



Acute pancreatitis: symptoms, diagnosis and management

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Pancreatitis is a relatively common condition that affects approximately one per cent of the population. Although the majority of attacks are mild, it is fatal in one in four people who develop it in a severe form. Clinical manifestations are so varied that the condition should be considered in the differential diagnosis of all upper abdominal pain until serum amylase concentrations become normal. Specific treatment is unavailable, therefore therapy is supportive, including the management of complications if and when they develop.

The pancreas is an elongated gland situated just behind the stomach. Inflammation of the gland (pancreatitis) is the most significant benign condition of the organ, and may be acute or chronic (Henry and Thompson, 2001). In acute pancreatitis the gland will return to normal, but in its chronic form there is permanent anatomical and functional abnormality (Henry and Thompson, 2001; Alexander et al, 2000; Haslett et al, 1999; Banks, 1997).

Acute pancreatitis affects approximately one per cent of the population (Lam and Lombard, 1999), but about 70 per cent of attacks are mild. However, of those individuals who develop severe forms of the disease, one in four will die (Forrest et al, 1995).

The function of the pancreas

The pancreas is an elongated structure approximately 15cm long, weighing between 85 and 100g. Its head lies near the duodenum while its tail extends towards the spleen. It is both an exocrine and endocrine gland. The exocrine portion consists of acini cells, which produce pancreatic juice, and duct cells, which produce a bicarbonate-rich fluid. Ducts leading from the acini cells converge on larger ducts, which eventually converge on the pancreatic duct, which carries pancreatic juices from the pancreas to the duodenum.

The endocrine portion of the pancreas consists of several hundred thousands of cells called pancreatic islets or islets of Langerhans, which produce hormones that enter the circulatory system. Each islet is composed of alpha, beta and delta cells, among others. Nerves from both the autonomic and sympathetic nervous systems innervate the islets of Langerhans and each network is surrounded by a well-developed capillary network (Germann and Stanfield, 2002; Seely et al, 1995).

The exocrine and endocrine portions of the pancreas secrete digestive enzymes and hormones (Lam and Lombard, 1999). The main secretions are shown in Box 1.

Relative to its weight, the pancreas secretes more protein than any other tissue in the body, most of it in the form of digestive enzymes. Because many of these enzymes (protease in particular) are capable of breaking down the molecules that make up the pancreas itself, they are stored within the acini cells in inactive forms.

The stimulation of pancreatic juice begins in the acini cells, which produce a relatively small volume of fluid containing water, electrolytes and digestive enzymes. As this fluid flows through the ducts leading from the acini cells, a larger volume of bicarbonate-rich fluid is added to it. Although both enzyme-rich and bicarbonate-rich fluids are secreted at the same time during a meal, the regulatory mechanisms that control their secretion are somewhat different, therefore the composition of pancreatic juice can be varied.

Pathophysiology

Acute oedematous pancreatitis

In pancreatitis, the pancreatic enzymes trypsin, lipase and protease, which are normally activated in the duodenum, are prematurely activated, causing auto-digestion of the pancreas. This leads to varying degrees of oedema, haemorrhage, necrosis, and abscess and cyst formation in and around the pancreas.

Activated enzymes, such as trypsinogen, chymotrypsinogen, phospholipase, elastase and catalase, are responsible for increased capillary permeability. This permits large amounts of fluid to escape into the peritoneal and retroperitoneal cavity, causing damage to the surrounding tissues.

Acute oedematous pancreatitis is characterised by interstitial inflammation and oedema. It may be self-limiting, or it may progress into necrotising pancreatitis.

BOX 1. SECRETIONS OF THE EXOCRINE AND ENDOCRINE PORTIONS OF THE PANCREAS

Exocrine secretions		Endocrine secretions
Lipase	trypsin	Bicarbonate
Chymotrypsin		Cholesterol esterase
Amylase		Ribonuclease
Protease		Deoxyribonuclease
Nuclease		
		Insulin
		Glucagon
		Somatostatin

Necrotising pancreatitis

Necrotising pancreatitis is a severe form of pancreatitis and causes necrosis of the pancreas. It results from microcirculatory stasis within the gland, leading to infarction. Tissues surrounding the pancreas may also develop necrotic changes (peripancreatic necrosis). The necrotic areas may become infected, possibly by organisms from the adjacent bowel (infected necrosis) (Henry and Thompson, 2001; Alexander et al, 2000; Haslett et al, 1999), and there may also be haemorrhage from eroded vessels (Henry and Thompson, 2001).

Causes of pancreatitis

The main causes of pancreatitis are as follows (Henry and Thompson, 2001; Haslett et al, 1999; Lam and Lombard, 1999; Travis et al, 1998):

- Gallstones: the passage of stones down the common bile duct may promote reflux of infected bile along the pancreatic duct. Patients with multiple small stones in the gallbladder are more likely to develop pancreatitis than those with large solitary stones. The stones cause intraductal activation of pro-enzymes, following which cell membranes are digested and oedema, proteolysis, vascular damage and necrosis may develop;
- Alcohol: alcohol itself can damage the pancreas, but often the gland has been previously damaged by alcohol. The precise mechanism of alcohol damage is unknown.

Other causes are:

- Endoscopic retrograde cholangiopancreatography;
- Drugs, including corticosteroids, azathioprine, sodium valproate, frusemide, tetracycline;
- Infections, for example, mumps, coxsackievirus, hepatitis A, hepatitis B;
- Metabolic irregularities, examples are hypercalcaemia, hyperlipidaemia;
- Trauma;
- Iatrogenic causes, for example, after surgery;
- Scorpion stings.

Signs and symptoms

The main symptom of pancreatitis is acute onset abdominal pain in the epigastric region that may radiate to the back and be associated with nausea and vomiting (Alexander et al, 2000).

A serum amylase more than four times the upper limit (over 1000iu/L) is diagnostic of pancreatitis, but late onset (more than 12 hours) of a perforated duodenal ulcer or an ectopic pregnancy may cause a similar rise in amylase. It is the severity of the symptoms that characterises pancreatitis (Travis et al, 1998).

Physically, the patient with pancreatitis may appear acutely unwell with signs of shock, abdominal tenderness and guarding/rigidity (Henry and Thompson, 2001). There may be bruising around the umbilicus (Cullen's sign) and in the loin region (Grey Turner's sign), but these are rare manifestations (Alexander et al, 2000). Bowel sounds may become absent as paralytic ileus develops (Haslett et al, 1999).

BOX 2. INVESTIGATIONS FOR PANCREATITIS

Investigations should include the following:

- Serum amylase test
- X-ray of the abdomen: this may reveal distended loops of small bowel with paralytic ileus over the pancreatic region (Henry and Thompson 2001; Alexander et al, 2000)
- X-ray of the chest: this may show pulmonary oedema; left pleural effusion may develop as a result of the release of toxins (Alexander et al, 2000). If air under the diaphragm is seen on the X-ray, the cause of the pain is gastrointestinal perforation and not pancreatitis (Henry and Thompson, 2001)
- Ultrasound scan: this may confirm diagnosis but in the early stages of pancreatitis the pancreas may not be grossly swollen (Haslett et al, 1999). The scan will also reveal whether gallstones are present. A well visualised normal pancreas makes pancreatitis unlikely (Travis et al, 1998)
- Computed axial tomography (CAT) scanning: this is indicated if the pancreas cannot be seen on ultrasound or if the attack is severe (Henry and Thompson, 2001; Travis et al, 1998). A CAT scan may also be used to assess the viability of the gland and to detect areas of necrosis, involvement of the colon, blood vessels and other structures (Alexander et al, 2000; Haslett et al, 1999)
- Blood tests for urea and electrolyte levels: these are important indicators of the state of hydration
- Full blood count: haemoglobin levels may be low if the patient has haemorrhagic pancreatitis. A raised white cell count ($9-20 \times 10^9/L$) will reveal an active inflammatory process (Alexander et al, 2000)

Diagnosis

The clinical manifestations of pancreatitis are so varied that the condition should be considered in the differential diagnosis of all upper abdominal pain until serum amylase concentrations become normal.

Hyperamylasaemia alone cannot be relied upon and must be evaluated in conjunction with the history and physical signs (Henry and Thompson, 2001). Amylase is efficiently excreted by the kidneys and may be normal if measured 24-48 hours after onset. In this situation the urinary amylase to creatinine ratio can be measured (Haslett et al, 1999). This can stay elevated for 10-14 days (Alexander et al, 2000).

Nursing implications

The role of the nurse in managing and treating patients who have pancreatitis has been described by Banks (1997) and Alexander et al (2000). The following are

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BOX 3. POTENTIAL COMPLICATIONS OF PANCREATITIS

- Cardiovascular collapse because of hypovolaemia or the release of inflammatory cytokines. Cardiac function may also be directly depressed
- Hypoxia: this is common and has many contributing factors; for example, abdominal distension, cytokine release and bacterial translocation from the gut. Adult respiratory distress syndrome may develop
- Hypocalcaemia as a result of calcium deposits in areas of fat necrosis
- Hyperglycaemia from disturbance of insulin metabolism
- Deranged coagulation
- Paralytic ileus, which exacerbates fluid and electrolyte imbalance
- Hypovolaemic shock from vomiting, hypoalbuminaemia, ascites and retro-peritoneal haemorrhage
- Acute renal failure as a result of local intravascular coagulation in the renal vascular bed and hypovolaemia
- A necrotic pancreas, which may need excision
- Pseudocysts, which may need to be drained

Sources: Alexander et al, 2000; Lam and Lombard, 1999; Travis et al, 1998.

- Offer regular analgesia to promote comfort. Anti-emetics may be needed to control nausea and vomiting;
- Give prescribed intravenous fluids and other products to correct hypovolaemia, and keep the patient well hydrated. Accurate fluid balance is essential – patients generally require hourly urine measurement while they are in the acute stage;
- Give oral care to nil-by-mouth patients and monitor for paralytic ileus. This will involve care of nasogastric tube aspiration and psychological support;
- Administer the prescribed antibiotics and ensure universal infection control measures are practised to protect the patient, who may be septic from other infections;
- Regularly turn the patient to help protect the skin, and encourage deep breathing exercises to help prevent atelectasis and promote removal of secretions;
- Take measures to protect the patient from thromboembolism, for example, ensure the correct size TED stockings are worn, and promote exercise. If the patient is lethargic, passive exercise may be appropriate in some cases. In addition, low molecular weight heparin may be prescribed for some patients;
- Undertake regular observations. In the acute stage it

may be necessary to take patients' blood pressure, pulse, temperature and respiration measurements at least hourly. Continuous electrocardiogram and oxygen saturation monitoring should also be undertaken. The nurse should act on the results accordingly;

- Give core nursing care, such as regular hygiene, as the patient may sweat considerably;
- Measure blood glucose levels four-hourly – insulin may need to be administered on a sliding scale;
- Psychological support is paramount, as the patient may be in pain, scared and feeling very ill;
- Provide support and explanation during investigations. These are shown in Box 2;
- Educate the patient on lifestyle measures; for example, advise keeping to a low fat diet to help prevent gallstones and/or to reduce alcohol consumption if this is appropriate for the particular patient.

Treatment options

An opiate analgesia such as pethidine should be used, rather than morphine, which can cause spasm of the sphincter of Oddi (Travis et al, 1998).

Patients who are haemorrhagic may require blood or fresh frozen plasma to maintain circulatory blood volume and adequate urine output (Henry and Thompson, 2001; Alexander et al, 2000).

Patients should be kept nil by mouth to reduce stimulation of pancreatic enzymes. Insertion of a nasogastric tube may be indicated for persistent vomiting or for paralytic ileus (Alexander et al, 2000; Haslett et al, 1999; Travis et al, 1998).

If there is evidence of infection, a broad spectrum antibiotic should be given intravenously (Alexander et al, 2000). Blood glucose levels should be monitored, as secondary diabetes can develop as a result of disruption of insulin metabolism (Henry and Thompson, 2001; Alexander et al, 2000; Haslett et al, 1999).

It may be necessary to monitor cardiac status if electrolyte derangement is shown to be severe (Alexander, 2000). Intravenous nutrition should be initiated if paralytic ileus persists (Henry and Thompson, 2001). Thromboembolism prophylaxis may be required (Haslett et al, 1999).

Because there is no specific treatment for pancreatitis, therapy is supportive, including the management of complications if and when they develop (Box 3).

Conclusion

Pancreatitis is a relatively common but serious condition from which patients may take several weeks to recover. It has a number of causes, and varied clinical manifestations. The condition should be considered in the differential diagnosis of all upper abdominal pain until serum amylase concentrations become normal.

There is no specific treatment for pancreatitis, and patients often require large amounts of nursing time. They will need support from a wide range of health care professionals. Psychological help may be appropriate for many patients. ■