Acute pancreatitis

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Abstract

Acute pancreatitis (AP) is a significant cause of morbidity worldwide, with a steadily rising incidence, particularly in Western nations. This article provides a comprehensive overview of the epidemiology, aetiology, pathophysiology, and management of AP. The various aspects of the Revised Atlanta classification system are emphasized for its utility in defining AP severity, stratifying risk and guiding treatment. Both the role of imaging and the 'step-up' approach to guide procedural and endoscopic management are discussed, demonstrating the shift towards minimally invasive techniques. Early recognition of organ dysfunction and supportive care, including judicious fluid management, nutritional support, and appropriate use of antibiotics, are critical to improving outcomes. Specific considerations and recommendations for the management of local and systemic complications are outlined, with recommendations for early referral to specialized centres for optimal care.

Keywords Acute pancreatitis; endoscopic management; necrotizing pancreatitis; Revised Atlanta classification; step-up approach

Epidemiology

The incidence of acute pancreatitis (AP) in the UK is as high as 56 cases per 100,000 individuals annually, with rates approaching 100 cases per 100,000 per year in other European countries. Over the past 50 years in Europe there has been an annual increase in AP incidence by about 3.4%, with the most notable increase amongst women under 35 years of age. According to a study conducted using the UK biobank, the incidence has risen from 21.4 to 48.2 per 100,000 annually from 2001 to 2020.[1] The rise in incidence is best explained by a combination of more prevalent risk factors and enhanced detection methods in contemporary practice. Outside of western countries, such as Asia, reported incidences remain stable. Globally there are approximately 3 million cases of AP per year leading to an estimated 115,000 deaths.

The demographics of the AP cohort varies with the geographical area and the dominant aetiology of the region. In the UK, the average age of onset is 57 years, with men and women being affected equally, although gender ratios differ with the aetiology. Those suffering from socioeconomic deprivation have over a three times higher likelihood of developing AP, but no association between deprivation and either severity or mortality has been identified.[2]

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Aetiology

There are a multitude of causes of AP, but the dominant aetiology is largely region-dependent. In the UK and other areas of western Europe, gallstone disease is the leading cause in approximately 50% of cases, followed by alcohol in 25% and various other causes constituting the remaining 25%. In 10% of cases the cause is not identified and this is classed as idiopathic AP. Although the exact mechanism behind the progression of AP is often unknown, multiple mechanisms have been hypothesized for each underlying aetiology.

Obstructive causes

Gallstones are very common, affecting approximately 15% of people in western populations, owing to the prevalence of its risk factors in such regions (e.g. obesity, sedentary lifestyle, and dyslipidaemia). Gallstones typically cause AP in 4%–8% of these patients, but the risk is higher in those with smaller stones, a wider cystic duct, and a higher number of stones. The exact process by which gallstones trigger AP is not fully understood, but it is thought to arise from temporary obstruction of the pancreatic duct by gallstones in the common bile duct (known as choledocholithiasis) resulting in dilation upstream of the blockage, enzyme activation and local inflammation.

The pancreatic ducts, which are crucial for the transport of digestive enzymes, undergo a critical fusion during embryonic development, where the ventral and dorsal ducts combine to form the main pancreatic duct. However, in some individuals, this fusion fails, resulting in a congenital ductal anomaly known as pancreas divisum. This condition is present in approximately 5%–14% of the population, is characterized by the dorsal and ventral ducts remaining separate, which can potentially lead to obstructive pancreatitis. However, the high prevalence of pancreas divisum calls into question the causative role it may play in AP.

Another congenital anomaly linked with AP, albeit much rarer, is annular pancreas. This condition, found in approximately 5–15 cases per 100,000 individuals, occurs when a ring of pancreatic tissue encircles the duodenum and can cause a narrowing of the duodenum.

Furthermore, strictures or tumours (e.g. pancreatic ductal adenocarcinoma, intraductal papillary-mucinous neoplasms (IPMNs) and neuroendocrine tumours) within the pancreatic ducts are other significant causes of obstruction. It is estimated that pancreaticobiliary tumours are associated with pancreatitis in up to 14% of cases, therefore underlying lesions should remain within the differential in older patients with AP, and particularly those without a clear aetiology. In those that have cross-sectional imaging, up to 30% of patients with unexplained dilation of the main pancreatic duct may harbour a malignancy, underscoring the importance of thorough evaluation. In smaller pancreatic ducts, ductal papillary hyperplasia — a condition characterized by the proliferation of cells lining the ducts — can cause narrowing of the lumen, contributing to the obstruction and subsequent pancreatitis.

Toxins

Alcohol misuse is a major cause of AP and is often the leading cause in Eastern Europe. Up to 10% of chronic alcohol drinkers

will develop AP, with a higher risk amongst young males and those with higher daily intake. A monotonic and partial exponential dose–response relationship has been described whereby the risk curve is flat at lower levels of consumption and steeper with increasing levels of consumption. The pathogenesis of alcohol-induced AP is not fully defined but many potential triggers of the activated proteolytic enzymes have been proposed: a direct toxic-metabolic effect of ethanol on pancreatic acinar cells, small duct obstruction due to proteinaceous precipitates or large duct obstruction. Patients with a history of significant alcohol use often also have gallstones and this should also be investigated. Whilst smoking is not a direct cause of AP, it is a critical cofactor in alcohol-related pancreatitis with increasing risks linked to higher doses and longer durations of smoking. Smoking is also a recognized risk factor for recurrent AP and the development of local complications and progression towards chronic pancreatitis.

Post-ERCP

Endoscopic retrograde cholangiopancreatography (ERCP) is the leading cause of iatrogenic AP with an incidence of 3%–10% following ERCP. Post-ERCP AP is diagnosed when a patient experiences abdominal pain and hyperamylasaemia requiring hospitalization or extended hospital stay after the procedure. Hyperamylasaemia without abdominal pain occurs in up to 75% of patients following ERCP and should not be mistaken for post-ERCP AP. Risk factors for post-ERCP pancreatitis include female sex, younger age, normal bilirubin levels, therapeutic procedures like balloon sphincteroplasty, lower procedural volume by the operator, sphincter of Oddi dysfunction, pancreatic duct injection, repeated failed cannulation, and previous episodes of post-ERCP AP. Several strategies can help mitigate the risk of AP after ERCP, as outlined in the guidelines from the European Society of Gastrointestinal Endoscopy.[3] These measures include rectal administration of diclofenac or indomethacin, placement of a prophylactic pancreatic stent, sublingual glyceryl trinitrate, minimizing cannulation attempts and the use of pre-cut fistulotomy. In view of the risk of post-ERCP pancreatitis and other highly morbid complications, as well as the ease of access to non-invasive diagnostic techniques (e.g. CT and MRCP), early diagnostic ERCP is no longer recommended, and is reserved for cases of diagnostic uncertainty or septic patients with cholangitis.

Autoimmune

Autoimmune pancreatitis (AIP) is a unique form of pancreatitis that is often a key part of the broader spectrum of IgG4-related diseases, which can affect multiple organs including the retroperitoneum (leading to retroperitoneal fibrosis), salivary glands (resulting in sclerosing sialadenitis), bile ducts (sclerosing cholangitis), and even the aorta, breast, and prostate. Early and accurate recognition of AIP is crucial because it typically responds well to corticosteroid treatment. Distinguishing AIP from pancreatic cancer is essential due to the vastly different treatment approaches.

AIP is categorized into two types (Type 1 and Type 2) each with their own distinct clinical and histological features; although, the precise differences in pathogenesis are still not fully understood. Type 1 AIP, which is more prevalent in East Asia, tends to occur in older individuals, with the average age of onset around 62 years. Patients with Type 1 AIP often present with elevated serum IgG4 levels, and approximately 50% of these patients have extra-pancreatic manifestations. Histologically, Type 1 AIP is distinguished by a marked infiltration of IgG4-positive plasma cells and lymphoplasmacytic inflammation within the pancreas.

Type 2 AIP, on the other hand, is more commonly seen in the United States and Europe, where it accounts for approximately 45% of AIP cases. Unlike Type 1, Type 2 AIP typically affects a younger demographic, with an average onset age ranging from 40 to 48 years. This form of AIP is frequently associated with inflammatory bowel diseases such as Crohn's disease and ulcerative colitis, suggesting a different underlying immune mechanism. Histologically, Type 2 AIP is characterized by granulocytic epithelial lesions, a feature that helps to differentiate it from Type 1 AIP. Moreover, unlike Type 1, Type 2 AIP generally does not exhibit the extra-pancreatic involvement seen in IgG4-related disease, making its presentation more confined to the pancreas.

Metabolic causes

Hypertriglyceridaemia is recognized as a trigger for AP and accounts for approximately 5% of cases. There is no definitive threshold which triggers AP, however previous studies report a risk commensurate with increasing triglyceride level. A level greater than 2000 mg/dL confers a risk of 10%–20%, where baseline levels are less than 150 mg/dL. Compared to other aetiologies, patients with hypertriglyceride-induced AP tend to be younger, male, obese and have higher rates of diabetes.

Hypercalcaemia, regardless of its cause (e.g. hyperparathyroidism, multiple myeloma) can also lead to AP, but accounts for less than 1% of cases. The causative mechanism of AP is unknown, but it is hypothesized that hypercalcaemia leads to faster conversion from trypsinogen to trypsin leading to pancreatic damage or may lead to the deposition of calcium crystals in the pancreatic ducts. It is estimated that those with primary hyperparathyroidism and hypercalcaemia have a 10-fold higher risk of AP than the general population.

Other causes

Medications are a relatively rare but recognized cause of acute pancreatitis (AP), contributing to about 0.1%–2% of all cases. Despite the rarity of medication-induced AP, several commonly prescribed medications have been implicated as potential triggers such as corticosteroids, thiazide diuretics, tetracyclines, furosemide, azathioprine and mesalazine. The mechanism by which these drugs induce pancreatitis is not fully understood and may vary depending on the medication, ranging from hypersensitivity reactions to direct toxic effects on the pancreatic cells. A comprehensive investigation is essential to rule out other more common causes of AP, such as gallstones, chronic alcohol use, hypertriglyceridaemia, or metabolic disorders. Following this, when drug-induced AP is suspected, the immediate step is usually the withdrawal of the offending medication which may lead to the resolution of symptoms. The most definitive evidence for drug-induced AP is the recurrence of pancreatitis symptoms upon re-exposure to the suspected drug, a phenomenon known as a positive rechallenge. Rechallenging with a drug is generally

avoided due to the risk of severe recurrence, unless absolutely necessary for diagnostic clarification.

Trauma is another significant cause of AP, especially in cases where the pancreas is compressed or injured, often against the vertebral column. Such injuries can occur in road traffic accidents, falls, or sports injuries. Clinicians need to be alert to the possibility of associated injuries to nearby organs, such as the liver, spleen, or intestines, which may complicate the clinical picture. While most cases of traumatic pancreatitis can be managed conservatively, severe injuries involving the pancreatic duct might require more interventions, including drainage procedures, embolization, endoscopic stenting, or surgical repair.

The notion that genetic factors play a role in the development of AP has been considered for many years, and recent research has begun to clarify the involvement of specific genetic mutations. One such mutation occurs in the PRSS1 gene, which encodes cationic trypsinogen, leading to increased activation of trypsin within the pancreas and resulting in autodigestion and inflammation. The CFTR gene, known for its association with cystic fibrosis, can also contribute to the development of pancreatitis, particularly in individuals who are compound heterozygotes or possess milder mutations that do not cause full-blown cystic fibrosis. Furthermore, mutations in the SPINK1 gene, responsible for producing a trypsin inhibitor, can impair the pancreas's ability to prevent premature enzyme activation, thus leading to recurrent episodes of pancreatitis. It is crucial for clinicians to consider a potential genetic predisposition to AP, especially in patients with a family history, recurrent idiopathic pancreatitis, or unexplained cases in children. In these instances, genetic counselling and testing may be advantageous, not only for the patient but also for at-risk family members. Gaining insight into the genetic underpinnings of AP in these patients can aid in guiding long-term management and may influence the choice of preventive measures or early interventions.

Pathophysiology

The exact pathophysiology of AP is not completely understood, and multiple theories have been proposed which largely depend on the aetiology. Nevertheless, a number of key concepts are widely accepted. The pathogenesis of acute pancreatitis begins at the cellular level within the acinar cells, which are responsible for the production of digestive enzymes. Under normal conditions, these enzymes are synthesized as inactive precursors (zymogens) and stored in secretory vesicles called zymogen granules. They are normally activated by a cascade of proteolysis upon reaching the duodenum, whereas in AP, a disruption occurs in this process, leading to the premature activation of these zymogens within the pancreas. One of the key enzymes involved is trypsinogen, which is converted into its active form, trypsin. This activates other digestive enzymes, such as chymotrypsin, elastase, and phospholipase A2, within the pancreatic tissue resulting in the digestion of the pancreatic parenchyma, leading to cell injury, inflammation, and in severe cases, necrosis.

The exact mechanisms behind the premature activation of trypsinogen remain unclear, but several hypotheses have been proposed. One theory suggests that an overload of calcium within the acinar cells leads to the activation of trypsinogen. Another theory involves the disruption of cellular organelles, such as the lysosomes and zymogen granules, which normally keep the enzymes separated. When these organelles fuse or are damaged, it can result in the enzymes being prematurely activated within the cell.

The autodigestion of pancreatic tissue by activated enzymes triggers a robust inflammatory response mediated by the release of cytokines and chemokines from the damaged acinar cells, which recruit immune cells such as neutrophils, macrophages, and lymphocytes to the site of injury. These immune cells release additional inflammatory mediators, including interleukins (e.g., IL-1, IL-6), tumour necrosis factor-alpha (TNF-α), and reactive oxygen species (ROS), which exacerbate tissue damage and inflammation. In some cases, the inflammation is localized to the pancreas; however, in other cases, the release of inflammatory mediators can lead to the systemic inflammatory response syndrome (SIRS) and cause multi-organ dysfunction.

Another critical aspect of the pathophysiology of AP is disruption of the pancreatic microcirculation. The inflammation and enzymatic damage leads to increased vascular permeability, causing fluid leakage into the interstitial space resulting in pancreatic oedema. At the same time, the release of vasoactive substances, such as endothelin and nitric oxide, can cause vasoconstriction and impaired blood flow within the pancreas. The combination of oedema and reduced blood flow can lead to pancreatic ischaemia, which exacerbates tissue injury and inflammation. Ischaemia-reperfusion injury, a phenomenon where the return of blood supply to the ischaemic tissue causes further damage due to the production of ROS, can also occur in acute pancreatitis, contributing to the severity of the disease.

In severe cases of AP, the ongoing inflammation and microcirculatory disruption can lead to extensive pancreatic necrosis. Necrotic tissue is a breeding ground for infection, and secondary bacterial infections are common in necrotizing pancreatitis. Infected pancreatic necrosis is associated with a high mortality rate and often requires endoscopic or surgical intervention for management.

The later stages of AP often involve the emergence of various complications. Following the systemic inflammation response, increased intestinal permeability permits the translocation of gut bacteria into the pancreatic tissues leading to secondary infections or necrosis. Furthermore, peripancreatic collections, which initially may consist of fluid and debris, can begin to organize and over time may develop a fibrous wall, transforming into what are known as walled-off necrosis (WON) or pseudocysts. These lesions can vary in size and complexity and may remain sterile or become infected.

Diagnosis

To diagnose acute pancreatitis (AP), two out of the following three criteria must be met: (i) characteristic abdominal pain; (ii) elevated serum amylase/lipase; (iii) imaging results characteristic of AP.

Symptoms

The characteristic symptom of AP is sudden onset, persistent and intense epigastric pain that often radiates to the back. It may also radiate across the upper abdomen to the right and left upper quadrants and may be associated with other symptoms such as

fevers, nausea, anorexia, vomiting or fatigue. The history should enquire about details which could point towards a possible aetiology: a history of gallstones, alcohol intake and other relevant comorbidities.

Clinical examination may elicit a distended abdomen with severe tenderness across the upper abdomen and patients may appear clammy and sweaty. Rarely Grey Turner sign (flank bruising) or Cullen's sign (periumbilical bruising) may be seen in patients with haemorrhagic pancreatitis. Patients may also have signs and symptoms (e.g. Murphy's positive, jaundice) which could indicate additional relevant biliary pathology (e.g. choledocholithiasis) requiring additional investigation and treatment.

The clinical status of patients varies significantly with the severity of the inflammation, duration of symptoms and their functional reserve. In severe cases, patients can present with signs of hypovolaemic shock, including hypotension, tachycardia and pyrexia, even in the absence of infection. They typically have poor urine output due to fluid sequestration or may have temporary hyperglycaemia because of impaired islet cell function. Differential surgical diagnoses include biliary colic, cholecystitis, peptic ulcer disease, bowel obstruction, bowel ischaemia/infarction, and ruptured aortic aneurysm.

Biochemistry

The primary diagnostic indicator is an elevated serum pancreatic enzyme level (either amylase or lipase), at least three times the upper limit of normal. Serum lipase has a higher sensitivity than amylase (95% vs. 64%−80%), yet it is only available to a minority of centres in the UK. Furthermore, lipase has a longer diagnostic window and can remain elevated for up to 2 weeks compared to approximately 5 days with amylase. The lower sensitivity of hyperamylasaemia can be problematic in patients with a delayed presentation, hypertriglyceridaemia and chronic pancreatitis. The baseline level of amylase is dependent on ethnicity and is higher in both Asian and Native American populations compared to Caucasians. It is also more likely to have a lower sensitivity (∼ 50%) in elderly populations (≥65 years).

The level of detected pancreatic enzymes is not proportionate with the severity of pancreatitis or the development of necrosis and should not be used as a prognostic measure. Furthermore, the trends of pancreatic enzymes offer limited benefit in diagnosis or treatment decisions. Although prolonged hyperamylasaemia may be positively associated with pancreatic pseudocysts, abscesses and necrosis and may be associated with alcohol use, this is of limited use in clinical practice.

Other biochemical markers help indicate AP. Elevated white blood cell (WBC) count and C-reactive protein (CRP) are often elevated and both are positively associated with the severity of pancreatitis. Raised bilirubin and transaminases may indicate a biliary aetiology. A low serum albumin is independently associated with increasing severity of pancreatitis, the development of complication (e.g. pleural effusion and ascites) and mortality.

Biochemical tests also play a role in the identification of the underlying aetiology. Biochemical testing can investigate for serum hypertriglyceridaemia, hypercalcaemia and IgG4, although this is generally performed after excluding more common aetiologies, specifically gallstones and alcohol.

Finally, high blood urea nitrogen (BUN) levels at admission have prognostic significance and reflect hypovolaemia as well as hypercatabolism in severe acute pancreatitis. A BUN-based assessment algorithm identified patients at increased risk for mortality during the initial 24 hours of hospitalization.[4]

Imaging

Multiple imaging modalities play a role in the diagnosis of AP and its complications. Upon admission with acute abdominal pain, an erect chest X-ray can help rule out pneumoperitoneum or lung pathology. Abdominal X-rays play a limited role in AP but again can help exclude alternative pathology (e.g. bowel obstruction) which may present with similar pain characteristics and examination findings. It may also show signs of an associated ileus or a sentinel loop of dilated bowel in the epigastrium indicating the diagnosis.

The role of transabdominal ultrasound (USS) is limited in the diagnosis of AP itself. Views of the pancreas are often limited by overlying bowel gas and only occasionally parenchymal changes are visualized. USS is typically reserved and performed routinely for the investigation of gallstones, cholecystitis or biliary dilatation. Focused USS does have a role in the evaluation and follow-up of AP complications such as pseudocyst or venous thrombosis.

In patients with acute abdominal pain and biochemical evidence of AP, computed tomography (CT) is often not immediately required. However, in cases of diagnostic uncertainty, particularly when pancreatic enzymes do not meet the diagnostic threshold, CT plays a vital role. In the critically unwell patient, CT can evaluate the severity of pancreatitis, identify complications as well as areas of necrosis.

Magnetic resonance cholangiopancreatography (MRCP) can be performed to investigate for ductal stones with superior diagnostic accuracy than liver function tests and USS. Although the sensitivity and specificity of MRCP range from 0.80 to 0.93 and 0.87 to 0.96, respectively, these are in fact inferior to endoscopic ultrasound (EUS). As such the International Association of Pancreatology (IAP)/American Pancreatic Association (APA) guidelines recommend the use of endoscopic ultrasound (USS) in patients with idiopathic AP to investigate for microlithiasis.[5]

The EUS procedure not only provides high-resolution images but also allows for fine-needle aspiration (FNA) of any suspicious lesions, which can be used to obtain tissue samples for histopathological analysis. This is especially important in distinguishing between benign and malignant masses, guiding the appropriate course of treatment. Early detection of a pancreatic or biliary tumour in patients presenting with AP can significantly impact their prognosis, as prompt intervention may prevent further episodes of pancreatitis and potentially improve survival rates in cases of malignancy. Moreover, the use of EUS in these patients can help identify other potential causes of ductal obstruction, such as benign strictures or cystic lesions (e.g. intraductal papillary mucinous neoplasms), which might also guide further surveillance or treatment decisions.

Severity scoring

Extensive literature exists on the prediction of severity in AP and subsequently various scoring systems have derived. The Ranson and Glasgow-Imrie scoring systems use clinical (e.g. age, arterial

oxygen) and laboratory parameters (e.g. white blood cell count, blood glucose) both within the first 48 hours of admission to assess the severity of AP. In both scoring systems a score of 3 or more predicts severe pancreatitis. They have both been criticized because it takes 48 hours to calculate the scores, they have low predictive value and may have a limited role in clinical practice as decisions derived from the scoring systems may result in treatment delays. The Ranson criteria is particularly cumbersome, requiring 11 parameters yet has consistently demonstrated inferior predictive value in comparison to alternative scoring systems.

The APACHE II score, commonly used in intensive care units, associates a score above 8 with poorer outcomes and is often more accurate than the Ranson and Glasgow-Imrie scores. Similar to other severity scores, its diagnostic ability to predict organ dysfunction, necrosis and mortality is higher at 48 hours compared to admission, which limits its utility.

The Marshall scoring system is a useful tool for evaluating organ dysfunction by assessing the function of three critical organ systems: respiratory, renal, and cardiovascular, using three parameters, PaO2/FiO2 ratio, serum creatinine levels, and systolic blood pressure. By offering a simple and standardized approach for early detection and continuous monitoring of organ failure, the Marshall scoring criteria guides clinicians in making informed decisions without the requirement for complex laboratory assays.

Simpler single laboratory metrics, such as serum C-reactive protein, urinary trypsinogen activation peptide, or procalcitonin levels also serve as severity indicators and offer clinical value. Clinically, CRP is frequently measured; both levels exceeding 150 mg/L at 48 hours and interval increase by >90 mg/L from admission correlate with severe AP.

Many of the clinical scores are of limited practical value due to their suboptimal diagnostic accuracy and the difficulty implementing the severity scores sufficiently early during admission. A recent comprehensive review of scoring systems in predicting severity in acute pancreatitis has shown that the most commonly used scoring systems to predict severe AP perform poorly and do not aid in decision making.[6] As such their role primarily serves as audit and research purposes. Instead, the advised clinical strategy focuses on the prompt recognition of organ dysfunction to ensure timely organ support instead of relying on severity scoring methods. Dynamic physiological scoring systems like the National Early Warning Score (NEWS) are valuable tools for identifying clinical deterioration; however similar to severity scores, they should be treated as one component of the clinical assessment and should be used in conjunction with all aspects of the clinical picture (e.g. clinical, biochemical, microbiological, and radiological).

The Revised Atlanta classification

The Atlanta classification plays a pivotal role in the management and understanding of acute pancreatitis by providing a standardized framework for classifying the severity of the disease and its associated complications. First introduced in 1992 and revised in 2012, the Atlanta classification divides AP into two distinct phases: the early phase, which occurs within the first week, and the late phase, which extends beyond the initial week. The classification categorizes the disease into three severity levels: mild, moderate, and severe.

Mild acute pancreatitis occurs in approximately 80% of patients and is characterized by the absence of organ failure and local or systemic complications, typically leading to a quick recovery. Moderate acute pancreatitis involves transient organ failure (lasting less than 48 hours) and/or the presence of local complications such as peripancreatic fluid collections or pancreatic necrosis. Severe acute pancreatitis is defined by persistent organ failure (lasting more than 48 hours), which can affect one or multiple organ systems, often leading to a more complicated clinical course and higher mortality rates. Approximately 15%–20% of patients progress to either moderate or severe AP and mortality ranges from 21% to 40% in severe AP, with half of deaths occurring within the first week. Patients with severe AP who survive the early phase are at heightened risk of developing secondary infections in the pancreatic necrosis which is strongly associated with mortality.

AP is classified into two main types: interstitial oedematous pancreatitis (IOP) and necrotizing pancreatitis (NP). Most AP patients experience IOP (80%–90% of cases), characterized by the diffuse and uniform enlargement of the pancreas due to inflammation. IOP occurs without necrosis and usually resolves quickly. About 5%–10% of AP patients develop necrosis, affecting both the pancreatic tissue and the surrounding peripancreatic area. Initial CT often underestimates the extent of necrosis, which becomes more apparent by non-enhancing areas of the pancreatic tissue after several days. Necrotizing pancreatitis should be classified as infected or sterile, given the association between infection and higher mortality, which approaches 30%. The presence of infection may be determined from positive cultures, the presence of gas on CT or from EUS fine-needle aspirate.

The Revised Atlanta classification also introduces terminology for local complications (e.g. acute peripancreatic fluid collections, pseudocysts, acute necrotic collections, and walled-off necrosis). These definitions are crucial for guiding clinical decision making, particularly regarding the timing and type of interventions required, such as drainage or surgery.

By offering a clear and consistent approach to categorizing the severity and complications of acute pancreatitis, the Revised Atlanta classification helps clinicians tailor treatment strategies, improve communication among healthcare providers, and standardize research studies. It has become an essential tool in both clinical practice and research, facilitating better patient outcomes through a structured approach to managing this complex and potentially life-threatening condition.

Complications

Local

Acute peripancreatic fluid collections (APFC) typically develop in the early stages of acute pancreatitis, especially in cases of IOP. They consist of homogeneous fluid without solid debris and are confined to the retroperitoneal space. These collections generally lack a defined capsule and are often sterile. Pancreatic pseudocyst may form from the maturation of APFCs and are defined from 4 weeks following the onset of pancreatitis. They have a

well-defined wall of fibrous or granulation tissue and contain no necrotic material. While some pseudocysts resolve on their own, others may require drainage, especially if they become symptomatic or infected.

Acute necrotic collections (ANC) develop in cases of necrotizing pancreatitis and consist of a mixture of fluid and necrotic tissue. These collections, which lack a defined wall in the early stages, can be either sterile or infected. Infection occurs in approximately 20% of ANC, which significantly worsens the prognosis and typically necessitates intervention, such as drainage or debridement. Walled-off necrosis (WON) may develop from ANC, typically developing more than four weeks after the initial episode, and is characterized by a well-defined inflammatory capsule.

Pancreatic fistulas are an abnormal entity defined by abnormal connections between the pancreatic ductal epithelium and another epithelial surface. Pancreatic fistulas may be associated with acid–base disturbance (e.g. metabolic acidosis) or in cases of spontaneous rupture of a pseudocyst into an adjacent viscus, bleeding or sepsis. On CT, fistulas often present with a gas/fluid level, unlike the gas pockets commonly seen in infected collections. Pancreatic fistulas are mostly managed conservatively with a somatostatin analogue (e.g. octreotide) and ERCP stenting. Rarely a distal pancreatectomy is required to excise fistulas arising from the tail or a decompression of the fistula tract by performing a pancreatico-jejunostomy. Inflammation and oedema from AP, particularly of the pancreatic head can also cause compression of the bile ducts or biliary strictures, leading to obstructive jaundice.

Vascular complications can occur as a result of pancreatitis-related vascular erosion. Psuedoaneurysms may form and rupture may lead to life-threatening haemorrhage. Prompt diagnosis and treatment, typically with angiographic embolization or surgery is required to control haemorrhage. The risk of splanchnic vein thrombosis (SVT) increases with the severity of AP and the degree of pancreatic necrosis. Up to 90% of SVT will be associated with underlying necrotizing pancreatitis, although the prevalence of SVT is likely underreported. A selective anticoagulation policy has shown high recanalization rates with little increase in haemorrhagic complications and is the recommended current practice.[7]

Systemic

SIRS is a critical systemic complication characterized by widespread inflammation that can cause multi-organ dysfunction syndrome (MODS). Organ failure, including respiratory, renal, and cardiovascular failure, is a severe complication of AP and a key determinant of prognosis. Respiratory failure may manifest as acute respiratory distress syndrome (ARDS), requiring mechanical ventilation. Renal failure often necessitates renal replacement therapy, while cardiovascular failure may lead to shock, requiring vasopressors and intensive care.

Disseminated intravascular coagulation (DIC) is a severe coagulopathy that can occur in the context of severe acute pancreatitis. It is characterized by widespread activation of the clotting cascade, leading to both thrombotic complications and bleeding due to the consumption of clotting factors. DIC requires prompt recognition and management, often involving supportive care and addressing the underlying cause.

Patients with AP are at increased risk for venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE). The hypercoagulable state induced by systemic inflammation, along with immobilization during severe illness, contributes to this risk.

Due to the destruction of insulin-producing beta cells in the pancreas, acute pancreatitis can lead to hyperglycaemia. In some cases, this may progress to permanent diabetes mellitus, especially in patients with recurrent or severe pancreatitis.

Early management of acute pancreatitis

AP is typically a self-limiting condition and for those with mild AP, monitoring and supportive care are typically sufficient. However, when managing a patient with acute pancreatitis (AP), a comprehensive and methodical approach is essential regardless of severity. Patient management centres around the early recognition of organ dysfunction, appropriate treatment and prompt escalation to intensive care units for patients with severe disease.

AP often leads to hypovolaemia due to a multitude of factors such as vomiting, reduced oral intake, ileus, and third-space fluid sequestration. Accurate assessment of cardiovascular status using clinical parameters (heart rate, blood pressure, urine output, central venous pressure), biochemical markers (serum urea and creatinine, blood pH, base excess, lactate level, and mixed venous oxygen saturation) and electrocardiograms is essential for identifying organ dysfunction and determining fluid status and requirements. Critically unwell patients frequently require central venous access, blood pressure monitoring, aggressive fluid administration and urinary catheterization. Although Ringer's lactate and normal saline are both commonly administrated, Ringer's lactate is associated with lower metabolic acidosis, has an anti-inflammatory effect and may mitigate against a prolonged SIRS process.[8] Whilst aggressive resuscitation is of utmost importance, excessive hydration can result in worsening sepsis, increased reliance on mechanical ventilation, and elevated mortality.[9]The WATERFALL trial demonstrated that a goal-directed strategy is optimal, which couples judicious fluid resuscitation with active assessment of fluid status.[10]

Overall, approximately 20% of patients will suffer from moderate or severe pancreatitis and need additional support. Patients with transient organ dysfunction should be admitted to a high dependency unit, and early consultation with the intensive care team is advised. Although supplementary oxygen is adequate in most patients with low oxygen saturations, positive pressure ventilation or high-flow oxygen therapy may be required in those with hypoxaemia.

Analgesia

Abdominal pain is a significant component of the patient experience in AP, primarily transmitted via the splanchnic nerves. Effective and timely pain management is essential to alleviate patient anxiety, reduce the risk of complications, and improve quality of life. Currently, the primary treatments for pain relief in acute pancreatitis are paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs) and opioids. Previous preclinical studies have reported increased severity of experimental AP in mice treated with morphine, fentanyl, or buprenorphine. Although in

human studies there were previous concerns that opioids might cause sphincter of Oddi spasm and worsen AP, current evidence does not support these concerns. Meta-analyses have failed to consistently show that opiates may worsen disease severity and offer conflicting evidence regarding the superiority of one treatment over the other. The decision-making process is further complicated by the absence of consistent guidelines for pain management, and variations between centres. A recent multicentre study emphasized the widespread use of opioids for managing pain in AP, which has raised concerns about the potential for over-reliance on opioids. Given the lack of definitive evidence for the optimal approach to pain management in acute pancreatitis, a practical approach based on the WHO pain ladder is recommended, where treatment is tailored to the intensity of pain and patient-reported pain levels.

Nutrition

Maintaining nutritional integrity is crucial in the management of patients with AP to mitigate against the catabolic state induced by severe pancreatitis. Patients' nutritional intake should be assessed within the first few days of admission and those with significant or persistent vomiting should be considered for alternative routes of nutrition. Both the NICE and the American Gastroenterological Association (AGA) advocate for initiating oral feeding early provided patients can tolerate it. In particular, NICE recommends that for those with severe or moderately severe AP, supplementary enteral nutrition should be started within 72 hours of symptom onset.

It is well accepted that total enteric nutrition is the optimal approach to avoid the complications of total parenteral nutrition. Meta-analyses indicate that enteral nutrition reduces rates of systemic infections (e.g. line sepsis), multi-organ failure and mortality compared to total parenteral nutrition. Enteral nutrition supports the maintenance of the gut mucosal barrier, which reduces bacterial translocation and lowers the risk of subsequent infectious complications. Nasogastric feeding is easy to place and is equivalent to nasojejunal feeding in terms of infective complications or pain. In cases of gastroparesis or gastric outlet obstruction, and where long-term enteric feeding is required (e.g. >30 days), a nasojejunal tube may be required. Both nasogastric and nasojejunal tubes may be positioned through a percutaneous-endoscopic approach, particularly in those who do not tolerate nasal insertion. Total parental nutrition is reserved for cases where enteric feeding is unsuccessful (e.g. luminal obstruction), poor tolerance to enteral feeding or when enteral feeding is contraindicated (e.g. fistulation or bowel obstruction).

Antibiotics

Patients with AP frequently have fevers as a result of SIRS, but this should not be mistaken for sepsis without supporting evidence. A meta-analysis investigating 10 recent randomized controlled trials on the use of prophylactic antibiotics did not identify any reduction in infected pancreatic necrosis, organ dysfunction, interventional rate or mortality.[11] Inappropriate antibiotic administration may instead lead to the development of resistant superinfections (e.g. *Candida*), which could hasten clinical deterioration if difficult to treat. Procalcitonin measurement may have a potential role in guiding clinicians during this assessment. A recent randomized trial demonstrated the beneficial role of a procalcitonin-guided algorithm for managing antibiotic therapy in patients with AP.[12] The study found that this approach reduced antibiotic use without affecting clinical outcomes, suggesting that procalcitonin effectively limited antibiotic administration to the most relevant cases. This has value in keeping the overuse of antibiotics low, avoids bacterial resistance development and ensures use of the right type of antibiotic regimens tailored to the need of the condition.

However, certain patient subgroups do benefit from antibiotic therapy. These include individuals with concurrent cholangitis, extra-pancreatic infections, and infected pancreatic necrosis. Culture-proven infected pancreatic necrosis is present in up to 50% of cases of infected necrosis and the most common organisms are *Escherichia coli* and *Klebsiella* pneumonia.[13] Signs of infected pancreatic necrosis may include clinical deterioration, blood culture, suggestion of a gas-forming organism on CT, or positive evidence from Gram stain and cultures from percutaneous aspirates. In such cases, antibiotics should be initiated empirically and later adjusted based on culture results when they become available.

The role of ERCP

Several clinical trials have investigated the effectiveness of endoscopic retrograde cholangiopancreatography (ERCP) in patients with AP caused by gallstones. These studies suggest that performing ERCP should only be considered in cases of cholangitis secondary to choledocholithiasis. In reality, most patients with associated choledocholithiasis do not demonstrate clinical signs of cholangitis and the need to perform an ERCP can be postponed or avoided in cases of spontaneous passage. Sequential liver function tests, magnetic resonance cholangiopancreatography (MRCP) or endoscopic ultrasound (EUS) can be used to monitor for spontaneous passage of the stone or development of obstructive jaundice.

Laparoscopic cholecystectomy

The PONCHO trial investigated the role of same-admission versus interval laparoscopic cholecystectomy for patients with mild gallstone pancreatitis and found that early cholecystectomy (within 2 weeks) reduces the rate of recurrent gallstone-related complications.[14] Furthermore, laparoscopic cholecystectomy can be performed safely in this group with an acceptable rate of cholecystectomy-related complications and rate of conversion to open. For moderate or severe pancreatitis, cholecystectomy should be postponed until after the resolution of post-pancreatitis complications, as performing the surgery too early has been associated with increased risk of morbidity and mortality as well as higher operative difficulty. In patients who are not candidates for cholecystectomy, prophylactic endoscopic biliary sphincterotomy via ERCP may reduce the risk of recurrent gallstone pancreatitis.

Procedural and surgical intervention for local complications

Most APFCs and ANCs tend to resolve spontaneously and do not require intervention, particularly in the absence of infection.

Spontaneous drainage of a post-acute collection into the gastrointestinal tract, or into the peritoneal cavity causing ascites are both likely to lead to clinical improvement without the need for intervention. However, when ANCs become infected, the patient may exhibit signs of sepsis and intervention should be considered. Furthermore, intervention is indicated for patients with pseudocyst or WONs which exhibit signs of infection or those causing mass-effect compression (e.g. gastric outlet obstruction), intractable pain, or failure to thrive. Patients with sterile necrosis should be managed conservatively.

Endoscopic approach

There are a number of indications for EUS in the management of pancreatitis and its complications. Currently EUS transmural drainage is first line for drainage of pseudocysts and WON, when indicated. EUS permits direct visualization, identification of blood vessels, identification of necrosis, and aspiration of PFC fluid to confirm placement within the collection. Double pigtail plastic stents (DPPSs) have traditionally been the primary treatment for EUS drainage. These stents are affordable, safe, widely available, and easy to remove, however often require multiple stent placements and dilation procedures to achieve adequate drainage. Achieving clinical success in WON is lower (30%−52%) compared to pseudocyst (85%−90%). More recently, lumen-apposing metal stents (LAMS) are commonly used which achieve more adequate drainage with their wider lumen. Risks of LAMS include perforation, bleeding and migration, although the biflanged shape helps reduce this risk. Metal stents may also become embedded as surrounding tissue grows over them, so removal within 3−4 weeks should be considered. If the patient does not improve after the initial drainage, endoscopic necrosectomy may be required. Multiple sessions may be required whereby the contents are suctioned and irrigated until clinical resolution. Additionally, inserting a nasocystic catheter may be beneficial if there is significant necrotic debris.

Persistent pancreatic fistulas may arise after procedures such as percutaneous drainage or necrosectomy. Although these fistulas typically resolve spontaneously after removing percutaneous drains, those that persist can be managed by placing a pancreatic ductal stent at ERCP. Persistent fistulas may also occur as a result of significant parenchymal damage or a disconnected pancreatic tail, where the main pancreatic duct is no longer intact. In such cases, EUS may be performed to drain the collection into the stomach.

Surgical intervention

Videoscopic retroperitoneal debridement (VARD) is a minimally invasive surgical technique used to treat infected pancreatic necrosis, whereby the surgeon accesses the necrotic area through the retroperitoneal space to debride and remove the necrosis under vision. An incision is made adjacent to an existing percutaneous drain and a laparoscope is used to guide the debridement. An alternative to VARD is minimally invasive retroperitoneal (MIRP) necrosectomy. In this technique, the percutaneous drain is replaced with a guidewire, followed by the insertion of Amplatz dilators to create a tract leading to the necrotic collection. A nephroscope is then introduced to flush the area and remove loose necrotic tissue. Once the procedure is complete, a large drain is placed to allow for continuous irrigation, facilitating additional necrosectomies if needed. Open surgical necrosectomy may be necessary if other methods fail, though it is typically avoided due to its higher associated mortality.

Step-up approach

In cases of infected necrosis, intervention via a step-up approach has been suggested, whereby management escalates from endoscopic (first line) to percutaneous and then to minimally invasive and open surgical options.[15] In 2010, the PANTER trial introduced the concept of a 'step-up approach' whereby percutaneous drainage followed by VARD was superior to open necrosectomy in terms of major complications and mortality.[15] Interestingly, 35% of patients were actually treated with percutaneous treatment only and did not require treatment escalation.

A more recent trial has since compared the endoscopic step-up approach with a percutaneous-surgical step-up approach. Although there were no differences in mortality, the former had a significantly lower rate of pancreatic fistulae and a shorter length of stay.[16] According to NICE guidelines, an endoscopic approach should be first line if anatomically possible; otherwise, percutaneous methods are suggested. Other considerations regarding choice of intervention include feasibility for endoscopic drainage, the patient's overall fitness, the amount of debris within the cyst, and available local expertise. Medially located retrogastric collections are more suitable for EUS drainage, whereas laterally positioned collections that are further from the stomach are better managed with percutaneous drainage. EUS offers the benefit of enabling drainage procedures directly in the intensive care unit (ICU), eliminating the need to transfer the patient to radiology. In some situations, a combination of percutaneous and endoscopic interventions may be necessary.

The timing of intervention in cases of pancreatic necrosis has been a topic of debate. Performing interventions within the first two weeks carries a high risk of morbidity and mortality and should be avoided. It is recommended to allow pancreatic fluid and necrotic collections to mature for at least 4 weeks before considering intervention, to from a discrete collection whilst monitoring for spontaneous regression. The POINTER trial compared early drainage of infected necrosis within 24 hours with drainage once WON had formed. The trial found no difference in complications between the two groups up to 6-months follow-up but found that less interventions were required in the delayed group.[17]

The recent DESTIN trial has shown that in stable patients with infected necrotizing pancreatitis and fully encapsulated collections, an approach incorporating upfront necrosectomy at the index intervention rather than as a step-up measure could safely reduce the number of reinterventions required to achieve treatment success.[18]

Angiography and embolization

In severe cases of acute pancreatitis (AP), patients may experience significant bleeding events, including haematemesis, rectal bleeding, internal haemorrhage, or bleeding from abdominal or retroperitoneal drains. Haemorrhage can also be a complication of early or overly aggressive necrosectomy. Often, a smaller 'herald bleed' occurs before a more severe haemorrhage, with an overall

mortality rate exceeding 30%. Arterial haemorrhage tends to present either early in the course of necrotizing pancreatitis or after ten weeks, further complicating the condition.

Bleeding may occur from pseudoaneurysms of the left gastric, splenic, gastroduodenal, or superior mesenteric arteries. It is critical to maintain a high level of suspicion for effective and timely intervention. Patients should be rapidly stabilized with circulatory support, and an emergency CT angiogram should be promptly performed. Upper gastrointestinal endoscopy is generally non-diagnostic in these cases and may delay necessary treatment. The best outcomes are typically achieved through formal angiography and embolization.

Referral for specialist treatment

According to NICE guidelines, patients who develop necrotic, haemorrhagic, or systemic complications from acute pancreatitis should be referred to a specialist pancreatic center. While most patients with AP can be treated at their local hospital, those requiring specialized radiological, endoscopic, or surgical interventions may need to be transferred to a specialized facility. Published evidence suggests this approach has improved outcomes and can reduce mortality in AP.[19]

Conclusion

In conclusion, the incidence of AP is rising globally, with regional variations influenced by dominant aetiologies and risk factors. Advancements in imaging, early recognition of organ failure, and the shift towards minimally invasive interventions have improved management strategies. The step-up approach to treating necrotizing pancreatitis has reduced the need for more invasive procedures, offering better outcomes. A significant focus must remain on early diagnosis, personalized treatment based on severity, and appropriate nutritional and supportive care to mitigate complications. Effective management of AP will continue to rely on multidisciplinary care, including timely referrals to intensive care units and specialized centers. ◆

REFERENCES

1 Spagnolo DM, Greer PJ, Ohlsen CS, et al. Acute and chronic pancreatitis disease prevalence, classification, and comorbidities: a cohort study of the UK BioBank. *Clin Transl Gastroenterol* 2022; **13**. https://doi.org/10.14309/ctg.0000000000000455.

2 Lim WB, Robertson FP, Nayar MK, et al. Social deprivation does not impact on acute pancreatitis severity and mortality: a single-centre study. *BMJ Open Gastroenterol* 2023; **10**. https://doi.org/10.1136/bmjgast-2022-001035.

3 Dumonceau JM, Kapral C, Aabakken L, et al. ERCP-related adverse events: European society of gastrointestinal endoscopy (ESGE) guideline. *Endoscopy* 2020; **52:** 127–49. https://doi.org/10.1055/a-1075-4080.

4 Wu BU, Bakker OJ, Papachristou GI, et al. Blood urea nitrogen in the early assessment of acute pancreatitis: an international validation study. *Arch Intern Med* 2011; **171:** 669–76. https://doi.org/10.1001/archinternmed.2011.126.

5 IAP/APA evidence-based guidelines for the management of acute pancreatitis. *Pancreatology* 2013; **13**(4 suppl. 2)**:** e1–15. https://doi.org/10.1016/j.pan.2013.07.063.

6 Capurso G, Ponz de Leon Pisani R, Lauri G, et al. Clinical usefulness of scoring systems to predict severe acute pancreatitis: a systematic review and meta-analysis with pre and post-test probability assessment. *United Eur Gastroenterol J* 2023; **11:** 825–36. https://doi.org/10.1002/ueg2.12464.

7 Thejasvin K, Chan SJ, Varghese C, et al. A selective anticoagulation policy for splanchnic vein thrombosis in acute pancreatitis is associated with favourable outcomes: experience from a UK tertiary referral centre. *HPB* 2022; **24:** 1937–43. https://doi.org/10.1016/j.hpb.2022.06.003.

8 Iqbal U, Anwar H, Aftab M, et al. Ringer's lactate vs normal saline in acute pancreatitis: a systematic review and meta-analysis. *Am J Gastroenterol* 2018; **113**(supplement). https://doi.org/10.14309/00000434-201810001-00002.

9 Mao EQ, Fei J, Peng YB, et al. Rapid hemodilution is associated with increased sepsis and mortality among patients with severe acute pancreatitis. *Chin Med J* 2010; **123:** 1639–44. https://doi.org/10.3760/cma.j.issn.0366-6999.2010.13.005.

10 de-Madaria E, Buxbaum JL, Maisonneuve P, et al. Aggressive or moderate fluid resuscitation in acute pancreatitis. *New Engl J Med* 2022; **387:** 989–1000. https://doi.org/10.1056/nejmoa2202884.

11 Vege SS, DiMagno MJ, Forsmark CE, et al. Initial medical treatment of acute pancreatitis: American Gastroenterological Association institute technical review. *Gastroenterology* 2018; **154:** 1103–39. https://doi.org/10.1053/j.gastro.2018.01.031.

12 Siriwardena AK, Jegatheeswaran S, Mason JM, et al. A procalcitonin-based algorithm to guide antibiotic use in patients with acute pancreatitis (PROCAP): a single-centre, patient-blinded, randomised controlled trial. *Lancet Gastroenterol Hepatol* 2022; **7:** 913–21. https://doi.org/10.1016/S2468-1253(22)00212-6.

13 Moka P, Goswami P, Kapil A, et al. Impact of antibiotic-resistant bacterial and fungal infections in outcome of acute pancreatitis. *Pancreas* 2018; **47:** 489–94. https://doi.org/10.1097/MPA.0000000000001019.

14 Da Costa DW, Bouwense SA, Schepers NJ, et al. Same-admission versus interval cholecystectomy for mild gallstone pancreatitis (PONCHO): a multicentre randomised controlled trial. *Lancet* 2015; **386:** 1261–8. https://doi.org/10.1016/S0140-6736(15)00274-3.

15 van Santvoort HC, Besselink MG, Bakker OJ, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. *New Engl J Med* 2010; **362:** 1491–502. https://doi.org/10.1056/nejmoa0908821.

16 van Brunschot S, van Grinsven J, van Santvoort HC, et al. Endoscopic or surgical step-up approach for infected necrotising pancreatitis: a multicentre randomised trial. *Lancet* 2018; **391:** 51–8. https://doi.org/10.1016/S0140-6736(17)32404-2.

17 Boxhoorn L, van Dijk SM, van Grinsven J, et al. Immediate versus postponed intervention for infected necrotizing pancreatitis. *New Engl J Med* 2021; **385:** 1372–81. https://doi.org/10.1056/nejmoa2100826.

18 Bang JY, Lakhtakia S, Thakkar S, et al. Upfront endoscopic necrosectomy or step-up endoscopic approach for infected necrotising pancreatitis (DESTIN): a single-blinded, multicentre, randomised trial. *Lancet Gastroenterol Hepatol* 2024; **9:** 22–33. https://doi.org/10.1016/S2468-1253(23)00331-X.

19 Nayar MK, Bekkali NLH, Bourne D, et al. Novel multidisciplinary hub-and-spoke tertiary service for the management of severe acute pancreatitis. *BMJ Open Gastroenterol* 2021; **8**. https://doi.org/10.1136/bmjgast-2020-000501.

Practice points

- The Revised Atlanta classification aids in stratifying AP severity and guiding management
- Early recognition of organ dysfunction is crucial in improving AP outcomes
- Minimally invasive approaches, such as endoscopic drainage and the 'step-up' approach, are preferred for managing local complications of pancreatitis
- Nutritional support should be initiated early, with enteral feeding preferred over parenteral
- Timely referral to specialist centres for complex cases of necrotizing pancreatitis improves patient outcomes