCTSI, range 0-10)	2004	+	Pancreatic swelling or fat stranding, pancreatic collection(s), presence and extent of parenchymal necrosis, extrapancreatic complications including vascular, parenchymal, gastrointestinal organs and pleural effusion and ascites	Inherent simplifications Easier to assess for non-experienced readers	Does not outperform the original CTSI
Retroperitoneal Extension Grade (I-V)	2006	-	Extension of peripancreatic inflammation to retroperitoneal spaces	Does not require intravenous contrast	Advanced interpretative skills required Not extensively studied [¶]
EPIC score (range 0-7)	2007	-	Pleural effusion, ascites, retroperitoneal and mesenteric inflammation	Relatively easy to assess Does not require intravenous contrast	Original study biased towards severe disease Not extensively studied [¶]
Renal Rim Grade (A-C)	2010	-	Extension of peripancreatic inflammation to pararenal and / or perirenal space	Easy to assess Does not require intravenous contrast	Not extensively studied [¶]
EXPV Volume (cut-off 100 mL)	2014	-	Volume of extrapancreatic exudate or fluid	Objective Does not require intravenous contrast	Not validated for early use* Additional software required for calculating volume Not extensively studied [¶]

*: within 24 hours of admission; [¶]: less than 5 studies in English literature.

Table 3. Balthazar Grade and CT Severity Index (CTSI)

Characteristics	Balthazar Grade	CTSI
Pancreatic inflammation		
Normal pancreas		0
Focal or diffuse enlargement of the pancreas	A	1
Peripancreatic inflammation / fat stranding	B	1
Single acute fluid collection	C	2
Two or more acute fluid collections	D	3
	E	4
Pancreatic parenchymal necrosis		
None		0
Less than 30%		2
Between 30 and 50%		4
More than 50%		6

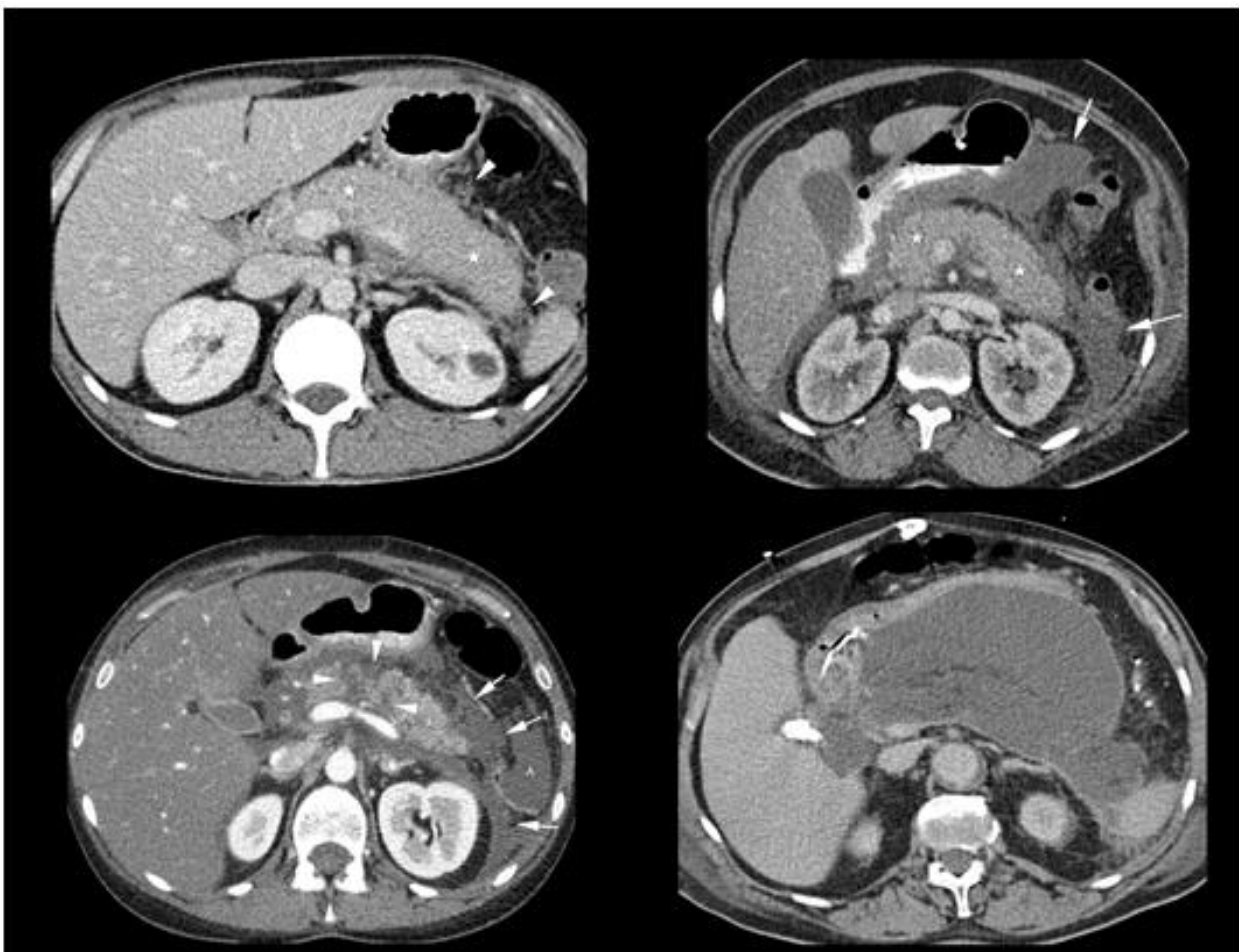


Figure 15. CT severity index.

CTSI (top left) of 2: swollen but normal enhancing pancreas (asterisks) with little peripancreatic fat stranding (arrowheads).

CTSI (top right) of 4: normal enhancing pancreatic parenchyma (asterisks) with more than 2 collections (arrows).

CTSI (bottom left) of 6: less than 30% nonenhancing pancreatic parenchyma at the level of pancreatic body (arrowheads) with associated necrotic collections (arrows).

CTSI (bottom right) of 10: extensive necrosis of more than 50% of pancreatic parenchyma with associated necrotic collections. Note, calcified stones in the gallbladder.

(CTSI 0-3, 4-6, and 7-10; corresponding to predicted mild, moderate, and severe disease, respectively) that have subsequent increases in morbidity and mortality (10). Main advantage of the CTSI is its intuitive design as it accurately depicts the order of increasing morphologic severity of acute pancreatitis. Interstitial pancreatitis is reflected by CTSI of 0 (normal pancreas), 1 (swelling of the pancreatic gland), and 2 (peripancreatic fat stranding). Extrapancreatic necrosis is potentially reflected by CTSI of 3 and 4 (1 or more pancreatic collections, respectively). In general, CTSI greater than 4 (i.e. CTSI 5-10) denotes the presence of pancreatic collections and parenchymal necrosis with more points accredited with increasing extent of necrosis. However, patients with less than 30% parenchymal necrosis without associated collections also have CTSI of 4, albeit this is a rare event.

Despite the profound heterogeneity in study design and the variable endpoints used among the different studies, all reports on the discriminatory power of radiographic scoring systems show a modestly positive correlation between the scoring system studied and patient outcome. Two recent studies compared the accuracy of several radiographic scoring systems, including the CTSI, and found comparable performance characteristics among the CT scoring systems studied in the prediction of disease severity and overall mortality (16, 91). Also, these studies show that CT scoring systems did not perform better than commonly used clinical scoring systems, such as BISAP and APACHE II score.

There are several explanations for the moderate performance characteristics of imaging-based scoring systems. First, the degree of morphologic abnormalities is largely influenced by the time interval between symptom onset and performance of the imaging study with increasing changes seen with increasing time

interval (with correspondent higher scores or grades). Second, radiographic scoring systems do not account for well-known risk factors, such as obesity, age, and pre-existent comorbid disease. Third, in a small but definite percentage of patients with acute pancreatitis, there is a non-linear relationship between morphologic findings and clinical severity. Also, some 30-40% of patients with parenchymal necrosis will have a relatively benign clinical course (without organ dysfunction or systemic complications) (16, 24, 43, 54, 80). Fourth, radiographic scoring systems correlate better with local complications (infected necrosis and need for intervention) than with systemic complications (primarily persistent organ failure, which signifies severe disease). Fifth, radiographic scoring systems are biased towards more severe disease as those with very mild symptoms often do not need or undergo cross-sectional imaging. Sixth, the use of most CT-based systems is confounded as reliable predictor by the subjective nature of its interpretation with variable interobserver agreement, which likely relates to readers' expertise and familiarity of imaging findings of acute pancreatitis. Seventh, as opposed to clinical scoring systems, radiographic scoring systems are not repeated routinely within a short time period such that an interval change in significant morphology may go unnoticed (e.g. interval detection of parenchymal necrosis on serial CT not visible on the index CT). Eighth, scoring systems (radiographic and clinical systems) do not correlate with the risk of specific extrapancreatic complications (e.g. abdominal compartment syndrome, bowel ischemia or perforation, or arterial pseudoaneurysm). Therefore, they fail to provide detailed information that instantly affects patient management on an individual basis. Finally, the fallacy of linking one imaging feature or a constellation of imaging features to severe clinical outcome falls short simply because of the intrinsic morbidity and mortality, albeit low in numbers, in patients with interstitial pancreatitis (95). Typically, in interstitial pancreatitis grave

imaging features are absent to foretell a dismal outcome. It is therefore unlikely that radiographic scoring systems will ever serve as an accurate means of correctly identifying all those with severe pancreatitis early on in the disease process. The limited efficacy of radiographic scoring systems for prognostication reflects the complexity, variability, and heterogeneity of acute pancreatitis with its myriad possible clinical expressions.

Clinicians need a powerful, simple, and easy to use predictive system early on in the disease process, preferably within several hours after admission, for directing patients to different levels of care or tailored therapy measures. Cross-sectional imaging studies performed within this timeframe will unlikely surpass clinical scoring systems as has been shown in aforementioned reports comparing the various radiographic scoring systems on the day of admission. In view of the abovementioned limitations of radiographic scoring systems, the added costs, efforts, and radiation burden associated with CT (32, 65, 96, 97), and the ease of use of some of the clinical scoring systems, it's the author's opinion that initial severity assessment should be based on clinical scoring systems rather than relying on imaging parameters. The decision about if and when to perform CT depends, therefore, on the overall clinical presentation. Undeniably, CT has its greatest merits in the later phase of the disease in those who have predicted severe acute pancreatitis by clinical assessment or those who do not improve clinically despite appropriate therapy when local complications (most commonly infection of necrotic tissue) largely direct clinical decision-making (20, 90).

6. Prognostic Cross-Sectional Imaging Findings

Irrespective of the etiologic factor, the degree of morphologic findings in acute pancreatitis depends on the severity of the attack and the time interval between onset of symptoms and

imaging. In general, morphologic findings are well-established 5-7 days after symptom onset. Mild disease presents with only mild pancreatic and peripancreatic abnormalities that resolve spontaneously. Severe disease presents with extensive peripancreatic abnormalities (including necrotic collections) and parenchymal necrosis, which may become infected and give rise to various extrapancreatic parenchymal, vascular, or visceral complications, potentially with significant impact on patient management.

Pancreatic Collections

In moderate to severe acute pancreatitis, pancreatic collections can accumulate in and around the pancreas. These collections may be single or multiple, vary in size, and lack a well-defined capsule initially, only confined by the anatomic space in which they arise. Many collections resolve spontaneously, but a certain percentage goes on to develop a complete wall, which usually takes around 4-5 weeks to develop. These collections may become symptomatic due to persistent pain, secondary infection or hemorrhage or by exerting mass-effect on surrounding structures (e.g. extrinsic biliary obstruction) (14, 35, 46, 53, 92, 101, 116, 120). Other complications include compression and occlusion of the splenic vein, which can result in extensive collaterization around the spleen and stomach. This may in time become a source of gastrointestinal bleeding. The most common sites of pancreatic collections are the lesser sac and left anterior pararenal space (14, 15, 104, 105). Larger collections can extend retroperitoneally over the psoas muscles to enter the pelvis and groin. Pancreatic collections may also involve the posterior pararenal space, perirenal space, transverse mesocolon, and small bowel mesentery. Notably, pancreatic collections should not be mistaken for areas where ascites reside, such as in the perihepatic and perisplenic areas, in the paracolic gutters, and pelvis. Management of pancreatic collections depend on the patient's clinical condition and whether they cause symptoms (**Figures 16, 17**).

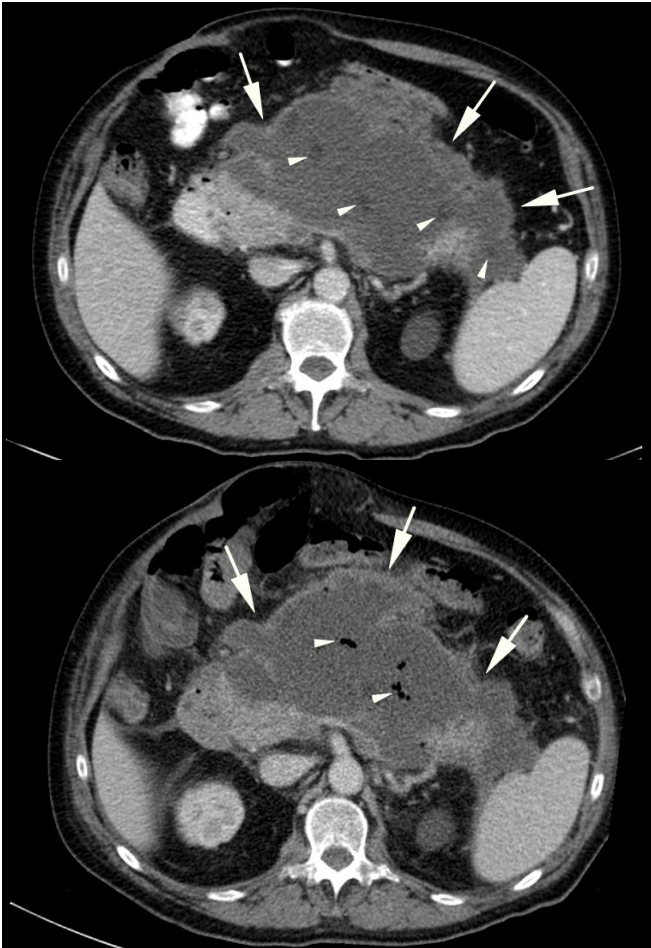


Figure 16. Walled-off necrosis (WON). CT (top) shows a fully encapsulated heterogeneous collection replacing a large part of pancreatic parenchyma (arrows pointing at the borders). Collection consist of fluid and non-liquid (fat) densities (small arrowheads). Follow-up CT (bottom) performed 1 week later because of fever now shows gas bubbles (small arrowheads) within the WON (arrows pointing at the borders), representing infected necrosis.

Pancreatic Necrosis

Pancreatic parenchymal necrosis represents a severe form of acute pancreatitis. In addition to the presence of parenchymal necrosis, its extent (particularly when more than 30% is involved in the necrotic process) has also been correlated with worse clinical outcome in some (16, 24, 54, 80), but not all (37, 42, 94), reports. The site of necrosis is deemed equally important, especially when the central part of the gland is involved with a viable pancreatic tail (**Figure 18**). Full thickness necrosis of the midgland (neck and/or body of the pancreas) may lead to pancreatic

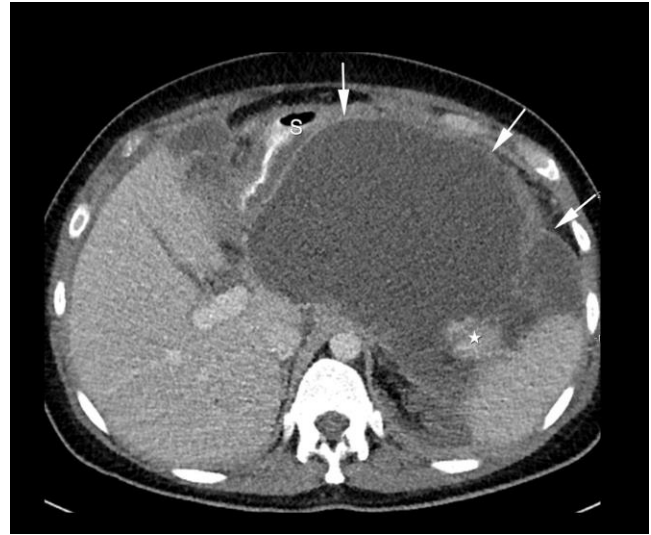


Figure 17. Gastric outlet obstruction due to large walled-off necrosis. CT shows a large fully encapsulated pancreatic collection (arrows pointing at the borders) which exerts mass effect on the stomach (S). Note, little preserved pancreatic parenchyma at the tail (asterisk).

duct disruption with increased need for intervention and definitive therapies to control the continuing secretion of pancreatic juice (48, 75). Isolated parenchymal necrosis is a rare event. In the majority of cases the necrosis is not confined to the pancreatic parenchyma alone, but often involves the peripancreatic tissues as well. Necrotic tissue or necrotic collections are prone to bacterial colonization from adjacent bowel structures with development of infected necrosis. Infected necrosis is regarded as one of the most feared local complications of acute pancreatitis, responsible for prolonged hospitalization, need for invasive intervention with high demand of health care resources (53, 111). Infected pancreatic necrosis is recognized at CT as bubbles of gas within areas of pancreas, or as a collection of gas and tissue within the retroperitoneum (**Figure 19**). Infected necrosis carries a grave prognosis compared with sterile necrosis with a two-to-threefold increase of mortality (101, 111, 116).

Vascular Complications

Vascular complications arising from acute pancreatitis include portosplenomesenteric



Figure 18. Central gland necrosis. CT three days after symptom onset (top) shows nonenhancement of the midgland with preserved pancreatic body and tail. Note, severe and longstanding ureteropelvic junction obstruction of the right kidney with loss of renal parenchyma. Follow up CT 3 weeks later (bottom) shows marked increase in size of necrotic collections exerting mass effect on the stomach and extending to the left retroperitoneal space.

venous thrombosis, arterial pseudoaneurysms, and hemorrhage due to vessel wall erosion by extravasated proteolytic pancreatic enzymes. Splenic vein thrombosis occurs most common and may result in complications such as gastric or esophageal varices and splenomegaly (left-sided portal hypertension) (**Figure 20**). Multiphasic CT accurately depicts sites of vascular thrombosis and demonstrates collateral vascular pathways (31, 39, 103). Erosion of arterial vessel wall initially results in a confined perivascular blood leak with subsequent arterial pseudoaneurysm formation. Injuries commonly involve the splenic artery, the

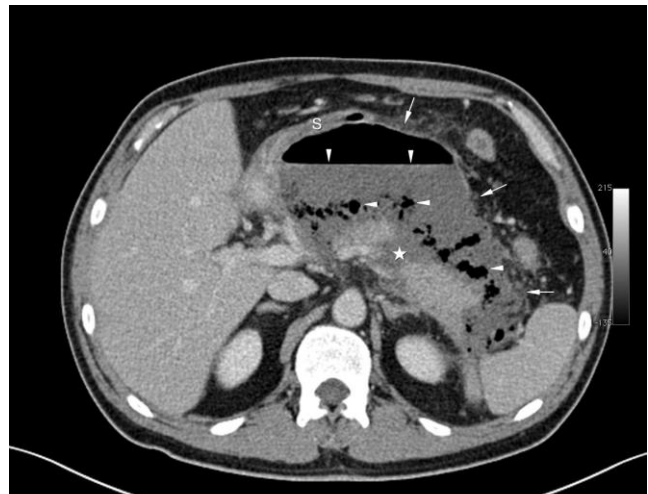


Figure 19. Infected pancreatic necrosis. CT shows a necrotic area at the junction of pancreatic body and tail (asterisk) with associated necrotic collections (small arrowheads pointing at the borders) which contains impacted gas bubbles (small horizontal arrowheads) in the retroperitoneal compartment and a gas-fluid level (small vertical arrowheads) in the lesser sac, signifying infection of necrosis. S: stomach.

pancreaticoduodenal or the gastroduodenal arteries, which are closely related to the pancreas. An arterial pseudoaneurysm is often the underlying etiology in cases of massive haemorrhage (8, 50, 68). CT with arterial phase multidetector CT or 3D CT angiography can routinely detect the presence and specific site of such pseudoaneurysms. Bleeding may also occur into a pre-existing pancreatic collection, often in areas of necrosis. Cross-sectional imaging is helpful in identifying the source of hemorrhage. Massive acute hemorrhage secondary to bleeding pancreatic collections or arterial pseudoaneurysm has an associated mortality rate of 10-35% (8, 50, 68). An easily overlooked complication on abdominal CT in patients who are bedridden because of their illness (i.e. not unique to acute pancreatitis) is the occurrence of deep vein thrombosis in the iliofemoral veins that may lead to pulmonary embolisms. In contrast to portosplenomesenteric vein thrombosis, this finding urgently necessitates the initiation of anticoagulant treatment.

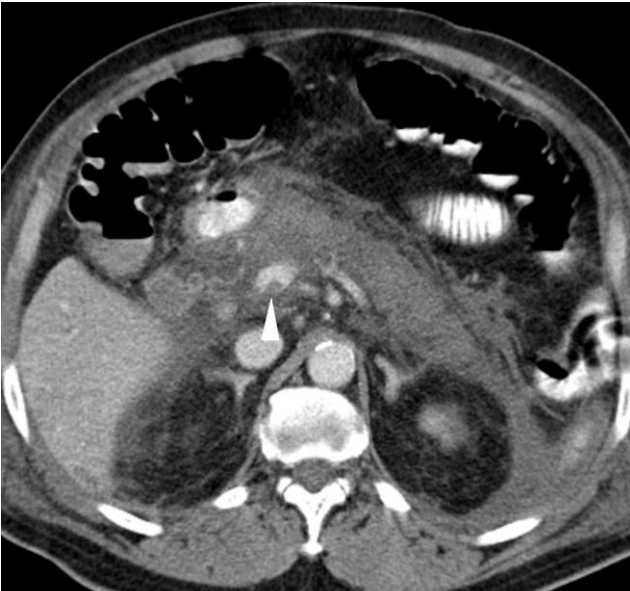


Figure 20. Portal vein thrombus in necrotizing pancreatitis. Small intraluminal filling defect is noted in the portal vein (arrowhead) in a patient with necrotizing pancreatitis.

Involvement of Extrapancreatic Organs

Typically, acute pancreatitis is a disease process where the inflammatory spread is not limited by adjacent organs, mesenteries, omentum, or peritoneal and retroperitoneal fascial planes. While pancreatitis most commonly involves the pararenal spaces and lesser sac it can extend to and involve adjacent organs.

Renal involvement is typically due to inflammatory extension into the anterior and sometimes posterior pararenal space. The left pararenal space is most commonly involved. Occasionally, a pancreatic collection can extend into the perirenal space and even beneath the renal capsule potentially resulting in a Page kidney due to compressive forces on the renal parenchyma requiring percutaneous drainage. Other unusual complications include renal vascular abnormalities such as narrowing of the renal vein, renal vein thrombosis, perirenal varices and obstructive hydronephrosis due to extrinsic ureteral compression (56, 66, 100).

Splenic involvement by pancreatitis is not uncommon given the close relationship of

pancreatic tail and the splenic hilum. In addition to vascular complications ranging from splenic artery pseudoaneurysm to splenic vein occlusion, pancreatic collection may extend deep into the spleen. This can result in complications including intrasplenic collections, splenic infarction, splenic abscess, and intrasplenic hemorrhage. Intrasplenic collections render the organ vulnerable to rupture with even minor trauma (36, 67). Similar complications may occur in the liver.

Biliary complications during the course of acute pancreatitis include cholecystitis, biliary obstruction, or rarely gallbladder perforation (21, 25, 78).

Gastrointestinal complications in severe necrotizing pancreatitis are not uncommon because the extravasated pancreatic enzymes may directly extend into the mesenteries of bowel structures. Besides the risk of bacterial translocation, other catastrophic and life-threatening complications are bowel ischemia and perforation that demand emergent surgery (40, 63, 110). Another complication is abdominal compartment syndrome (ACS), which is increasingly recognized in necrotizing pancreatitis (see Chapter 32)(17, 51, 109). ACS is an important cause of multi-organ dysfunction associated with high mortality if left untreated. Although ACS is a clinical diagnosis, at times the diagnosis is suggested on CT in patients who exhibit the “round-belly sign”, defined as abdominal distension with an increased ratio of anteroposterior-to-transverse abdominal diameter (ratio >0.80) (1, 76). Particularly, the change in girth compared with prior CT scans may suggest ACS in the appropriate clinical setting. Finally, multiple pulmonary complications may be seen during the course of severe acute pancreatitis that includes the presence of pleural effusions, pulmonary infiltrates, pulmonary emboli and associated infarction, and, more rarely, pulmonary empyema and pneumothorax (28, 83).

7. Conclusion

Imaging is an indispensable tool, increasingly utilized in the care of patients with acute pancreatitis by providing critical information for clinicians, especially in those with severe disease. Multi-detector CT is the imaging modality of choice that allows for a quick and accurate overall assessment of acute pancreatitis and its complications with (E)US and MRI reserved for elucidating the etiology of the pancreatitis or as problem solving tools. Imaging-based predictive systems are useful for identifying groups of patients at risk for local complications or comparing outcomes of different groups in clinical research. However, for the individual patient, providing a radiographic grading score will not directly affect clinical management as opposed to some

specific cross-sectional imaging findings. Among these are the presence of extended necrosis (more than 30%), especially when the midgland is involved (associated with increased need for intervention), signs of infected necrosis (requiring empirical antibiotics or invasive intervention), massive hemorrhage or detection of an arterial pseudoaneurysm (indication for angiographic coiling or surgery), deep vein thrombosis or detection of pulmonary emboli (indication for anticoagulant therapy), acute cholecystitis (amenable for percutaneous drainage or cholecystectomy), bowel ischemia or perforation (indication for emergent surgery), and findings of ACS (requiring percutaneous drainage of ascites or surgery). Most of these complications are not included in any radiographic scoring system but will help guide individual patient management.

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