Acute Pancreatitis

Definition
A disorder of the exocrine pancreas, and is associated with acinar cell injury with local and systemic inflammatory responses. The inflammation may range from mild oedema to peri-pancreatic necrosis.

Epidemiology
Acute pancreatitis is a potentially lethal disease that is increasing in incidence. Its mortality has improved as a result of a better understanding of the natural history of the disease and improvement of critical care. Incidence varies from 4.5 to 79.8 per 100,000 per year in different countries. This variation is due to different diagnostic criteria, geographical factors, and changes over time. A 10-fold increase in its incidence from 1960 to 1980, with a mortality rate from 1% to 9%, was noted. Approximately 210,000 patients are admitted to hospitals each year with acute pancreatitis, with approximately 20% meeting criteria for severe pancreatitis alone in the US. The mortality rate is influenced by the severity of the disease, and several prognostic factors have been investigated and described. In contrast to the milder form of the disease, which has a mortality rate of 1%, the mortality associated with severe acute pancreatitis is 10% with sterile and 25% with infected pancreatic necrosis. Gallstone pancreatitis is more common in white women >60 years of age, especially among patients with microlithiasis. Alcoholic pancreatitis is seen more frequently in men.

Aetiology
Several aetiological factors have been described for acute pancreatitis, but in 10% to 20% of cases an aetiological factor cannot be identified. These cases are then considered idiopathic. The presence of microlithiasis or biliary sludge accounts for 80% of idiopathic pancreatitis. In the US, gallstones followed by alcohol intake are responsible for 80% to 90% of cases of acute pancreatitis. The most common cause worldwide is alcohol consumption. Other causes include:

- Hypertriglyceridaemia
- Hypercalcaemia
- Pancreatic malignancy
- Post-endoscopic retrograde cholangiopancreatography (ERCP) (2% to 3%)
- Trauma
- Infections (mumps, mycoplasma, Epstein-Barr virus, Ascaris lumbricoides, HIV-related co-infections)
- Drugs (sulphonamides, azathioprine, thiazides, furosemide, oestrogens, valproic acid)
- Autoimmune conditions (collagen vascular diseases)
- Pancreas divisum
- Sphincter of Oddi dysfunction
- Heredity.

Pathophysiology
The exact mechanism by which pancreatitis occurs is unknown. Several pathophysiological processes have been described that ultimately lead to intra-pancreatic zymogen activation and auto-digestion with destruction of the acinar cell. Pancreatic ductal obstruction and hypersecretion have been mentioned as factors that contribute to the initiation of the inflammatory process.

Intra-acinar activation of trypsinogen still plays a central role in the pathogenesis of acute pancreatitis, resulting in activation of proteases that ultimately causes cell damage. Some investigations have led to newer hypotheses, including ischaemia/reperfusion injury and enzymatic co-localisation. Ethanol-induced pancreatitis has different pathophysiological mechanisms. Studies have described that ethanol is a direct toxic insult to the acinar cell, causing inflammation and membrane destruction. Other mechanisms include sphincter of Oddi dysfunction, induction of hypertriglyceridaemia, or formation of free oxygen radicals. Some studies have demonstrated that ethanol causes an increase in ductal pressures secondary to protein deposition within the pancreatic duct, favouring retrograde flow and intra-pancreatic enzymatic activation.

Classification

Balthazar classification
This is a classification based on the extent of pancreatic inflammation and the presence or absence of fluid collections or gas suggestive of necrosis on CT with IV contrast.

- A: Normal
- B: Focal or diffuse gland enlargement; small intra-pancreatic fluid collection
- C: Any of the above plus peri-pancreatic inflammatory changes and <30% gland necrosis
- D: Any of the above plus single extra-pancreatic fluid collection and 30% to 50% gland necrosis
- E: Any of the above plus extensive extra-pancreatic fluid collection, pancreatic abscess, and >50% gland necrosis.

General pathological classification
- Surgical textbooks often distinguish between oedematous and haemorrhagic pancreatitis, based on pathological/histological features:
- Oedematous pancreatitis: pancreatic parenchyma and surrounding retroperitoneal structures are engorged with interstitial fluid and infiltration of inflammatory cells
• Haemorrhagic pancreatitis: bleeding into the parenchyma and surrounding retroperitoneal structures with extensive pancreatic necrosis.

**Secondary prevention**

The most important aspect of prevention is patient education. Eating a balanced, low-fat diet, maintaining adequate triacylglyceride control, and decreasing the amount of alcohol intake are a few dietary and behavioural measures that may decrease the incidence of acute pancreatitis. Effectively, addressing gallstone disease by any means available (such as cholecystectomy, endoscopic retrograde cholangiopancreatography [ERCP], ursodeoxycholic acid), may decrease the ductal obstruction risk and hence the risk of pancreatitis. Other risk factors may be controlled through patient education and medicine dose adjustments. Probiotics, antioxidants and immune nutrition have no role in the prevention of acute pancreatitis. Several studies have addressed the use of pharmacological treatment (somatostatin, gabexate, glycerine trinitrate, nafamostat mesylate), adequate patient selection, and stent placements during ERCP to prevent pancreatic injury. The use of somatostatin has been linked to a better protection against ERCP-induced pancreatitis than gabexate in some studies, but two meta-analyses yielded different conclusions. Stents are an option for endoscopists with experience in the field, but the manipulation to obtain biliary access (rather than patient characteristics or endoscopist experience) is the main factor in the development of ERCP-induced pancreatitis.

The use of a guidewire bile duct cannulation technique during ERCP has been shown to decrease the incidence of post-ERCP pancreatitis in comparison with the standard contrast injection cannulation. Those with idiopathic chronic pancreatitis, recurrent acute pancreatitis, or a family history of pancreatitis should be considered for genetic testing, especially in the setting of pancreatic cancer. The clinical relevance and the therapeutic consequences of the gene mutations leading to pancreatitis are still controversial, and genetic testing is recommended when a patient with idiopathic pancreatitis is under 25 years of age at diagnosis or when one or more family members have either pancreatitis or pancreatic cancer. Genetic analysis of asymptomatic family members should only be offered after adequate genetic counselling, and antenatal diagnosis is not recommended.

**Diagnostic tests**

<table>
<thead>
<tr>
<th>1st tests to order</th>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>serum amylase</strong></td>
<td>Normal range: 35 to 118 units/L. Amylase levels should be checked in every patient with severe abdominal pain. It has a sensitivity of 75% to 92% and a specificity of 20% to 60% with a positive predictive value approaching 100%. The sensitivity of the test is limited by hypertriglyceridaemia and alcoholism, and the specificity by inflammatory intra-abdominal processes and parotid and submandibular salivary gland inflammation. Levels start rising over the first 2 to 12 hours, peak at 48 hours, and return to normal within 3 to 5 days. Tends to be higher in patients with gallstone pancreatitis than in alcoholic pancreatitis.</td>
<td>3 times the upper limit of the normal range</td>
</tr>
<tr>
<td><strong>serum lipase</strong></td>
<td>Lipase is more sensitive (50% to 99%) and specific (86% to 100%) than amylase; however, the utility is limited in acute pancreatitis due to discrepancies in measurement method, patient selection, and cut-off points. Hence, the determination of lipase is not necessary in a patient with a clinical diagnosis of acute pancreatitis and 3 times the normal value of serum amylase. Its use can be helpful in patients with a clinical presentation suggestive of pancreatitis and normal amylase. Due to additional cost and lack of benefit in the majority of patients, utilising serum lipase in conjunction with serum amylase is considered inappropriate. Levels start rising 4 to 8 hours after the onset of pain, peak at 24 hours, and last for 8 to 14 days. Patients with alcoholic pancreatitis have higher levels of lipase than those with gallstone pancreatitis.</td>
<td>can be elevated if amylase normal</td>
</tr>
<tr>
<td><strong>ratio of serum lipase:amylase</strong></td>
<td>Low sensitivity. Favours alcoholic pancreatitis.</td>
<td>&gt;5</td>
</tr>
<tr>
<td><strong>urinary amylase</strong></td>
<td>&gt;5000 international units/24 hours</td>
<td></td>
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<tr>
<td><strong>AST/ALT</strong></td>
<td>Low sensitivity and specificity for pancreatitis. if &gt;3 times the upper normal limit, predicts gallstone disease as aetiology in 95% of cases</td>
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<tr>
<td><strong>FBC and differential</strong></td>
<td>Mild leukocytosis with left shift and elevated haematocrit as a result of dehydration or low haematocrit as a result of haemorrhage can be seen. The development of haemoconcentration has been associated to predict the risk of developing necrotising pancreatitis. leukocytosis</td>
<td></td>
</tr>
<tr>
<td><strong>haematocrit</strong></td>
<td>Indicator of severity and prognosis. if &gt;44% on admission, is a predictor of pancreatic necrosis</td>
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arterial blood gas
It is important to monitor the arterial oxygenation, since patients may be hypoxaemic, requiring supplemental oxygen. During the initial management, consider arterial blood gases every 12 hours for the first 3 days to assess both oxygenation and acid-base status.

abdominal plain film
Abnormal in two-thirds of patients.

ultrasound
Sensitivity in detecting pancreatitis is 62% to 95%. Is non-invasive, easy to perform at the bedside, and inexpensive. Limited by obesity, bowel gas, and is operator-dependent. Useful when biliary causes are suspected. The use of endoscopic ultrasound allows tissue diagnosis and has replaced endoscopic retrograde cholangiopancreatography (ERCP).

CXR
may show atelectasis and pleural effusion (especially in the left side)

ultrasound
may show pancreatic inflammation, peri-pancreatic stranding, calcifications, or fluid collections

Tests to consider

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
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<tbody>
<tr>
<td>C-reactive protein (CRP)</td>
<td>Indicator of severity. Useful after first 36 to 48 hours. if &gt;200 units/L, is associated with pancreatic necrosis</td>
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<tr>
<td>abdominal CT scan</td>
<td>CT scan with IV contrast is the most sensitive and specific study for confirming diagnosis of pancreatitis. Has a sensitivity of 90% and specificity of 100%. It is used when clinical and biochemical findings are equivocal. Ranson score of &gt;3 or APACHE II score of &gt;8 to detect and stage complications, and when patients have persisting organ failure, show signs of sepsis, or present with clinical deterioration or do not improve after 48 to 72 hours of treatment. Signs of complicated pancreatitis are usually seen 3 days after the onset of abdominal pain. findings may include diffuse or segmental enlargement of the pancreas with irregular contour and obliteration of the peri-pancreatic fat, necrosis, or pseudocysts</td>
</tr>
<tr>
<td>magnetic resonance cholangiopancreatography (MRCP)</td>
<td>MRCP is superior to CT scanning for staging acute pancreatitis and detecting complications. It also has the advantage of not requiring IV contrast. findings may include stones, diffuse or segmental enlargement of the pancreas with irregular contour and obliteration of the peri-pancreatic fat, necrosis, or pseudocysts</td>
</tr>
<tr>
<td>endoscopic retrograde cholangiopancreatography (ERCP)</td>
<td>ERCP has a limited use as a diagnostic tool in acute attacks of acute pancreatitis and has been mostly replaced by ultrasound and MRCP. Indications are preoperative evaluation to verify duct condition in patients with traumatic pancreatitis, in patients with severe pancreatitis and suspected biliary obstruction (allows sphincterotomy, stone removal, stent placement, tissue diagnosis) that do not improve after 24 hours of conservative management, and as work-up for idiopathic pancreatitis. Studies have shown a reduction of morbidity and mortality in patients with early ERCP (&lt;24 hours) and biliary disease. identifies stones and allows their retrieval during the same intervention; can identify duct filling defects and strictures</td>
</tr>
<tr>
<td>fine needle aspiration</td>
<td>If sepsis is suspected in patients with pancreatic necrosis, a percutaneous needle aspiration can be performed to rule out bacterial colonisation. identification of causative organism if bacterial colonisation</td>
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</table>

Emerging tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
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</thead>
<tbody>
<tr>
<td>urinary trypsinogen-2</td>
<td>Elevated</td>
</tr>
<tr>
<td>serum IL-6</td>
<td>Elevated</td>
</tr>
<tr>
<td>serum IL-8</td>
<td>Elevated</td>
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</tbody>
</table>
Differential diagnosis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Differentiating signs/symptoms</th>
<th>Differentiating tests</th>
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</thead>
</table>
| **Peptic ulcer disease**   | ● Longstanding epigastric pain, which does not generally radiate to the back; reflux; heartburn; and anorexia. Identifiable causes such as non-steroidal anti-inflammatory drug (NSAID) use, *Helicobacter pylori*, Zollinger-Ellison’s syndrome may be present.     | ● May improve with proton pump inhibitors, lifestyle modifications, and *H pylori* treatment.  
● Normal lipase and amylase.  
● Tonometry may show evidence of reflux.  
● Laboratory evaluation will show normal values of amylase and lipase.  
● Endoscopic evaluation will be diagnostic after visualising erosions, erythema, or ulcers, and allows biopsies to be performed. |
| **Perforated viscus**      | ● Will present with acute abdomen, peritoneal signs, tachycardia, and sepsis. Generally the abdomen is rigid and tender in all 4 quadrants, with guarding.                                                                                       | ● Normal lipase. May have elevated amylase (usually less marked than that seen in acute pancreatitis).  
● Plain x-rays show sub-diaphragmatic air.                                                                                                                   |
| **Oesophageal spasm**      | ● Dysphagia, odynophagia, weight loss, history of retrosternal pain. Physical examination may be normal.                                                                                                                        | ● A swallow study may demonstrate a contracted and abnormal-appearing oesophagus with increased pressures on oesophageal manometry. |
| **Intestinal obstruction** | ● History of abdominal surgeries (especially colon resection, caesarean sections, and aortic procedures).  
● Hernias in the physical examination.  
● Presents with abdominal distension (depends on the level of obstruction), tympanism, decreased bowel sounds, anorexia, emesis (quality depends on location of obstruction), obstipation, or constipation.   | ● Normal lipase and amylase.  
● Acute abdominal series will show ground glass appearance, air-fluid levels, distended bowel loops, absence of distal gas, pneumatosis.  
● An abdomen/pelvic CT scan may be more diagnostic, and will show point of transition and potentially identify aetiology (such as volvulus, hernias, intussusception, masses). |
<p>| <strong>Abdominal aorta aneurysm</strong> | ● Cardiovascular risk factors; hyperlipidaemia, tobacco, diabetes                                                                                                                                                    | ● High index of suspicion is necessary to make a rapid diagnosis and improve outcomes. In stable patients, where history and physical examination are equivocal, a CT angiography may be useful as a rapid way to make diagnosis. |</p>
<table>
<thead>
<tr>
<th>Condition</th>
<th>Signs and Symptoms</th>
<th>Management</th>
</tr>
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<tbody>
<tr>
<td>Mellitus, homocystinaemia</td>
<td>• Acute tearing-like abdominal pain, pulsating abdominal mass, hypotension, and mottled lower extremities with decreased pulses and abdominal distension.</td>
<td>If too unstable for radiographic evaluation, patients usually go directly to surgery.</td>
</tr>
<tr>
<td>Cholangitis</td>
<td>• Charcot's triad (jaundice, right upper quadrant pain, and fever) present in 70% of patients, altered mental status, and hypotension indicate biliary sepsis, usually caused by gram-negative bacteria.</td>
<td>Several clinical findings are present more frequently in cholangitis, such as fever (95%), right upper quadrant pain (90%), and jaundice (80%).</td>
</tr>
<tr>
<td>Choledocholithiasis</td>
<td>• Severe right upper quadrant pain of sudden onset, jaundice, acholia, choluria, and hx of cholelithiasis. May occlude the common bile duct and cause pancreatitis.</td>
<td>Normal lipase and amylase.</td>
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<tr>
<td></td>
<td>• Normal lipase and amylase.</td>
<td>Ultrasound will show gallstones, stones within the common bile duct with extra-hepatic and/or intra-hepatic duct dilatation.</td>
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<td>• Chemistry will show biochemical obstruction, with increased levels of total and direct bilirubin, alkaline phosphatase, gamma-GT, and a slight increase in ALT/AST but normal levels of pancreatic enzymes (especially lipase).</td>
<td>Blood cultures are usually positive, especially during episodes of chills, with <em>Escherichia coli</em> and <em>Klebsiella</em> as the most common micro-organisms isolated from infected bile.</td>
</tr>
<tr>
<td>Cholecystitis</td>
<td>• Pain is generally triggered after a fatty meal and localised in the right upper quadrant. More common in overweight females between 40 and 50 years of age.</td>
<td>Normal lipase and amylase.</td>
</tr>
<tr>
<td></td>
<td>• Anorexia, nausea, and vomiting may be present. May show a positive Murphy's sign and low-grade fever.</td>
<td>A right upper quadrant ultrasound will show thickened gallbladder wall, stones with acoustic shadows, biliary sludge, peri-cholecystic fluid, and sonographic Murphy's sign, and allows evaluation of the duct system. Can suggest pancreatic head inflammation. May show mild leukocytosis and a very mild elevation of liver enzymes.</td>
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<td>A hepatobiliary iminodiacetic acid (HIDA) scan is diagnostic when there is no filling of the gallbladder or with delayed emptying of the radiotracer.</td>
</tr>
<tr>
<td>Viral gastroenteritis</td>
<td>• Generalised non-specific abdominal pain, anorexia, nausea, emesis, diarrhoea, and dehydration.</td>
<td>Normal lipase and amylase.</td>
</tr>
<tr>
<td></td>
<td>• Is usually a self-</td>
<td>Important to obtain serum electrolytes and an FBC.</td>
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<td>Hypokalaemia and alkalosis may be seen secondary to diarrhoea, vomiting, and dehydration.</td>
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<td>Stool examination for microscopy, culture, osmolality, ova, parasites, <em>Clostridium</em></td>
</tr>
</tbody>
</table>
limiting viral infection but if fever is documented, bacterial and invasive organisms should be suspected.

- Consider in travellers and immunosuppressed patients.
- Consider osmotic and secretory diarrhoea from hx.

**Hepatitis**

- Jaundice, right upper quadrant pain, anorexia, and general malaise. Choluria and acholia may be seen.
- Examination: tenderness to palpation over the right upper quadrant and enlarged liver.
- Normal lipase and amylase.
- Elevated liver function tests are characteristic. AST/ALT in the range of the 1000 units/L is not rare. Serological titres can make diagnosis of aetiological cause.
- Radiographic studies not important for its diagnosis.

**Mesenteric ischaemia**

- Patients are usually older, may have a history of atrial fibrillation and risk factors for peripheral vascular disease.
- Hypercoagulable states may lead to bowel necrosis. Pain is usually out of proportion to the finding of the physical examination.
- High index of suspicion of diagnosis is necessary. Angiography and CT scan may be useful in diagnosis as well as lactic acid levels.
- Normal lipase. May have elevated amylase (usually less marked than that seen in acute pancreatitis).

**Myocardial infarction**

- Pain is usually retrosternal with radiation to jaw, neck, and left upper extremity. Associated with shortness of breath, nausea, vomiting, and diaphoresis. Cardiovascular risk factors in the history.
- Elevated cardiac enzymes (creatine kinase or creatine phosphokinase, troponins), ECG changes, and clinical scenario make the diagnosis.
- Normal lipase and amylase.
- Cardiac catheterisation, perfusion scans, and echocardiograms are useful during the work-up of cardiac ischaemia.

**Step-by-step diagnostic approach**

The diagnosis of pancreatitis is always one of exclusion, so it must be included with any complaint of severe abdominal pain. History and examination can be indicative of acute pancreatitis; however, the keys for diagnosis are elevated levels of amylase and lipase, which need to be at least 3 times their normal value.

**History**

A detailed history is imperative to narrow the large number of differentials of abdominal pain. Metabolic, nutritional, and procedural aetiologies of pancreatitis should be considered during history-taking. A detailed family history is important to rule out collagen vascular diseases, cancer, or hereditary pancreatitis. Any medicine and indications for their use should be reviewed, since many medications can have pancreatic injury as an adverse effect. Age and sex are important demographic variables, since the 2 most common causes of acute pancreatitis differ. Gallstone
pancreatitis is seen most commonly in patients with gallbladder disease - the 5 “Fs”: fat, forty, female, fertile, and family history. Alcoholic pancreatitis is seen more frequently in men, generally younger than those with gallstone pancreatitis. Patients usually manifest after an average of 4 to 8 years of alcohol intake, and bingeing behaviour increases the risk of acute pancreatitis.

Patients may present with agitation and confusion, and in severe distress. They may give a history of anorexia, nausea, and vomiting with poor oral intake. The most common symptom is severe mid-epigastric pain that radiates to the back (band distribution), worsens with movement, and is alleviated when assuming the fetal position (bent over, with spine, hips, and knees flexed). Gallstone pancreatitis may be more acute in onset than alcoholic pancreatitis, which may be preceded by a few days of mild epigastric discomfort.

**Physical examination**

Signs of hypovolaemia (decreased skin turgor, dry mucous membranes, hypotension) are usually found. Patients may appear diaphoretic, tachycardic, and tachypnoeic. Fever may indicate a complicated pancreatitis or may simply represent cytokine release as part of the inflammatory process. Decreased breath sounds may be detected if there is a pleural effusion (more common on the left side); this is seen in up to 50% of patients with acute pancreatitis. The abdominal examination may reveal a tender and distended abdomen with diminished bowel sounds (if an ileus has developed) and voluntary guarding to palpation of the upper abdomen. There may be a mild rigidity without re-bound tenderness. Clinical signs of hypocalcaemia are rare but may be evident, such as facial muscle spasm when facial nerve is tapped (Chvostek’s sign) and carpopedal spasm when blood pressure cuff is applied (Trousseau’s sign). Complicated haemorrhagic pancreatitis may exhibit ecchymotic discoloration of several areas, including the peri-umbilical skin (Callen’s sign), over both flanks (Grey-Turner’s sign) or over the inguinal ligament (Fox’s sign), and may be seen as soon as presentation or 24 to 48 hours after the onset.

**Laboratory work-up**

Any patient with an acute abdomen should have an FBC with a differential and a blood chemistry including renal, liver, and pancreatic function tests. Mild leukocytosis with left shift and elevated haemocrit as a result of dehydration or low haemocrit as a result of haemorrhage can be seen. The development of haemoconcentration is associated with an increased risk of developing necrotising pancreatitis. As a result of dehydration there may be some degree of pre-renal azotaemia, manifested by elevated creatinine and urea. In the absence of cholestocholiathiasis, liver function tests are usually normal, but a slight increase in alkaline phosphatase and bilirubin may be seen. The key for diagnosis is elevated levels of amylase or lipase; amylase levels more than 3 times the normal value are highly specific for acute pancreatitis. The degree of pancreatic inflammation is not directly correlated with the amylase or lipase absolute value. Due to additional cost and lack of benefit in the majority of patients, utilising serum lipase as a first test in conjunction with serum amylase is considered inappropriate. Measuring lipase can be helpful in patients with a clinical presentation suggestive of pancreatitis in whom the amylase levels were normal.

It is important to monitor arterial oxygenation, since patients may be hypoxaemic, requiring supplemental oxygen. During initial management, arterial blood gases should be considered every 12 hours for the first 3 days to assess both oxygenation and acid-base status. CRP (C-reactive protein) is performed subsequently and is an indicator of severity; useful after the first 36 to 48 hours.

If sepsis is suspected in patients with pancreatic necrosis, a percutaneous needle aspiration can be performed to rule out bacterial colonisation.

**Imaging**

Radiographic studies are not used for diagnosis of acute pancreatitis, but may determine possible causative factors and exclude other diagnoses. A CXR may show pleural effusion and basal atelectasis, and a sentinel loop (isolated dilatation of a segment of gut) may be seen in a KUB. Plain abdominal x-ray may reveal a sentinel loop adjacent to the pancreas, gas distending the right colon that abruptly stops in the mid- or left transverse colon (cut-off sign), or calcifications. Magnetic resonance cholangiopancreatography (MRCP), endoscopic retrograde cholangiopancreatography (ERCP), trans-abdominal ultrasound, and endoscopic ultrasound are usually indicated in patients with elevated liver function tests suggestive of bile duct obstruction, to exclude strictures, neoplasms, or stones. Ultrasound is considered to be the preferred initial study in suspected gallstone pancreatitis as it is inexpensive, easy to perform at the bedside, and allows examination of the gallbladder and bile duct system. Its sensitivity in detecting pancreatitis is 62% to 95%. It is limited by obesity and bowel gas, and is operator-dependent. The use of endoscopic ultrasound allows tissue diagnosis and has replaced ERCP.

The advantage of the ERCP is that it has the added benefit of treating certain aetiological conditions (e.g., by stone removal, stent placement, or sphincterotomy), but it has been largely superseded as a diagnostic modality by MRCP and ultrasound. MRCP is generally used in patients with renal insufficiency, in whom the use of CT with IV contrast is discouraged. It is superior to CT scanning for staging acute pancreatitis and detecting complications.

Patients who show signs of systemic inflammatory response or sepsis, or who do not improve, should have a CT scan to rule out peri-pancreatic collections, necrosis, and abscess. Areas of non-perfusion indicate infected pancreatic necrosis.

**Emerging tests**

Urine trypsinogen-2 is now considered a better serological screening test than amylase but is not yet used clinically; sensitivity of 94% and specificity of 95%. Interleukins IL-6 and IL-8 are inflammatory mediators that have been recently described as predictive serum markers for development of severe acute pancreatitis.

**Treatment Options**

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Tx Line</th>
<th>Treatment</th>
</tr>
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<tbody>
<tr>
<td>all patients</td>
<td>1st</td>
<td>initial resuscitation</td>
</tr>
<tr>
<td></td>
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<td>• IV hydration with crystalloids is essential, and an effort to keep urinary output above 30 mL/hour is necessary to avoid potential kidney damage.</td>
</tr>
</tbody>
</table>
- Pain control is important when pain is present, and the most commonly used pharmacological drugs are the opioids. Pethidine, fentanyl, or morphine can be used, either for breakthrough pain or as patient-controlled analgesia. Monitor respiratory and CNS depression. Meperidine is considered superior to morphine because it does not increase sphincter of Oddi pressures.
- Ketorolac, a non-steroidal anti-inflammatory drug (NSAID), can be used in patients with intact renal function.
- Ondansetron is the most commonly used antiemetic.

**Primary Options**

crystalloids: 20-40 mL/kg intravenously

and

pethidine: 25-100 mg subcutaneously/intramuscularly every 4 hours when required

and

ondansetron: 2-4 mg intravenously every 4-6 hours when required

**Secondary Options**

crystalloids: 20-40 mL/kg intravenously

-- AND --

morphine sulphate: 1-5 mg intravenously every 4 hours when required

or

fentanyl: 50-100 micrograms intravenously, followed by 50 micrograms every 1-2 hours when required

or

ketorolac: 10 mg intravenously/intramuscularly initially, followed by 10-30 mg every 4-6 hours when required for 2 days, maximum 90 mg/day

-- AND --

ondansetron: 2-4 mg intravenously every 4-6 hours when required

**nutritional support**

- Initially patients should be kept nothing by mouth. Oral intake is re-started when there is notable clinical improvement, nausea and abdominal pain has resolved, and amylase levels have dropped to normal. Premature resumption of diet may result in exacerbation of the disease. In patients who are expected to have a prolonged nothing by mouth status, a trans-pyloric nasojunostomy feeding tube should be placed, allowing enteral nutrition without stimulating the pancreas.
- The regimen should provide 25 to 35 kcal/kg/day energy, 1.2 to 1.5 g/kg/day protein, 3 to 6 g/kg/day carbohydrates, and 2 g/kg/day lipids.
- Parenteral nutrition should be reserved for patients who do not tolerate enteral feeding or in whom an adequate infusion can not be reached within 2 to 4 days.

**calcium replacement therapy**

- In severe cases of pancreatitis, hypocalcaemia should be identified and treated accordingly. Calcium should be titrated to normal serum ionic calcium levels. Calcium chloride is used less frequently, since it must be given by a central line.

**Primary Options**

calcium gluconate: 2-15 g/day intravenously given as infusion or in divided doses, or see local protocol for dosing guidelines; 500-1000 mg orally four times daily

**magnesium replacement therapy**

- Magnesium should be replaced if low levels are identified, commonly seen in alcoholic patients. Renal function (creatinine) should be checked before magnesium administration. It may need daily replacement.

**Primary Options**
**magnesium sulphate**: 1-2 g intravenously every 6 hours on first day, followed by 60 mg/kg/day as an infusion, or see local specialist protocol for dosing guidelines

**adjunct insulin**
- Blood glucose control and insulin administration to keep glucose <8.33 mmol/L (<150 mg/dL) has been associated with reductions in morbidity and mortality in critically ill patients. In less severe cases, regular insulin sliding scales can be used.
- See local specialist protocol for dosing guidelines.

**adjunct antibiotic therapy**
- Decontamination of the gut, where antibiotics are given to decrease the intra-luminal bacterial count, has not been proven effective in decreasing the incidence of pancreatic sepsis, but a meta-analysis of 8 randomised controlled trials found reduction in mortality of patients with severe acute pancreatitis when prophylactic IV antibiotics with pancreatic penetrance were administered.
- It is important, however, to limit the use of antibiotics for this subset of patients to avoid fungal superinfection.
- Imipenem is the antibiotic most studied because of its pancreatic penetration.
- In patients with necrotising pancreatitis, antibiotic use should be restricted to patients in whom there are signs, symptoms, and laboratory tests indicating that infection is present (fever, leukocytosis, organ failure, and positive cultures).
- There is no consensus in the literature to date of how long patients need to be on antibiotics. However, clinical improvement, with resolution of organ failure and improvement of systemic markers of inflammation, can be considered as reasonable indicators that antimicrobials can be stopped.

**Primary Options**

- cilastatin+imipenem: 500-1000 mg intravenously every 6 hours
- More

**Secondary Options**

- ceftriaxone: 1-2 g intravenously every 12 hours
- OR
- ampicillin: 500 mg intravenously every 6 hours
- OR
- ciprofloxacin: 400 mg intravenously every 12 hours

**with gallstones: surgical candidates**

**adjunct cholecystectomy**
- In patients in whom the diagnosis of acute gallstone pancreatitis is obtained by ultrasound, a cholecystectomy with common bile duct exploration (either surgical or postoperatively with endoscopic retrograde cholangiopancreatography [ERCP]) should be performed during the same hospitalisation for the acute attack, soon after the attack resolves. A longer delay, even a few weeks, is associated with a high recurrence (80%).
- When a laparotomy is performed for diagnosis and mild to moderate pancreatitis is found, cholecystectomy with intra-operative cholangiogram should be performed but the pancreas should be left alone. For severe pancreatitis, the lesser sac should be opened and the pancreas fully inspected. Some surgeons place drains and irrigating catheter around the pancreas.

**with gallstones: non-surgical candidates**

**adjunct endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy**
- In patients who are not candidates for surgery because of comorbidities with a high ASA index, sepsis, or severe disease, ERCP must be considered.
- The use of ERCP and sphincterotomy, performed within 72 hours after the onset of the disease, has been shown to decrease the incidence of concomitant biliary sepsis and systemic complications in severe but not in mild forms of pancreatitis. Contradictory results have been obtained for mortality rates.
- A meta-analysis of 4 randomised controlled trials of endoscopic sphincterotomy in patients with severe acute pancreatitis showed that sphincterotomy reduced complications and mortality. The role of ERCP in patients without biliary obstruction or cholangitis is unknown. Cholecystectomy has to be considered after discharge.

**with alcohol-induced disease**

**plus benzodiazepines**
Patients with alcohol-induced pancreatitis may need alcohol-withdrawal prophylaxis. Lorazepam is generally used in this group of patients.

**Primary Options**

**lorazepam**: 1-2 mg orally/intravenously/intramuscularly every 6-8 hours

**vitamin and mineral replacement**

- The objective of replacement of thiamine in chronic alcoholism is to replenish the stores in patients. The treatment has to be continued until the patient can return to eating a well-balanced meal during hospitalisation.
- Other water-soluble vitamins that are supplemented during hospital or emergency department course include folic acid and cyanocobalamin. Cyanocobalamin can be given orally except in states when absorption is impaired.

**Primary Options**

**thiamine**: 100 mg orally/intravenously/intramuscularly once daily

**folic acid**: 1 mg orally/intramuscularly once daily

**cyanocobalamin**: 1000 micrograms intramuscularly/orally once daily for 1-2 weeks, followed by 1000 micrograms once every 1-3 months

With infected pancreatic necrosis plus **necrosectomy**

- If sepsis is considered, a percutaneous needle aspiration can be performed to rule out bacterial colonisation. Necrosectomy involves resection of the necrotic pancreatic tissue and placement of irrigating drains within the debrided spaces, allowing postoperative lavage. The only criteria for debridement are multi-organ failure (with or without infection) with CT evidence of necrotic tissue.
- Outcomes are better if surgery is delayed until the necrosis has organised, usually about 4 weeks after the onset of abdominal pain. Allowing organisation of the inflammation makes the dissection through planes easier.

**Treatment approach**

The main goal of initial treatment is to prevent complications of severe pancreatitis by reducing pancreatic secretory stimuli and correction of fluid and electrolyte abnormalities. Initially, the patients should be fluid resuscitated and kept nothing by mouth with bowel rest when nausea, vomiting, and abdominal pain are an issue. At diagnosis supportive care continues until pain is resolved and diet re-started. The majority of patients will improve within 3 to 7 days of conservative management. Patients with organ failure or with poor prognostic signs (Glasgow score >3, APACHE score >8, and Ranson score >3) should be admitted to the intensive care unit.

**Initial resuscitation**

Resuscitation with IV fluids, analgesics, and antiemetics are the initial treatments even before diagnosis is made. IV hydration with crystalloids is essential, and an effort to keep urinary output above 30 mL/hour is necessary to avoid potential kidney damage. The patient should be catheterised to monitor urinary output in severe cases of acute pancreatitis. The adequacy of fluid replacement is the single most important aspect of the medical management. In haemorrhagic pancreatitis, blood transfusion may be necessary. Pain control is important when pain is present, and the most commonly used pharmacological drugs are opioids. Pethidine, fentanyl, or morphine can be used, either for breakthrough pain or as patient-controlled analgesia (PCA). Ketorolac, a non-steroidal anti-inflammatory drug (NSAID), can be used in patients with intact renal function. It is important to monitor the arterial oxygenation, since patients may be hypoxaemic, requiring supplemental oxygen. During the initial management, consider arterial blood gases every 12 hours for the first 3 days to assess both oxygenation and acid-base status.

**Severe pancreatitis**

In severe cases of pancreatitis, hypocalcaemia should be identified and treated accordingly. Treatment of this electrolyte imbalance is important, since hypocalcaemia may lead to cardiac dysrhythmias. Magnesium should be replaced if low levels are identified, commonly seen in alcoholic patients. Blood glucose control and insulin administration to keep glucose <8.33 mmol/L (<150 mg/dL) has been associated with reductions in morbidity and mortality in critically ill patients. Insulin sliding scales, insulin drips, or long-acting insulin should be used in patients with hyperglycaemia that is difficult to treat. The use of antibiotics is not a routine practice, and controversial results have been obtained in recent studies. However, some studies have shown some benefit in cases of severe necrotising pancreatitis. The main indication for necrosectomy is infection in severe necrotising pancreatitis.
**Nutrition**

A period of nothing by mouth is required initially, especially during the first 24 to 48 hours after the onset of pancreatitis (the phase of resuscitation and control of nausea and pain). This reduces exocrine stimulation by cholecystokinin and secretin, and reduces the risk of aspiration if a dynamic ileus is present. Oral intake is re-started when there is notable clinical improvement, nausea and abdominal pain has resolved, and amylase levels have dropped to normal. Premature resumption of diet may result in exacerbation of the disease. In patients who are expected to have a prolonged nothing by mouth status, a trans-pyloric nasojejunostomy feeding tube should be placed beyond the ampulla of Vater. This allows enteral nutrition without stimulating the pancreas. Parenteral nutrition should be reserved for patients who do not tolerate enteral feeding or in whom an adequate infusion cannot be reached within 2 to 4 days. When compared with parenteral nutrition, enteral feeds are associated with better outcomes, less mortality, and better blood glucose control. They also protect the gut barrier by preventing intestinal atrophy, leading to less sepsis and fewer infectious complications. The recommended nutrient requirements in severe acute pancreatitis are as follows: energy 25 to 35 kcal/kg/day, protein 1.2 to 1.5 g/kg/day, carbohydrates 3 to 6 g/kg/day, and lipids 2 g/kg/day.

**Alcohol-induced pancreatitis**

Patients with alcohol-induced pancreatitis may need alcohol-withdrawal prophylaxis. Lorazepam, thiamine, folic acid, and multi-vitamins are generally used in this group of patients.

**Gallstone pancreatitis**

In patients in whom the diagnosis of acute gallstone pancreatitis is obtained by ultrasound, imaging of the common bile duct is required. If the presence of stones in the common bile duct is confirmed, a cholecystectomy with common bile duct exploration (either surgical or postoperatively with endoscopic retrograde cholangiopancreatography [ERCP]) should be performed during the same hospitalisation in mild to moderate disease soon after the attack resolves. A longer delay, even of a few weeks, is associated with a high recurrence (80%). If the pancreatitis is severe, some allow a few months for the inflammation to completely resolve before performing a cholecystectomy. In patients who are not candidates for surgery because of comorbidities with a high American Association of Anesthesiology (ASA) index, sepsis, or severe disease, ERCP must be considered. Urgent ERCP is indicated in patients with biliary sepsis and obstructive jaundice that show no improvement in 48 hours after the onset of the attack. ERCP is a diagnostic and therapeutic intervention. It allows defining of the biliary system anatomy, to remove stones causing jaundice, sepsis, and pancreatitis, as well as placement of stents for strictures or pancreatic duct leaks (secondary to pancreatitis or trauma), and permits facilitation of biliary drainage by performing of sphincterotomy.

**Pancreatitis found on laparotomy**

When a laparotomy is performed for diagnosis and mild to moderate pancreatitis is found, cholecystectomy with intra-operative cholangiogram should be performed but the pancreas should be left alone. For severe pancreatitis, the lesser sac should be opened and the pancreas fully inspected. Some surgeons place drains and irrigating catheter around the pancreas.

### Step by step

**Monitoring**

Long-term monitoring is not necessary. Patients usually resolve after their acute attack. If they modify their risk factors, another episode may not recur later in life. Lipids should be monitored in those with hypertriglyceridaemia.

**Patient Instructions**

Before discharge from hospital after an acute attack of acute pancreatitis, patients should be advised to modify lifestyle risk factors. For example, alcoholic patients need to stop drinking, especially bingeing behaviour, modify diet in order to control hypertriglyceridaemia, and use lipid-lowering medicines such as statins or niacin. Patients taking medicines (e.g., furosemide, didanosine, oestrogens) should be educated about the adverse effects and how to recognise an acute attack of pancreatitis.

Patients should be advised to eat small, low-fat meals of carbohydrates and proteins, with a gradual increase in quantity over a period of 3 to 6 days as tolerated.

<table>
<thead>
<tr>
<th>Complication</th>
<th>Timeframe</th>
<th>Likelihood</th>
</tr>
</thead>
<tbody>
<tr>
<td>acute renal failure</td>
<td>short term</td>
<td>high</td>
</tr>
<tr>
<td><strong>see our comprehensive coverage of Acute renal failure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seen in patients with severe acute pancreatitis. May be caused by circulating toxins or rhabdomyolysis. Hypovolaemia and inflammatory mediators. Acute renal failure is a complication with poor outcome.</td>
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<td></td>
</tr>
<tr>
<td>necrotising pancreatitis</td>
<td>short term</td>
<td>medium</td>
</tr>
<tr>
<td>Secondary to inadequate fluid resuscitation, vasoactive and toxic substances (phospholipases, endotoxins, activated trypsin, complement activation, thromboxane, and elastase).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pancreatic abscess</td>
<td>short term</td>
<td>low</td>
</tr>
<tr>
<td>Occurs when the peri-pancreatic fluid collections become colonised and infected. Invariably fatal if not treated surgically. Follows secondary bacterial contamination of necrotic pancreatic tissue and haemorrhagic exudates. It is unknown whether prophylactic antibiotics given early in the course of the disease decrease the incidence of abscess. Generally</td>
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</tbody>
</table>
patients present 2 to 4 weeks after the onset of pancreatitis, with fever, and clinically worsen. CT is diagnostic, showing a ring-enhancing fluid collection with gas. Its treatment is drainage (surgical versus percutaneous) and antibiotics to cover *E. coli, Bacteroides, Staphylococcus, Klebsiella, Proteus*, and *Candida albicans*.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Duration</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatic insufficiency</td>
<td>long term</td>
<td>low</td>
</tr>
<tr>
<td>Recurrent attacks may lead to exocrine pancreatic insufficiency more commonly than endocrine failure.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic pancreatitis</td>
<td>long term</td>
<td>low</td>
</tr>
<tr>
<td>Recurrent attacks of acute pancreatitis may lead to chronic scarring, and, if the aetiological factor is not treated, may present with the classic characteristics of chronic pancreatitis: glucose intolerance, pancreatic insufficiency, and calcifications.</td>
<td></td>
<td></td>
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<tr>
<td>Portal vein/splenic thrombosis</td>
<td>long term</td>
<td>low</td>
</tr>
<tr>
<td>Ongoing pancreatic inflammation may cause irritation and inflammation of the portal vein and/or splenic vein, leading to portal hypertension. Suspect splenic vein thrombosis in patients with recurrent pancreatitis, splenomegaly, and bleeding from gastric varices.</td>
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<td></td>
</tr>
<tr>
<td>Enteric fistulas</td>
<td>long term</td>
<td>low</td>
</tr>
<tr>
<td>Resulting from inflammation surrounding the pancreas and adjacent duodenum or transverse colon.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intestinal obstruction</td>
<td>long term</td>
<td>low</td>
</tr>
<tr>
<td>Ileus may be frequently seen in pancreatitis, a result of dehydration, electrolyte abnormalities, or adjacent bowel inflammation. Intestinal obstruction can be seen later in the course of the disease, when a pseudocyst or abscess causes a mechanical compression of the bowel (generally duodenum or transverse colon).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multorgan failure</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>The gut mucosa plays a central role in the development of multi-organ failure. Several descriptions about how the gut modulates the inflammatory response by priming neutrophils and secreting cytokines can be found in the literature.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreatic ascites</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>Consists of accumulated pancreatic fluid in the peritoneal cavity. It is due to chronic leakage of a pseudocyst, but some cases may be due to duct disruption. Clinically manifested by weight loss and unresponsiveness of the ascites to diuretics. Initial treatment involves hyper-alimentation and somatostatin. If no improvement is obtained in 2 to 3 weeks, endoscopic retrograde cholangiopancreatography (ERCP) and surgery should be considered.</td>
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<tr>
<td>Pancreatic effusion</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>Secondary to pancreatic fistula draining into the chest. The diagnosis is based on thoracentesis with fluid rich in amylase and a CT scan/retrograde pancreatogram that shows the fistula. Its treatment consists of drainage with a chest tube, somatostatin, and total parenteral nutrition. If fistula persists, operative intervention with fistula resection or distal pancreatectomy should be performed.</td>
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</tr>
<tr>
<td>Infected pancreatic necrosis</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>Infection is responsible for 80% of deaths. Gram-negative bacteria (<em>Esterichia coli, Pseudomonas, Klebsiella, Proteus, Enterobacter</em>) are more common than gram-positive micro-organisms. Previous studies describe a mortality rate of 50% to 80% in the absence of operative treatment and 10% to 40% among patients who receive debridement.</td>
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</tr>
<tr>
<td>Acute lung injury/ARDS</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>See our comprehensive coverage of Acute respiratory distress syndrome</td>
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<tr>
<td>The production and excretion of inflammatory mediators (such as cytokines, prostaglandins, and thromboxanes) during pancreatitis may damage the alveolocapillary membrane, leading to destruction of pneumocytes and decrease in the amount of surfactant. This leads to airway dysfunction, increase in superficial tension, and inadequate oxygenation. Patients usually present with hypoxaemia, requiring higher levels of supplemental oxygen, with bilateral interstitial infiltrates, PaO2:FIO2 ratio &lt;300, and a normal pulmonary capillary wedge pressure. Patients may require mechanical ventilation during the course of their disease.</td>
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<td></td>
</tr>
<tr>
<td>Disseminated intravascular coagulation</td>
<td>variable</td>
<td>low</td>
</tr>
<tr>
<td>See our comprehensive coverage of Disseminated intravascular coagulation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe acute pancreatitis, especially if associated with necrosis, has been linked to liberation of cytokines and systemic inflammatory response, with activation of the complement, coagulation, and fibrinolytic cascades, leading to a state of coagulopathy and disseminated intravascular coagulation with elevated levels of split fibrin products and d-dimer with low fibrinogen.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sepsis</td>
<td>variable</td>
<td>low</td>
</tr>
<tr>
<td>See our comprehensive coverage of Sepsis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The gut mucosa plays a central role in the development of sepsis. Several descriptions about how the gut modulates the inflammatory response by priming neutrophils and secreting cytokines can be found in the literature. Gram-negative bacteria are the main cause of sepsis in patients with acute pancreatitis, and the gut mucosa is considered as the source of such organisms. Therefore, it is important to maintain integrity of the anatomical barrier by providing enteral nutrition.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudocyst</td>
<td>variable</td>
<td>low</td>
</tr>
</tbody>
</table>

See our comprehensive coverage of Acute respiratory distress syndrome.
Pseudocysts are encapsulated collections of fluid with high enzyme concentrations. The walls are formed by inflammatory fibrosis of the peritoneal, mesenteric, and serosal membranes, which limits the spread of the pancreatic fluid. Pseudocysts have no epithelial lining. Pseudocyst diagnosis should be suspected when a patient fails to respond after 1 week of treatment or symptoms recur. Pain is the most common finding, followed by a palpable mass. CT scan is the diagnostic study of choice. Pseudocysts can be complicated with infection, rupture (in 5%), and haemorrhage. The principal indications for treatment are to improve symptoms and to prevent complications. Expectant management is important in the first 6 to 12 weeks of existence of cysts that have arisen during an acute attack of acute pancreatitis. The chance of spontaneous resolution is 40%. Thereafter, for cysts >5 cm in size, treatment is usually recommended over conservative management. Treatment options include excision of the cyst, external drainage (surgical or percutaneous), or internal drainage (preferred method of treatment), which can be either a cystojejunostomy Roux-en-Y, cystogastrostomy, or cystoduodenostomy.

**gastrointestinal bleeding**

| variable | low |

**see our comprehensive coverage of Assessment of upper GI bleed**

From adjacent inflamed stomach or duodenum, ruptured pseudocyst, or peptic ulcer.

**intra-peritoneal bleeding**

From coeliac or splenic artery rupture or acute splenic vein thrombosis.

**Prognosis**

The majority of patients with acute pancreatitis will improve within 3 to 7 days of conservative management. The cause of pancreatitis should be identified, and a plan to prevent recurrence should be initiated before the patient is discharged from hospital. In gallstone pancreatitis, a cholecystectomy should be considered before discharge in mild cases and a few months after the discharge date in patients with severe symptoms. In patients who are not candidates for surgery, endoscopic retrograde cholangiopancreatography (ERCP) must be considered.

Long-term prognosis is based on the aetiological factor and patient compliance to lifestyle modifications. Acute pancreatitis generally resolves and leaves pancreatic function intact. May progress to chronic pancreatitis in the event of recurrent alcoholic intake, pancreas divisum, or cystic fibrosis. The most commonly used prognostic scores are APACHE II, Ranson, Glasgow, Balthazar, and Atlanta.