Abstract
Acute pancreatitis is a potentially life-threatening condition primarily associated with gallstones or prolonged and excessive alcohol intake. Although the initial triggers of the condition can vary, the resulting pathophysiology is broadly similar irrespective of the cause. This article explores the pathophysiology of the main causes of acute pancreatitis, and discusses nursing management of the condition in the acute setting and the long-term issues to consider. It also outlines the conservative management of the condition, which includes pain management, provision of fluids and nutritional care. In the management of acute pancreatitis, nursing care can often overlap with medical management, especially as the condition deteriorates. Therefore, it is important that nurses develop comprehensive knowledge of the management of acute pancreatitis.

Aims and intended learning outcomes
This article aims to review the function of the pancreas and the causative factors, presentation and nursing management of acute pancreatitis. It also considers potential long-term complications associated with the condition, such as pancreatic damage leading to diabetes mellitus. After reading this article and completing the time out activities you should be able to:
» Understand the anatomy and physiology of the pancreas.
» Outline the common and rare causes of acute pancreatitis.
» Explain how the progression and outcomes of acute pancreatitis can be predicted.
» Discuss the treatment and management options for patients with acute pancreatitis.

Introduction
Acute pancreatitis is the most common pancreatic disease globally (Xiao et al 2016), and its incidence is increasing. Despite medical advances and the development of clinical guidelines, acute pancreatitis continues to be associated with significant mortality rates (Greenberg et al 2016). However, the incidence of, and mortality associated with, pancreatic diseases varies significantly worldwide, together with disparities in the reported incidence between World Health Organization regions (Xiao et al 2016). This might be because of more accurate reporting in developed countries compared with developing countries.

Acute pancreatitis is a potentially serious, reversible condition that features sudden onset of abdominal pain triggered by inflammation of the pancreas. It is most commonly associated with gallstones, with their migration causing transient obstruction of the pancreatic duct, or prolonged and excessive alcohol intake (Johnson et al 2014, Porth 2015, Forsmark et al 2016). Individuals with acute pancreatitis usually present with acute abdominal pain and are admitted to...
surgical units for assessment. In cases of cholecystectomy, surgery such as cholecystectomy (surgical removal of the gallbladder) may be required, while in other cases a conservative approach involving pain management, provision of fluids and nutritional care should be implemented. In addition, there are often long-term care considerations, particularly when the cause of acute pancreatitis is alcohol-related.

**Anatomy and physiology of the pancreas**

The pancreas is a small organ that is divided into the head, body and tail. The pancreas sits in the abdominal cavity behind the greater curve of the stomach, with the tail extending towards the spleen (Figure 1) (Tortora and Derrickson 2013, VanPutte et al 2016). The pancreas has several important endocrine and exocrine functions related to digestion and metabolism (VanPutte et al 2016), for example producing enzymes that break down food (Marieb 2015).

The pancreas is directly linked to the duodenum (a section of the small intestine) via the pancreatic duct, which allows pancreatic juice containing digestive enzymes to flow to the duodenum. Bile, which is produced by the liver and stored in the gallbladder, reaches the duodenum via the common bile duct, which joins with the pancreatic duct at the hepatopancreatic ampulla, commonly known as the ampulla of Vater. This creates a shared entry point to the duodenum, which may be anatomically significant in the development of acute pancreatitis because it is a site of potential occlusion.

**TIME OUT 1**

A nursing student asks you to summarise the main endocrine functions of the pancreas. Prepare a teaching session outlining these functions and how they might be affected by acute pancreatitis.

**Endocrine function of the pancreas**

The endocrine function of the pancreas is associated with glycaemic control as well as gastric function. Clusters of cells in the pancreas known as the islets of Langerhans or pancreatic islets produce varying levels of the hormones insulin and glucagon, depending on blood glucose levels (Tortora and Derrickson 2013, VanPutte et al 2016). The two other main hormones produced by the pancreatic islets, somatostatin and pancreatic polypeptide, have a role in controlling digestion.

**Exocrine function of the pancreas**

Exocrine glands are those that secrete substances onto an epithelial surface. For example, the liver and pancreas secrete bile and pancreatic juice into the gastrointestinal tract to support digestion. Pancreatic acinar cells secrete an alkaline fluid containing digestive enzymes into the pancreatic ducts (VanPutte et al 2016). The pancreatic duct enters the duodenum via the hepatopancreatic ampulla, where pancreatic fluid mixes with intestinal fluid in the duodenum, providing a buffer against the acid chyme arriving from the stomach (Tortora and Derrickson 2013).

The pancreatic acinar cells produce enzymes that digest carbohydrates, proteins and fats (lipids). These enzymes include lipase, pancreatic amylase and several protein-digesting enzymes, in particular trypsin and chymotrypsin (VanPutte et al 2016). The pancreatic acinar cells also produce various enzymes that break down deoxyribonucleic acid (DNA) and ribonucleic acid (RNA). The protein-digesting enzymes secreted in the pancreas are produced in inactive form to prevent digestion of pancreatic tissue itself. In addition, the pancreatic acinar cells produce trypsin inhibitors to prevent any accidentally activated enzymes acting on the pancreatic tissue. Protein-digesting enzymes are usually activated in the duodenum by enterokinase (VanPutte et al 2016). Enterokinase activates trypsin, which in turn activates further pancreatic protein-digesting enzymes, enabling them to participate in digestion (Tortora and Derrickson 2013, VanPutte et al 2016).

**Causes of acute pancreatitis**

There are several causes of acute pancreatitis, as listed in Box 1. The most common causes are gallstones, which accounts for approximately 50% of cases, and prolonged and excessive alcohol intake, which accounts for approximately 25% of cases (Johnson et al 2014).
Acute pancreatitis is a potentially serious, reversible condition that features sudden onset of abdominal pain triggered by inflammation of the pancreas. It is most commonly associated with gallstones, with their migration causing transient obstruction of the pancreatic duct, or prolonged and excessive alcohol intake.

■ Pain is a significant presenting feature of acute pancreatitis and must be considered as part of the initial and ongoing management of the condition.

Gallstone-associated acute pancreatitis

Bile aids the digestion of lipids in the small intestine. It is produced by the liver and stored in the gallbladder. Gallstones commonly form in the gallbladder when there is excessive excretion of cholesterol, and to a lesser extent bilirubin (a waste by-product of the breakdown of red blood cells), into the bile. This thickens the bile and potentially leads to gallstone formation (Porth 2015). When protein and fatty acid-rich contents enter the small intestine from the stomach, cholecystokinin stimulates the gallbladder to contract, forcing bile along the common bile duct and into the duodenum (Porth 2015, VanPutte 2016).

Where gallstones have formed in the gallbladder, contraction can cause these to be forced out along with bile, often causing sharp pain as they travel along the bile duct. Smaller gallstones can more easily pass into the duodenum, whereas larger gallstones can become lodged in the common bile duct or pancreatic duct, potentially blocking the outflow of pancreatic juice (Porth 2015). Gallstones that obstruct the sphincter of Oddi (the sphincter that controls the flow of bile and pancreatic juice through the hepatopancreatic ampulla and into the duodenum) can cause it to spasm, leading to reflux of duodenal contents into the pancreatic duct; while gallstones that become lodged in the common bile duct can lead to reflux of bile into the pancreatic duct. The presence of duodenal contents and/or bile in the pancreas can trigger premature activation of the pancreatic enzymes inside the pancreas, leading to digestion of pancreatic tissue, which causes inflammation (Lankisch et al 2015).

Alcohol-related acute pancreatitis

There are several theories about the development of alcohol-related acute pancreatitis. Earlier theories explored the larger pancreatic ducts that carry pancreatic juice to the duodenum. Alcohol in the circulation was thought to trigger spasms in the sphincter of Oddi, causing bile to backflow into the ducts and leading to enzyme activation, digestion of pancreatic tissue and inflammation (Lankisch et al 2015).

There has also been increased focus on the smaller ducts and the development of protein plugs created by microscopic activation of enzymes that partially digest proteins produced by the pancreas. These partly broken-down proteins can increase in size, damaging the adjacent pancreatic tissue, blocking smaller ducts and preventing the outflow of pancreatic juice (Lankisch et al 2015). In addition, alcohol has a direct toxic effect on the pancreatic acinar cells and how these activate digestive enzymes. Finally, the alcohol and metabolites of alcohol breakdown are thought to directly damage cells inside the pancreas (Lankisch et al 2015).

Key points

■ Acute pancreatitis is a potentially serious, reversible condition that features sudden onset of abdominal pain triggered by inflammation of the pancreas. It is most commonly associated with gallstones, with their migration causing transient obstruction of the pancreatic duct, or prolonged and excessive alcohol intake.

■ The more inflamed the pancreas becomes, the greater the risk of extensive and potentially severe pancreatic damage. Therefore, early identification and intervention is vital. In addition, patients with severe acute pancreatitis are at risk of developing a range of complications such as hypoxaemia (low concentration of oxygen in the blood), sepsis, pleural effusion and collections of fluid outside the pancreas (Krenzer 2016).

■ Pain is a significant presenting feature of acute pancreatitis and must be considered as part of the initial and ongoing management of the condition.

Part 2: Other causes of pancreatitis

There are several other possible causes of pancreatitis. Gallstones are the commonest cause, occurring in approximately 50% of cases. Other possible causes include hypercalcaemia, hypertriglyceridaemia, high alcohol intake and idiopathic pancreatitis (without known cause), which occurs in approximately 10% of cases. Other rare causes of pancreatitis include gallstones (approximately 5% of cases). Gallstones can cause inflammation of the pancreas, leading to digestion of pancreatic tissue, which causes pain.

In addition, patients with severe acute pancreatitis are at risk of developing a range of complications such as hypoxaemia (low concentration of oxygen in the blood), sepsis, pleural effusion and collections of fluid outside the pancreas. Early intervention is vital in these cases.

Pancreatic inflammation

Regardless of the cause of acute pancreatitis, it is thought that trypsin is activated initially, which triggers the activation of other digestive enzymes (Porth 2015). Once in active form, the enzymes begin digesting pancreatic tissue and damaging pancreatic acinar cells, which triggers an inflammatory response inside the pancreas (Lankisch et al 2015). In some cases, the inflammatory process progresses to sepsis, potentially leading to multiple organ failure (Porth 2015).

Inflammation is the body’s normal response to tissue injury and involves an initial vascular phase followed by a cellular phase. The vascular phase is an immediate response to cell injury and involves vasodilatation leading to increased capillary wall permeability, which allows the

BOX 1. Causes of acute pancreatitis

Common causes

- Gallstones – approximately 50% of cases
- Prolonged and excessive alcohol intake – approximately 25% of cases
- Idiopathic pancreatitis (without known cause) – approximately 10% of cases

Rare causes – less than 5% of cases

- Many drugs, including valproate (used in epilepsy), corticosteroids, azathioprine (immunosuppressive medication)
- Endoscopic retrograde cholangiopancreatography – a technique that uses endoscopy and fluoroscopy to diagnose and treat biliary or pancreatic conditions
- Hypertriglyceridaemia (elevated levels of triglycerides) or lipoprotein lipase deficiency (a condition that disrupts the breakdown of fatty acids)
- Hypercalcaemia (high calcium levels in the blood)
- Pancreas divisum (genetic disorder of the pancreatic ducts)
- Viral infections

(Johnson et al 2014; Forsmark et al 2016)
movement of fluid into the tissue surrounding the injury (Porth 2015). Therefore, the cell damage that occurs in acute pancreatitis leads to the accumulation of fluid inside the pancreas, which causes local swelling.

In the cellular phase, leucocytes are attracted to the injured area by chemical mediators released by the damaged pancreatic cells. These white blood cells move out of the circulation and into the tissue, where they undertake phagocytosis and activate the full inflammatory response. If the local acute inflammatory response continues, this can trigger a widespread response, activating acute phase proteins, a white blood cell response and sepsis. The liver will increase production of several proteins, including C-reactive protein, and there will be an increase in levels of white blood cells as an increasing number are released from the bone marrow in response to the inflammatory process (Porth 2015).

This inflammatory process will progress while local tissue injury continues and fluid accumulates in the pancreas, worsening the pain and further damaging the pancreatic tissue (Porth 2015). The exudate that enters the pancreas as a result of inflammation is likely to be serous (clear fluid) in nature, although haemorrhage into the pancreas can result in the collection of blood where there has been significant tissue damage and blood vessels have been eroded (Porth 2015). This movement of fluid from the circulation to the pancreas will eventually lead to fluid from the pancreas entering the abdominal cavity, resulting in abdominal swelling (Krenzer 2016). Movement of fluid from the circulation will also cause a fall in blood pressure and the possibility of the development of circulatory shock in response to reduced cardiac output.

The more inflamed the pancreas becomes, the greater the risk of extensive and potentially severe pancreatic damage. Therefore, early identification and intervention is vital. In addition, patients with severe acute pancreatitis are at risk of developing a range of complications such as hypoxaemia, sepsis, pleural effusion and collections of fluid outside the pancreas (Krenzer 2016). Pleural effusion develops when there is significant pancreatic swelling, which blocks lymphatic drainage from the thoracic cavity, or if there is leakage of pancreatic enzymes in the abdomen, which then move through to the thoracic cavity. This can cause damage to the external lung tissue leading to collections of fluid in the thoracic cavity.

Predicting the progression and outcomes of acute pancreatitis

The progression and outcomes of acute pancreatitis can be challenging to predict, and various scoring tools have been developed to assist healthcare professionals; however, as Kuo et al (2015) noted, these may not be available during an emergency admission. Greenberg et al (2016) suggested that the patient’s C-reactive protein levels should be assessed on admission; a C-reactive protein level of 105mg/dL indicates severe acute pancreatitis and could predict suboptimal outcomes such as increased severity and risk of long-term complications.

An Acute Physiology Assessment and Chronic Health Evaluation II (APACHE II) (Knau et al 1985) score should also be undertaken on the patient’s admission, since a higher score will indicate severe disease – such as acute pancreatitis – and suboptimal clinical outcomes (Greenberg et al 2016). APACHE II is an acute disease severity prediction tool that considers several variables including age, vital signs, blood oxygenation and blood chemistry. Yang et al (2016) compared tools that could be used to predict severity in acute pancreatitis, finding that APACHE II was reliable. However, Kuo et al (2015) commented that C-reactive protein level alone correlates well with the severity of acute pancreatitis, adding that newer tools such as the Bedside Index of Severity in Acute Pancreatitis (Wu et al 2008) and the Harmless Acute Pancreatitis Score (Lankisch et al 2009) compare favourably in terms of accuracy to traditional tools such as APACHE II.

Clinical assessment

Patients with acute pancreatitis will usually present with severe and constant acute abdominal pain. On examination of the abdomen, acute pain tends to be epigastric and accompanied by nausea and/or vomiting. However, these signs are non-specific and can be associated with other acute conditions such as cholecystitis (inflammation of the gallbladder) or perforated peptic ulcer (Johnson et al 2014). Therefore, diagnosis of acute pancreatitis is based on the presence of at least two of the following (Wu and Banks 2013, Dooley et al 2015, Greenberg et al 2016):

- Acute abdominal pain, which has a sudden onset, is persistent, epigastric and usually radiating to the back.
- Elevated serum lipase or amylase levels.
- Characteristic findings of acute pancreatitis on a computed tomography (CT) scan or magnetic resonance imaging, such as abdominal fluid collections.

Lipase is the optimal biomedical indicator of acute pancreatitis and is a more specific marker than amylase (Dooley et al 2015). Lipase remains raised for longer following the onset of acute pancreatitis (Johnson et al 2014). Greenberg et al (2016) suggested that a serum lipase test should be undertaken in all patients with suspected acute pancreatitis, with a serum lipase level of at least three times the upper normal limit (normal range: 0-160 units/L) required for diagnosis. In the UK, testing for amylase is more widely available than testing for lipase; therefore, the patient’s serum amylase is more likely to be used as a biomedical indicator of acute pancreatitis. A serum amylase level of three times the normal upper limit (normal range: 40-140 units/L) is highly indicative of acute pancreatitis (Johnson et al 2014).

Abdominal imaging is important in the assessment and diagnosis of acute pancreatitis, because it can identify complications such as fluid collections. Bollen (2016) stated that abdominal ultrasound can clearly identify gallstones as the cause of acute pancreatitis, while in the later stages it can assist in differentiating between solid tissue and fluid collections. To clarify the extent of any pancreatic damage and/or damage to the surrounding...
tissue, a CT scan is recommended at least 72-96 hours following the onset of symptoms (Working Group International Association of Pancreatolgy (IAP)/American Pancreatic Association (APA) Acute Pancreatitis Guidelines 2013). If the patient is not responding to treatment or demonstrating signs of a systemic response such as shock, CT scanning is important to identify significant fluid collections inside and/or outside the pancreas, as well as identifying necrotic tissue (Bollen 2016). However, the IAP/APA (2013) guideline emphasises that an early CT scan is not likely to be of any diagnostic benefit because CT scoring systems do not predict severity any more effectively than a clinical scoring system.

The IAP/APA (2013) guideline also states that an early CT scan can assist in the identification of bowel ischaemia or intra-abdominal perforation in individuals presenting with both acute pancreatitis and ‘acute abdomen’ (abdominal pain of recent onset requiring urgent surgery assessment) (Watson 2010).

The revised Atlanta Classification is used to assess the severity of acute pancreatitis and subsequently guide its management, as follows (IAP/APA 2013, Sarr et al 2013, Dooley et al 2015, Lankisch et al 2015, Greenberg et al 2016):

- Mild acute pancreatitis – no organ failure and an absence of local or systemic complications.
- Moderately severe acute pancreatitis – organ failure that resolves within 48 hours and/or local or systemic complications without persistent organ failure.
- Severe acute pancreatitis – persistent single or multiple organ failure for more than 48 hours.

**Nursing management**

Following a detailed clinical assessment of the patient, which will indicate the level of severity and appropriate nursing management, there are several areas to consider when caring for someone with acute pancreatitis. Conservative management consists primarily of pain management, provision of fluids and nutritional care (Greenberg et al 2016). There is consensus in the literature that prophylactic antibiotics should not be used because the potential adverse effects, such as increased risk of systemic fungal infection, are greater than the benefits (IAP/APA 2013, Johnson et al 2014, Greenberg et al 2016).

**Pain assessment and management**

Pain is a significant presenting feature of acute pancreatitis and must be considered as part of the initial and ongoing management of the condition. Nurses caring for individuals with acute pancreatitis have an essential role in assessing and managing pain, ideally guided by a standardised pain assessment tool. The pain assessment tool used should enable the patient to describe their pain, provide consistent results irrespective of the assessor using the tool, and enable analgesics to be reviewed effectively. Falch et al (2014) recommended the use of a standard numerical rating scale when assessing pain severity in individuals with acute abdominal pain because these scales are quick and straightforward to use and demonstrate accurate results in adults.

Pain assessment is often undertaken separately from early warning score assessments, which allocate scores to routine physiological measurements such as respiration, pulse, systolic blood pressure and consciousness (Royal College of Physicians 2017), perhaps reducing the significance of pain. Purser et al (2014) suggested including pain as a fifth vital sign, with pain assessment undertaken as part of the vital sign observations, which could improve completion of pain assessment and lead to improved pain management. In Scotland, pain assessment forms part of the National Early Warning Score tool as an additional assessment (Scottish Intercollegiate Guidelines Network 2014).

Greenberg et al (2016) suggested adopting a polypharmacy approach to achieve the optimal analgesic effect in people experiencing acute pancreatitis. Opiates, non-steroidal anti-inflammatory drugs (NSAIDs) and regular paracetamol can have a role in maximising pain relief, although possible contraindications must be considered for each patient, for example caution in the use of NSAIDs in those with asthma. Krenzer (2016) stated that opiates, such as fentanyl or hydromorphone hydrochloride, can reduce pancreatic enzyme secretions, which can be effective in reducing pain and inflammation in acute pancreatitis. Overall, the literature does not provide specific guidance on the type of analgesics to use in acute pancreatitis; therefore, this must be guided by pain severity and local acute pain management guidelines.

**TIME OUT 4**

List three pain assessment tools that could be used in patients with acute pancreatitis. What would be the advantages and disadvantages of using each of these tools?

**Intravenous fluid therapy**

The first aim of treatment is to restore or maintain haemodynamic stability, because there is a significant risk of dehydration caused by a shift of fluids from the circulation to the pancreas and abdominal cavity (Greenberg et al 2016, Krenzer 2016). Therefore, providing fluids is vital when caring for a patient with acute pancreatitis. The IAP/APA (2013) guideline recommends target-directed intravenous fluid resuscitation, initially at a rate of 5-10mL/kg per hour until the target is reached. The fluid resuscitation target will vary according to the individual and the severity of the condition, although a mean arterial blood pressure of 65-85mmHg, pulse rate below 120 beats per minute and a urinary output of more than 0.5-1mL/kg per hour is suggested for patients being treated on a general ward (IAP/APA 2013). Patients who are critically ill should undergo invasive monitoring such as intrathoracic blood volume measurement, to provide precise haemodynamic monitoring and ensure that fluid therapy can be maintained (IAP/APA 2013). There is limited evidence to recommend specific types of fluid, although the IAP/APA (2013) guideline recommends the use of Ringer’s lactate solution (a solution that includes sodium chloride, calcium chloride, sodium lactate and potassium chloride) for initial fluid resuscitation.

**Nutrition**

Nutritional intake for patients presenting with acute pancreatitis...
is guided by the severity of the condition and whether any nutritional therapy is likely to exacerbate the patient’s symptoms. Krenzer (2016) suggested that the patient with acute pancreatitis should not take oral nutrition until any nausea subsides. However, specific and stage-related nutritional recommendations state that, in those presenting with mild acute pancreatitis, normal oral nutrition should commence from admission (Johnson et al 2014, Greenberg et al 2016). In these circumstances, there is no need to commence enteral nutrition since this has no benefit over oral nutrition in terms of symptom development.

If symptoms such as nausea or abdominal pain are exacerbated by oral nutrition, the person should be advised to gradually increase their oral intake from zero to a full diet, as tolerated (Johnson et al 2014, Greenberg et al 2016). In severe acute pancreatitis with significant symptoms, the IAP/APA (2013) guideline states that enteral nutrition should be used. This is administered either via the nasogastric or nasojejunal route, although Greenberg et al (2016) noted that jejunal feeding is not significantly superior to nasogastric feeding; therefore, the commencement of nutrition should not be delayed until a jejunal tube can be placed. Johnson et al (2014) and Greenberg et al (2016) asserted that no specific feed type has been found to be superior. Parenteral nutrition is only required in rare and severe cases where oral or enteral nutrition is not tolerated (IAP/APA 2013).

Nutritional support is an important aspect of the management of acute pancreatitis. The National Confidential Enquiry into Patient Outcome and Death (NCEPOD) (2016) recommended that all patients admitted with acute pancreatitis should have their risk of malnutrition assessed, using an assessment tool such as the Malnutrition Universal Screening Tool (British Association for Parenteral and Enteral Nutrition 2011), which would indicate referral for additional nutritional support, if required.

**TIME OUT 5**

Consider the provision of nutrition for patients with acute pancreatitis in your clinical area. Contact your local nutritional support team and ask about the latest evidence-based standards and guidelines for nutritional support in patients with this condition

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Specific management of gallstone-associated acute pancreatitis

Specific management during and after gallstone-associated acute pancreatitis will be influenced by the severity of the condition. Potential interventions include cholecystectomy during acute admission, planned cholecystectomy following recovery and endoscopic retrograde cholangiopancreatography (ERCP). ERCP combines endoscopy and fluoroscopy to diagnose and treat pancreatic conditions; for example, it can be used to assist in removing gallstones or inserting stents to promote drainage (Krenzer 2016). The IAP/APA (2013) guideline suggests that ERCP should be undertaken when there is bile duct obstruction seen on imaging and when the patient has cholangitis (infection of the bile duct). Recurrence of gallstone-associated acute pancreatitis is common and therefore cholecystectomy may eventually be required.

In severe acute pancreatitis, it is necessary for a patient to receive treatment for, and then recover from, the acute episode; therefore, a conservative approach involving pain management, provision of fluids and nutritional care may be used initially, before the patient is admitted later for ERCP or cholecystectomy (Krenzer 2016).

**Potential complications and long-term considerations**

If the standard management interventions do not resolve acute pancreatitis, or if there is clinical deterioration, the nurse should consider the possibility of symptomatic pancreatic fluid collections, necrotising pancreatitis or systemic complications such as hypovolaemic shock, which results from a significant loss of fluid or blood supply (Greenberg et al 2016, Krenzer 2016). Local complications such as pancreatic fluid collection are likely to resolve without intervention. Necrotic tissue within the pancreas can be sterile or infected, and, while there may be multiple areas of sterile necrosis indicating damaged tissue, it usually does not require any intervention. However, if there are signs of infected necrosis in the pancreas, drainage might be required (Dooley et al 2015). Pseudocysts and abscesses inside the pancreas can develop more than four weeks after the initial onset of acute pancreatitis and may require endoscopic drainage. Fluid collections near the pancreas are likely to self-resolve (Dooley et al 2015).

There are several possible systemic complications associated with severe disease that nurses must be aware of when there has been significant fluid loss from the circulation to the pancreas and abdominal cavity, for example respiratory dysfunction, renal dysfunction and metabolic acidosis associated with the development of shock (Krenzer 2016). Therefore, monitoring fluid and electrolyte balance, as well as arterial blood gas monitoring, are important elements of systemic monitoring in severe pancreatitis to ensure early and appropriate intervention.

Glycaemic control may be disrupted because of the effect of pancreatic damage and the physiological stress associated with acute illness on insulin production. Therefore, monitoring blood glucose levels should be a standard aspect of the nursing assessment of acute pancreatitis to ensure early intervention, such as the administration of insulin in response to high blood glucose levels. Hyperglycaemia is regarded as transient (Lankisch et al 2015), with blood glucose monitoring being discontinued after the patient is discharged. However, Das et al (2014) found that new diabetes and/or prediabetes was observed in 37% of 1,102 patients following an episode of acute pancreatitis. They also identified that the risk of developing diabetes doubled in the five years following an episode of acute pancreatitis, suggesting that the long-term monitoring of blood glucose levels is important (Das et al 2014).
Nikkola et al (2017) noted that there is an increased risk of long-term pancreatic damage leading to diabetes in those with alcohol-related acute pancreatitis. Patients with alcohol-related acute pancreatitis are at risk of developing chronic pancreatitis, with 13% of these patients developing chronic pancreatitis within 13 years (Lankisch et al 2015). Similarly, smoking significantly increases the risk of developing chronic pancreatitis, with 32% of individuals who smoke and have two episodes of acute pancreatitis developing chronic pancreatitis after three to four years (Lankisch et al 2015).

Reducing the risk of recurrence of acute pancreatitis and the development of chronic pancreatitis should be considered as part of any long-term interventions undertaken. Nordback et al (2009) found that a two-year follow-up care plan, including six-monthly gastrointestinal outpatient appointments, reduced recurrence of acute pancreatitis, as opposed to a single intervention during the acute admission. Alcohol abstinence is the optimal method for reducing the risk of recurring alcohol-related acute pancreatitis (Nikkola et al 2013), and although discussing these risks with the patient may not be possible in the acute phase of the condition, nurses have a responsibility to educate them about future risks. Support programmes designed to reduce alcohol intake over an extended period have the potential to reduce the recurrence of acute pancreatitis (Nordback et al 2009). The NCEPOD (2016) recommends that all hospitals have a seven-day smoking cessation service and that nurses should seek its advice on any patient admitted with suspected acute pancreatitis.

Conclusion

Acute pancreatitis is a complex and potentially life-threatening condition that requires early assessment and intervention to reduce the risk of deterioration.

Nurses have an important role in the management of acute pancreatitis since they are responsible for assessing fluid balance, pain and nutritional status, which is an essential aspect of its management. Understanding the altered physiology involved in acute pancreatitis will improve nurses’ understanding of appropriate management interventions and assist them in providing effective, evidence-based care.

References


(last accessed: 12 June 2018).


TIME OUT 6
Consider how assessing and managing patients with acute pancreatitis relates to The Code: Professional Standards of Practice and Behaviour for Nurses and Midwives (Nursing and Midwifery Council 2015) or, for non-UK readers, the requirements of your regulatory body.

TIME OUT 7
Now that you have completed the article, reflect on your practice in this area and consider writing a reflective account: rcni.com/reflective-account

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Acute pancreatitis
TEST YOUR KNOWLEDGE BY COMPLETING THIS MULTIPLE-CHOICE QUIZ

1. Acute pancreatitis is:
   a) A reversible condition that features sudden onset of abdominal pain triggered by inflammation of the pancreas
   b) Caused by permanent damage to the pancreas as a result of inflammation
   c) An irreversible condition that features the gradual onset of abdominal pain
   d) A non-serious condition that involves mild abdominal pain and has a low mortality rate

2. One function of the pancreas is to:
   a) Filter waste products from the blood
   b) Secrete and store bile
   c) Produce enzymes that break down food
   d) Regulate blood pressure and body temperature

3. Which of the following hormones is not produced by the pancreas?
   a) Insulin
   b) Somatostatin
   c) Oestrogen
   d) Glucagon

4. What are the two main causes of acute pancreatitis?
   a) Hypercalcaemia and hypertriglyceridaemia
   b) Viral infections and pancreas divisum
   c) Endoscopic retrograde cholangiopancreatography and use of corticosteroids
   d) Gallstones and prolonged and excessive alcohol use

5. Which of the following is not a diagnostic sign or symptom of acute pancreatitis?
   a) Acute abdominal pain
   b) Elevated serum lipase or amylase levels
   c) Depressed respiratory rate
   d) Abdominal fluid collections on computed tomography (CT) scan

6. Which statement is false?
   a) Patients with acute pancreatitis will usually present with severe and constant acute abdominal pain
   b) Amylase is the optimal biomedical indicator of acute pancreatitis
   c) A CT scan is recommended to clarify the extent of any pancreatic damage and/or damage to the surrounding tissue
   d) Abdominal ultrasound can clearly identify gallstones as the cause of acute pancreatitis

7. In the revised Atlanta Classification, severe acute pancreatitis is defined as:
   a) Persistent single or multiple organ failure for more than 48 hours
   b) Local or systemic complications without persistent organ failure
   c) Organ failure that resolves within 48 hours
   d) No organ failure and an absence of local or systemic complications

8. Which of the following is a conservative management strategy for acute pancreatitis?
   a) Assessing and managing pain
   b) Provision of fluids
   c) Nutritional care
   d) All of the above

9. What is one potential intervention for gallstone-associated pancreatitis?
   a) Blood transfusion
   b) Cholecystectomy
   c) Electrolyte replacement
   d) Prophylactic antibiotics

10. Patients with alcohol-related pancreatitis:
    a) Are at lower risk of developing chronic pancreatitis than the general population
    b) Should be advised that the risk of recurring acute pancreatitis is not reduced by abstaining from alcohol
    c) Are less likely to experience long-term pancreatic damage leading to diabetes
    d) Can reduce the risk of recurrence of acute pancreatitis through support programmes designed to reduce alcohol intake over an extended period

How to complete this quiz
This multiple-choice quiz will help you to test your knowledge. It comprises ten questions that are broadly linked to the CPD article. There is one correct answer to each question.

- You can test your subject knowledge by attempting the questions before reading the article, and then go back over them to see if you would answer any differently.
- You might like to read the article before trying the questions.

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This multiple-choice quiz was compiled by Jason Beckford-Ball

The answers to this multiple-choice quiz are:

This activity has taken me minutes/hours to complete. Now that I have read this article and completed this assessment, I think my knowledge is:

Excellent □ Good □ Satisfactory □ Unsatisfactory □ Poor □

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