Understanding Acute Pancreatitis

Two (2.0) contact hours

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Purpose and Objectives

The purpose of this course is to provide an overview of the assessment, diagnosis and management of the patient with acute pancreatitis.

After successful completion of this course, the participant will be able to:

1. Describe the incidence, pathophysiology and presentation of acute pancreatitis.
2. Discuss the differences between alcohol-induced pancreatitis and biliary pancreatitis.
3. Identify the rationale of using selected diagnostic tests in the evaluation of acute pancreatitis.
4. Describe the expected medical and surgical management of acute pancreatitis.
5. Identify potential life-threatening complications of pancreatitis.

Introduction

Acute pancreatitis is the inflammation of the pancreas that results in auto-digestion by its own pancreatic enzymes. Pancreatitis, or inflammation of the pancreas, has a variety of etiologies. Severity of the disease can range from its mildest form, which resolves quickly with few complications, to its most severe form, necrotizing pancreatitis, which is associated with an increased risk for developing multiple system organ failure and mortality (Andris, 2013).

As a healthcare professional, you are likely to be familiar with the different functions of the pancreas. The pancreas is a gland with both endocrine and exocrine functions. If you have ever provided care to an individual diagnosed with acute pancreatitis, you are aware that it can cause excruciating pain.

The management of acute pancreatitis aims to eliminate the etiologic factors for the disease while managing its complications and preventing further disease progression (Andris, 2013). Patients with mild forms of pancreatitis may improve with symptom management, whereas those with more severe disease will need significant supportive interventions.

What is Pancreatitis?

Pancreatitis is inflammation of the pancreas, which is a large gland situated behind the stomach and close to the duodenum. The pancreas secretes digestive enzymes into the duodenum through the pancreatic duct. Pancreatic enzymes join with bile (produced in the liver and stored in the gallbladder) to digest food. The pancreas also releases the hormones insulin and glucagon into the bloodstream to help regulate blood glucose levels.

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Normally, digestive enzymes secreted by the pancreas do not become active until they reach the small intestine. But when the pancreas is inflamed, the enzymes inside it attack and damage the tissues that produce them.

Pancreatitis occurs more often in men than women.

Statistics

Acute pancreatitis is one of the most common gastrointestinal disorders requiring acute hospitalization, with a reported annual incidence of about 210,000 people in the United States (NDDIC, 2014).

According to the Cleveland Clinic (2014), 80% of all cases of acute pancreatitis are mild; the remaining 20% are severe. The overall mortality rate for AP is around 5% (Cleveland Clinic, 2014).

The hospitalization rates of Caucasian patients related to acute pancreatitis are almost triple than that for African Americans. In addition, males are more likely to be hospitalized than females (Cleveland Clinic, 2014). The median age of onset of acute pancreatitis depends on the etiology or cause.

Types of Pancreatitis

Pancreatitis can be acute or chronic. Acute pancreatitis refers to an acute attack in a previously healthy person and symptoms that resolve with the attack. Chronic pancreatitis usually refers to repeated attacks and continued symptoms of exocrine and endocrine insufficiency. Both forms are serious and can lead to complications. In severe cases, bleeding, infection, and permanent tissue damage may occur (National Digestive Diseases Information Clearinghouse [NDDIC], 2014).

Acute pancreatitis is inflammation of the pancreas that occurs suddenly and usually resolves in a few days with treatment. Acute pancreatitis can be a life-threatening illness with severe complications.

Mild forms of acute pancreatitis are known as edematous or interstitial pancreatitis. Mild acute pancreatitis is rarely fatal. These patients usually recover quickly, often without any complications (NDDIC, 2014).

With severe acute pancreatitis (otherwise known as necrotizing or hemorrhagic pancreatitis), patients often suffer serious complications and mortality is high.

Pancreatitis can also be classified according to etiology (cause).

Etiology of Acute Pancreatitis

The most common cause of acute pancreatitis is the presence of gallstones (small, pebble-like substances made of hardened bile) that cause inflammation in the pancreas as they pass through the common bile duct.

Chronic, heavy alcohol use is also a common cause. Acute pancreatitis can occur within hours or as long as two days after consuming alcohol.

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Alcohol Induced Pancreatitis

Alcohol is the second leading cause of pancreatitis and accounts for 35% of the known cases of acute pancreatitis (Cappell, 2008 in Andris, 2013). Alcohol abuse often results in chronic pancreatitis, yet the etiology is not clearly understood.

Although the exact mechanism of how alcohol causes pancreatitis is not known, alcohol induced pancreatitis is thought to be caused primarily by the eventual blocking of the small ductules in the pancreas that drain into the pancreatic duct. Chronic alcohol ingestion leads to the intracellular accumulation of digestive enzymes and their early activation and release from the pancreas. Alcohol also increases the permeability of the small ductules of the pancreas which allows the pancreatic digestive enzymes to reach the pancreatic parenchyma or tissue.

In addition, alcohol increases the protein content of the pancreatic fluid and decreases bicarbonate levels and trypsin inhibitor concentrations. This leads to the formation of protein plugs that block the pancreatic outflow. When the normal pancreatic enzymes are released into these blocked ductules, they eventually begin to “back-up.” This blockage further increases the permeability of the pancreatic ducts, also leading to leakage of the pancreatic enzymes into the pancreatic tissue. Since most of the enzymes trying to pass through the ducts are digestive in nature, the pancreas finds itself being auto-digested by its own enzymes.

Did you know?

Several studies have been conducted to determine the specific amount of alcohol per day that would put a person at high risk of developing pancreatitis. From research findings, it appears that consuming 5 to 8 drinks per day for at least 5 years puts a patient at significant risk (Cappell, 2008 in Andris, 2013).

It has also been shown that smoking poses an additional risk of developing pancreatitis in conjunction with heavy alcohol usage. However, Yadav et al (2009 in Andris, 2013) determined that smoking was a significant risk factor for pancreatitis independent of alcohol use. In this study, however, heavy smokers tended to be heavy drinkers, thus compounding the risk factors for pancreatitis.

Test Yourself

Alcohol abuse is believed to damage the pancreas by:

A. Forming protein plugs that block pancreatic outflow.
B. Increasing the permeability of pancreatic ducts and stimulating auto-digestion.
C. Both of the above.

The correct answer is: C. Both of the above.

Alcohol increases the protein content of the pancreatic fluid and decreases bicarbonate levels and trypsin inhibitor concentrations. This leads to the formation of protein plugs that block the pancreatic outflow. When the normal pancreatic enzymes are released into these blocked ductules, they eventually begin to “back-up.” This blockage further increases the permeability of the pancreatic ducts, also leading to leakage of the pancreatic enzymes into the pancreatic tissue. Since most of the enzymes trying to pass through the ducts are digestive in nature, the pancreas finds itself being auto-

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digested by its own enzymes.

Biliary Induced Pancreatitis

Gallstones, the most frequent cause of acute pancreatitis in women is usually caused by the blockage of the cystic duct or the common bile duct with a stone, particularly at the Sphincter of Oddi, located at the junction of the biliary and pancreatic ducts with the duodenum.

Pancreatic fluid that can no longer escape to the digestive tract becomes trapped in the pancreas. The fluid contains digestive enzymes in an inactive form and inhibitors that block the activation of enzymes on route to the duodenum. If the blockage cannot be resolved quickly, the enzymes accumulate, increasing pancreatic ductile permeability, and eventually overwhelm the pancreatic enzyme inhibitors. Similar to alcohol induced pancreatitis, the pancreas then begins to auto-digest itself, leading to bleeding, necrosis, and abdominal fluid accumulation.

Risk Factors

Risk factors for acute pancreatitis in patients ***under*** the age of 50 years usually include:

- AIDS
- Vasculitis
- Alcohol abuse
- Illicit drug use

Risk factors that usually affect those ***over*** the age of 50 years usually include:

- Biliary tract disease
- Trauma
- Endoscopic retrograde cholangiopancreatography (ERCP)

In men over the age of 50, alcohol is primarily the most common cause of pancreatitis.

For women in the United States, gallstones are the most common cause.

Certain drugs, trauma, and surgery can also precipitate acute pancreatitis.

Risk Factors

Some drugs that can cause pancreatitis include:

- Azathioprine
- Estrogens
- Pentamidine

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- Tetracycline
- Valproic acid
- NSAIDs
- 6-mercaptopurine
- Furosemide
- Sulfonamides
- Thiazide diuretics
- Dideoxyinosine

Test Yourself
The most common cause of acute pancreatitis in women is:
A. AIDS
B. Gallstones
C. Alcohol abuse
The correct answer is: gallstones.
In men over the age of 50, alcohol is primarily the most common cause of pancreatitis. For women in the United States, gallstones are the most common cause.

Other Causes of Pancreatitis

Kourey and Deeba (2007) cite other miscellaneous causes of pancreatitis which include:

- Peptic ulcer disease
- Hypertriglyceridemia: Triglyceride values greater than 1000 mg/U, greatly increases the risk of developing pancreatitis
- Abdominal or cardiopulmonary bypass surgery which may affect the pancreas's blood supply
- Trauma to the abdomen or back causing a sudden compression of the pancreas against the spine
- Pancreatic cancer can lead to pancreatic ductile obstructions
- Viral infections, including mumps, coxsackie virus, cytomegalovirus (CMV), hepatitis, Epstein-Barr virus (EBV), and rubella
- Bacterial infections, such as mycoplasma
- Intestinal parasites, such as Ascaris, which can block the pancreatic ductules
- Scorpion and snake bites

Overview of the Digestive System

Understanding the function, location, and cells that comprise the pancreas aids in understanding the pathology of pancreatitis.

Anatomy of the Pancreas

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The pancreas is an organ located in the mid abdomen in a retroperitoneal position behind the stomach. Anatomically, the pancreas is divided into the head, the body, and the tail. The head lies within the arms of the duodenum, whereas the tail is adjacent to the spleen.

The pancreatic duct passes from the tail through the body and the head and empties into the duodenum at the ampulla of Vater. The sphincter of Oddi allows pancreatic secretions to enter the duodenum where they aid in digestion.

In addition, the common bile duct also enters the duodenum at the ampulla of Vater. Bile produced by the liver is a key component in the digestion and emulsification of fats.

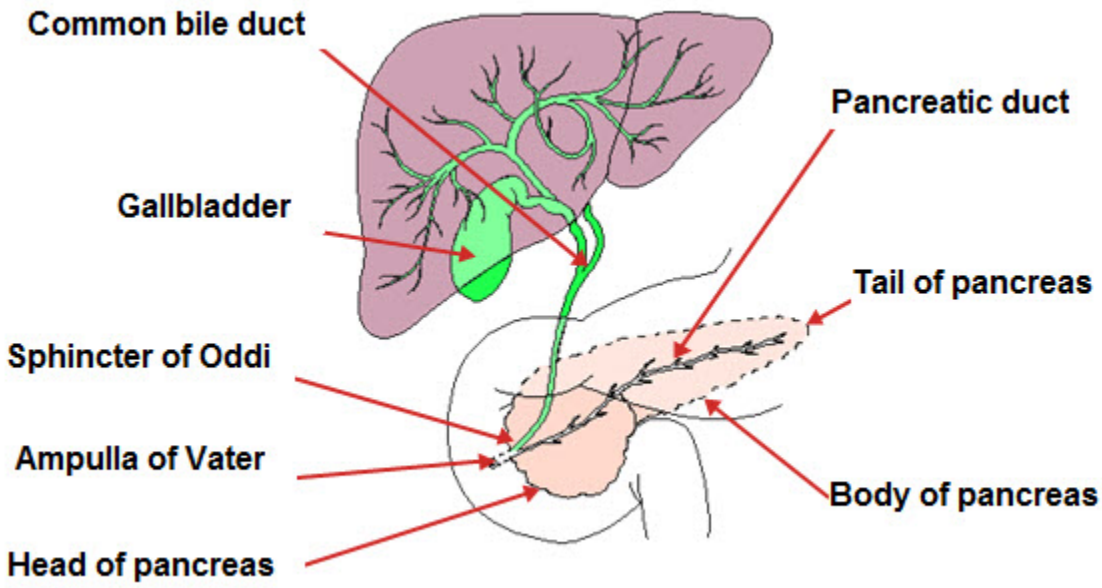

Illustration of the gallbladder and liver attaching to the duodenum via the biliary tree.

Test Yourself

The Sphincter of Oddi:

A. Carries bile from the liver to the duodenum.
B. Controls the release of pancreatic secretions into the duodenum.
C. Passes from the tail through the body and the head and empties into the duodenum at the ampulla of Vater.

The correct answer is: B. Controls the release of pancreatic secretions into the duodenum.
The Common Bile Duct carries bile from the liver to the duodenum. The Sphincter of Oddi controls the release of pancreatic secretions into the duodenum, and the pancreatic duct passes from the tail through the body and the head and empties into the duodenum at the ampulla of Vater.

Physiology of the Pancreas

The pancreas is an unusual gland with both endocrine and exocrine functions. The pancreas is composed of 98% exocrine cells and 2% endocrine cells (Parker, 2004 in Andris, 2013).

Exocrine Functions of the Pancreas

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The acinar cells are responsible for the exocrine function of the pancreas. Acinar cells synthesize enzymes in both the inactive form (eg, trypsinogen) and the active form (eg, amylase and lipase). The enzymes are secreted into the duodenum via the pancreatic duct and are required for fat, protein, and carbohydrate digestion (Hughes, 2004 in Andris, 2013).

The exocrine enzymes of the pancreas include:

- Trypsin
- Chymotrypsin
- Lipase
- Amylase and others

The pancreas secretes approximately 2.5 L of pancreatic juice daily, mainly composed of bicarbonate and enzymes.

Physiology of the Pancreas

Three distinct phases of pancreatic secretion have been identified:

The cephalic phase is characterized by stimulation related to the sight and smell of food and accounts for 10% to 15% of secretion.

The gastric phase is stimulated by the presence of food within the stomach and by gastric distension and also accounts for 10% to 15% of secretion.

The intestinal phase is activated by food entering the duodenum and accounts for the majority of stimulation (70% to 80%).

Physiology of the Pancreas

The endocrine cells of the pancreas are classified into three types: alpha, beta, and delta. As an endocrine organ, the pancreas produces hormones that are released into the bloodstream.

The alpha cells produce glucagon that is used to increase and maintain blood glucose levels, whereas the beta cells produce insulin that reduces blood glucose levels by aiding its entry into cells. Delta cells also function as inhibitory cells to the acinar, alpha, and beta cells by secreting somatostatin, which inhibits the secretion of growth hormone, thyroid stimulating hormone, insulin, glucagon, and various other gastrointestinal hormones.

Endocrine hormones produced by the pancreas are:

- Insulin
- Glucagon
- Vasoactive intestinal polypeptide (VIP)

Test Yourself

The endocrine function of the pancreas ensures that:

A. Promote fat, carbohydrate and protein digestion.

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B. Digestive enzymes are produced by the pancreas.
C. Insulin and glucagon are produced to stabilize blood glucose levels.

The correct answer is: C. Insulin and glucagon are produced to stabilize blood glucose levels.
The exocrine function of the pancreas ensures that digestive enzymes are produced and these enzymes are responsible for the metabolism of fats, proteins and carbohydrates. The endocrine function of the pancreas is to produce the hormones insulin and glucagon to stabilize blood glucose levels.

Pathophysiology of Pancreatitis

The primary pathology of pancreatitis is pancreatic inflammation and parenchymal necrosis. A variety of causative factors for pancreatitis have been identified. The key mechanism appears to be one that causes pancreatic ductal obstruction, ductal hypertension and possible ductal rupture, premature activation of pancreatic enzymes leading to pancreatic autodigestion, persistent inflammation, edema, and possible pancreatic necrosis (Andris, 2013).

This inflammatory process can be significant, leading to life-threatening complications including severe fluid shifts, hemorrhage, sepsis, and multiple system organ failure (Andris, 2013).

Both exocrine and endocrine functions can be affected, presenting implications for treatment in terms of nutrition and glucose management.

Signs and Symptoms of Acute Pancreatitis

Acute pancreatitis is a clinical syndrome consisting of epigastric abdominal pain, often radiating to the back, and this intense and unrelenting pain is often the defining characteristic of acute pancreatitis. The pain is often described as diffuse, dull, and constant. The pain typically begins suddenly and is epigastric in nature, often presenting in the upper abdomen, below the sternum. Often the pain radiates to the back and may improve when the patient lies flat on his back, or curls into the fetal position.

Nausea, vomiting, and diarrhea are all associated symptoms that may or may not be present. Eating typically intensifies the pain, as pancreatic enzymes are stimulated.

Signs and Symptoms of Acute Pancreatitis

Typically patients will seek medical attention after several days of persistent pain. When they arrive to their care provider, they often present with the following signs and symptoms:

- Fever
- Chills
- Swollen, tender abdomen with muscle guarding
- Tachycardia (either from bleeding, fever, and/or pain)
- Dehydration in severe cases, leading to weakness, irritability and confusion
- Hypoglycemia in severe cases when damage to the islet cells of the pancreas has occurred. As a result, diabetes is often a clinical sequel to pancreatitis. Hypoglycemic symptoms include irritability, diaphoresis, nausea, confusion, cool, clammy skin, and syncope.
- Hypocalcemia: present in up to 30% of cases, due to calcium binding to areas of fat necrosis around the pancreas. Symptoms of hypocalcemia may include:

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- Positive Chvostek's sign (spasm of the facial muscles when the facial nerve is tapped)
- Positive Trousseau's sign (flexion of the wrist, hyperextension of the fingers and flexion of the thumb after inflating a blood pressure cuff above the systolic BP for several minutes)
- Numbness and tingling of the extremities
- Tetany
- Seizures
- Fatigue
- Muscle cramps
- Laryngeal and bronchial spasms (Goltzman, 2014)

Test Yourself
A positive Trousseau's sign is:

A. Spasm of the facial muscles when the facial nerve is tapped.
B. Numbness and tingling of the extremities when the patellar is tapped.
C. Flexion of the wrist, hyperextension of the fingers and flexion of the thumb after inflating a blood pressure cuff above the systolic BP for several minutes.

The correct answer is: C. Flexion of the wrist, hyperextension of the fingers and flexion of the thumb after inflating a blood pressure cuff above the systolic BP for several minutes.
A positive Chvostek's sign is spasm of the facial muscles when the facial nerve is tapped. A positive Trousseau's sign is flexion of the wrist, hyperextension of the fingers and flexion of the thumb after inflating a blood pressure cuff above the systolic BP for several minutes. Numbness and tingling of the extremities are not related to a patellar tap.

Diagnosis of Acute Pancreatitis

Acute pancreatitis is frequently misdiagnosed. The diagnosis of acute pancreatitis depends not only on the clinical presentation but also on serologic evidence of abnormal pancreatic function or confirmatory results obtained from a cross-sectional pancreatic imaging study, such as a contrast-enhanced computed tomography (CT) scan (Baillie et al., 2013).

Early diagnosis of pancreatitis is crucial to minimize disease progression. The most important diagnostic tool is the patient assessment. An accurate patient history is needed to assess for risk factors associated with pancreatitis such as gallbladder disease and alcohol consumption.

Eating fatty foods and consumption of alcohol may exacerbate this pain (Despins & Cox, 2005 in Andris, 2013). Pain may also be positional. Usually, an upright position increases the pain level, whereas the fetal position decreases stretching of the pancreas and alleviates pain (Andris, 2013).

The onset of symptoms is usually sudden and accompanied by nausea and vomiting. Bowel sounds are often decreased or absent because of an associated paralytic ileus. Hypovolemia may present with hypotension, tachycardia, and decreased peripheral perfusion. A fever may also be present early in the disease process because of inflammation of the pancreas (Sargent, 2006).

Although patients may present with various symptoms, 95% exhibit a constant epigastric pain (Sargent, 2006). The epigastric pain may radiate to the back or

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chest and usually increases quickly in severity, lasting for an extended period of time.

Laboratory Monitoring

On admission, the following laboratory studies should be conducted:

- Complete blood cell count
- Metabolic panel
- Amylase and lipase levels
- Lipid profile
- Liver function tests

A provisional diagnosis of acute pancreatitis must be confirmed by serological testing. Note that the normal reference ranges provided in this course may vary slightly from other laboratory reference ranges and are intended to be used as a guide only.

The key laboratory results for diagnosis of pancreatitis are elevated amylase and lipase levels.

Amylase

The pancreas produces amylase, which is an enzyme that breaks dietary starch down into sugars (disaccharides and trisaccharides) which are converted by other enzymes to glucose, to supply the body with energy.

In acute pancreatitis, serum amylase levels are typically elevated within 2 hours of symptoms and then quickly decrease by 36 hours. If the patient seeks medical care soon after the symptoms begin, an amylase level of 3 times normal indicates pancreatitis (Despins et al., 2005 in Andris, 2013).

Note that serum amylase is a small molecule that is rapidly and completely cleared by the kidneys. The serum amylase level may return to normal if it is measured more than 24 to 48 hours after onset of symptoms, or it can remain elevated for up to 14 days, but usually return to normal within one week.

A normal amylase level is between 60-80 units/L.

Did you know?

An elevated serum amylase level can be triggered by numerous non-pancreatic causes, including perforated duodenal ulcer, ruptured ectopic pregnancy, and small bowel obstruction.

Lipase

Lipase is an enzyme that helps to digest fats by breaking down triglycerides into diglycerides and fatty

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acids.

In acute pancreatitis, the serum lipase level rises about 4 to 8 hours after symptom onset, and peaks at around 24 hours. Lipase levels then continue to be elevated up to 14 days after initial symptoms (Parker, 2004 in Andris, 2013).

A lipase level 3 times normal is needed to be diagnostic for acute pancreatitis. A longer period of elevation of serum lipase levels makes it a more accurate diagnostic tool in pancreatitis than serum amylase levels. Therefore, interpretation of amylase and lipase levels is best made within the context of the duration of symptoms. Many clinicians have now stopped relying on serum amylase levels and instead favor serum lipase estimations for detecting acute pancreatitis. (Baillie et al., 2013).

A normal lipase level is between 0-160 units/L.

Did you know?

Serum lipase has high specificity, but low sensitivity in diagnosing pancreatitis. About 85% of the time, when lipase levels are elevated, acute pancreatitis is present.

Hepatic Function Studies

Hepatic function studies, particularly Aspartate Transaminase (AST) and Alanine Transaminase (ALT), may be elevated in patients with pancreatitis caused by chronic alcohol exposure or cholelithiasis with obstruction.

Since many conditions can cause elevations in these levels, they are not particularly reliable in diagnosing pancreatitis. However, they are useful in evaluating the extent of the disease process.

Normal AST Levels are between 12-51 IU/L in males and 12 – 51 IU/L in females.

Normal ALT levels are between 21-70 IU/L in males and 9-61 IU/L in females.

Hematology

WBCs

Leukocytes, or white blood cells, protect the body from bacteria and infection. The total WBC count increases in response to infection or trauma and is an indicator of how well the body is fighting infection.

In pancreatitis, WBC evaluation is useful in determining the extent of the disease process, and will mostly likely be elevated if infection or abscess is present.

A normal WBC is between 4,500 - 11,000 mm3.

Hematology

RBCs (Erythrocytes), Hemoglobin (Hgb) & Hematocrit (Hct)

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Red blood cells and hemoglobin go hand in hand. One unit of packed red blood cells equals one full number increase in hemoglobin value. For example, if a patient's hemoglobin is 7.0 g/dL, and he receives one unit of packed red blood cells, his hemoglobin should come up to 8.0 g/dL, providing that there is no internal bleeding.

Hematocrit is an expression of the total percentage of blood volume that is composed of red blood cells, and is also known as the packed cell volume of blood. Often, hematocrit is monitored to determine the extent of hypovolemia. In hemorrhagic pancreatitis, this value can predict extent of blood loss.

Normal erythrocyte levels are between 4.3 -5.9 x106 /mcL in males and 3.5 – 5 x 106/mcL in females.

Normal hemoglobin (Hgb) is between 13.8 – 17.5 g/dL in males and 12.1 – 15.3 g/dL in females.

Normal hematocrit (Hct) levels are between 42 – 56% in males and 36-48% in females.

Test Yourself
In hemorrhagic pancreatitis, hematocrit levels can be used to determine the extent of hypovolemia and predict the extent of blood loss.

A. True
B. False

The correct answer is: A. True.
Hematocrit is monitored to determine the extent of hypovolemia. In hemorrhagic pancreatitis, this value can predict extent of blood loss.

Chemistries

Electrolyte abnormalities seen in pancreatitis are often associated with dehydration, prolonged vomiting, and calcium deposits in pancreatic fat.

Serum potassium and BUN elevations may suggest hypovolemia and should be monitored and treated with fluid resuscitation and electrolyte replacements if indicated.

One particular electrolyte, calcium, is very important in monitoring the progress of the disease process.

Normal calcium value is 8.5-10.1 g/dL for patients who have a normal serum albumin (3.3 -5.2 g/dL).

For patients who have a low serum albumin, the calcium level must be corrected based upon their serum albumin concentration.

The following formula can be used to determine the corrected calcium in patients with hypoalbuminemia:

$Ca_{corrected}$ = [(4.0-albumin) X 0.8 mg/dL] + $Ca_{uncorrected}$

For example, for a patient with a serum Ca = 7.3 mg/dL (low), and a serum albumin of 2.7 g/dL:

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$Ca_{corrected}$ = [(4.0-2.7g/dL) X 0.8 mg/dL] + 7.3 mg/dL
$Ca_{corrected}$ = 8.34 mg/dL

The patient's corrected calcium value is still below normal, but not as deficient as the uncorrected calcium of 7.3 mg/dL.

Imaging Studies

X-rays

Standard x-rays are of limited use in diagnosing acute pancreatitis. Abnormal findings on abdominal x-ray usually suggest chronic pancreatitis and may include findings of a gas-filled duodenum, secondary to obstruction.

Imaging Studies

MRI and CT Scanning

Magnetic resonance imaging (MRI) of the biliary tree and pancreas has developed rapidly over the past several years, but its role in detecting the complications and sequel of acute pancreatitis has yet to be defined (Baillie et al., 2013). The technique could serve as an alternative to abdominal CT scanning for patients who have renal impairment or a history of severe reaction to intravenous contrast media. Unfortunately, the sickest patients with acute pancreatitis are the least likely to undergo MRI, because they are usually treated in an intensive care unit (ICU), attached to mechanical ventilators and other medical machines. In addition, the sensitivity of MRI in detecting pancreatic necrosis has yet to be determined (Baillie et al., 2013).

These drawbacks limit the utility of MRI during the acute phase of pancreatitis. However, the test can help locate the causes of unexplained, recurrent pancreatitis, including pancreatic ductal disruption, fluid collections, and occult tumors (Baillie et al., 2013).

Contrast-enhanced abdominal CT scanning is a sensitive and specific test for acute pancreatitis and pancreatic necrosis.

Imaging Studies

Abdominal Ultrasound

An ultrasound of the abdomen is often indicated in pancreatitis to evaluate biliary causes of pancreatitis, and to assess for pancreatic pseudocysts. Ultrasonography may provide evidence of a dilated common bile duct, presence of gallstones, and swelling of the pancreas; however, it is often difficult to visualize the pancreas itself with this study, and further investigation is often needed. An advantage is that an ultrasound is inexpensive and non-invasive.

Imaging Studies

ERCP

Endoscopic Retrograde Cholangiopancreatography (ERCP) is utilized most often in patients with severe pancreatitis, where a biliary cause is suspected. ERCP is a procedure used to identify stones,

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tumors, or narrowing in the bile ducts. The procedure is done through an endoscope, inserted through the mouth, and passed through the esophagus and stomach until it reaches the duodenum. A catheter is then passed through the endoscope and inserted into the ducts of the pancreas and gallbladder. Dye is injected and x-rays are taken, to visualize stones, tumors, and any areas that have become narrowed (Medline, 2014).

In addition, instruments can be placed through the endoscope and into the ducts to open the entry of the ducts into the bowel, stretch out narrow segments, extract tissue samples and drain blocked areas.

Assessing Severity of Acute Pancreatitis

Three scoring systems are used to determine the severity of the disease, namely the Ranson's Criteria, APACHE II and BISAP score.

Scoring Systems

Ranson's Criteria

The Ranson criteria are a set of clinical predictions used to assess the severity of acute pancreatitis. They were introduced in 1974, and have been in use for more than thirty years. Ranson's criteria assesses 11 risk factors to determine the risk of mortality (death) from acute pancreatitis.

Age, selected laboratory values, arterial blood gas results, and fluid balance are used to determine severity.

Although the Ranson criteria have been proven reliable, one drawback to this scoring tool is that it is completed at onset of symptoms and 48 hours later. This time lag prolongs assessment of severe pancreatitis.

Typically, the Ranson criteria assessment tool is used in the first 48 hours of symptom onset. A patient is given 1 point for each of a possible 9 factors present. A score of 3 or more is an indication of severe pancreatitis.

A benefit of using this assessment tool is that it allows for immediate severity screening, with only age and laboratory testing needed for completion.

The modified Glasgow criteria, in contrast with the Ranson criteria, can be scored daily.

Ranson's Criteria

The parameters used in Ranson's criteria **at admission** include:

- Patient's age (patients over the age of 55 years have an increased risk)
- Increased white blood cell count (>16000 cells/mm3)
- Elevated blood glucose (>200 mg/dL)
- Elevated serum AST levels (>250 IU/L) and elevated serum LDH levels (>350 IU/L)

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The parameters used in Ranson's criteria **within 48 hours** include:

- Low serum calcium < 2.0 mmol/L (< 8.0 mg/dL)
- Fall in hematocrit (> 10%)
- Hypoxemia (PO2 < 60 mmHg)
- Increased BUN (5 mg/dL or greater) after IV fluid hydration
- Base deficit / negative base excess (> 4 mEq/L)
- Sequestration of fluids (> 6 L)

One point is given for each category that is applicable, with a total of 11 points possible. A score of 3 or more is considered to be a case of severe pancreatitis, and the patient should be closely monitored for the risk of organ failure.

A score of 3 to 5 is related to a 10% to 20% rate of mortality, with a score above 5 increasing mortality risk to more than 50% (Despins, 2005 in Andris, 2013).

The Ranson criteria are applicable to non-gallstone pancreatitis.

The Ranson's criteria requirement for a second assessment at 24 to 48 hours after the first is an indication of how long the systemic complications of acute pancreatitis take to develop.

APACHE II

Since the Ranson's criteria were developed, clinicians have devised other, less complex scoring systems, such as the APACHE II scoring system. This scoring system is currently in use in ICUs, and it provides similar results in predicting disease severity, but is cumbersome and takes longer to provide the desired prediction.

BISAP Score

The Bedside Index for Severity in Acute Pancreatitis (BISAP) score is a relatively new severity assessment tool that evaluates vital signs, blood work, and imaging tests. This tool is relatively easy to score and uses impaired mental status and signs of systemic inflammatory response syndrome as indicators of severe disease (Andris, 2013).

A score of 0 to 5 is obtained, with a score of 3 or more being a significant indicator for mortality, pancreatic necrosis, and organ failure (Singh et al., 2009 in Andris, 2013).

The parameters used in the BISAP Score include:

- Patient's age (patients over the age of 60 have an increased risk)
- Impaired mental status: Glascow Coma Score of <15
- Elevated serum urea nitrogen level (>25 md/dL)
- Evidence of pleural effusion on imaging tests
- Evidence of a systemic inflammatory response syndrome, in which 2 or more of the following criteria are present:

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- Temperature <36^{O} C or >38^{O}C
- Respiratory rate >20 breaths/min or $Paco_2$ <32 mmHg
- Heart Rate >90 beats/min
- White Blood Cell Count <4000 or >12000 / mm3

Other Predictive Criteria

Certain serum markers can also be useful in predicting the severity and outcomes associated with acute pancreatitis. These serum markers include C-reactive protein, interleukin-6, interleukin-8, phospholipase A_2, procalcitonin, and trypsinogen activation peptide (Carroll, Herrick, & Gipson, 2007).

C-Reactive Protein

Any elevation in C-reactive protein is generally seen as a non-specific response to inflammation. It is thought that elevations in C-reactive protein can be used to predict prognosis and severity of pancreatitis.

Within the first 24 to 48 hours of pancreatic disease, high levels of C-reactive protein are associated with pancreatic necrosis and severe disease (Carroll, Herrick, & Gipson, 2007).

Interleukin-6 and Interleukin-8

Interleukins are types of biological response modifiers. These substances function to help improve the body's natural response to infection and disease.

High levels of interleukin-6 and interleukin-8 within 18 to 48 hours and 12 to 24 hours respectively have been identified as early indicators of severe pancreatic disease (Carroll, Herrick, & Gipson, 2007).

Phospholipase A2

Phospholipase A_2 aids in the initial digestion of phospholipid compounds in dietary fat. They require calcium to function normally and become elevated in inflammatory responses, such as pancreatitis.

Elevations in phospholipase A_2 within the first 24 hours is associated with development of pancreatic necrosis and pulmonary failure (Balzthar, 2002 & Carroll, Herrick, & Gipson, 2007).

Procalcitonin

Procalcitonin is the precursor of the hormone calcitonin. Measurements of procalcitonin can be used as an indicator for severe sepsis and correlates accordingly with varying degrees of sepsis. Therefore, this test is utilized to detect degrees of infection with pancreatitis (Marshall, 2003).

Elevations in procalcitonin within 24 to 36 hours allows the early detection of both infection and necrosis (Carroll, Herrick, & Gipson, 2007).

Trypsinogen and Trypsinogen Activation Peptide

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In the mid-1990s, researchers discovered that trypsinogen and trypsinogen activation peptide in the urine was clinically more sensitive and specific in diagnosing acute pancreatitis than serum amylase and lipase. Elevations that occur in trypsinogen activation peptide within a few hours of symptom onset are considered both diagnostic and predictive of the severity of the pancreatitis (Carroll, Herrick, & Gipson, 2007).

Since elevations in serum amylase and lipase can be absent in 20% of patients who truly have pancreatitis, this test can be useful when the clinician is fairly certain that pancreatitis exists, yet amylase and lipase levels are normal or only slightly elevated.

Management of Acute Pancreatitis

The management of acute pancreatitis is aimed at treating both its manifestations and complications. Mild pancreatitis is typically self-limiting. The symptoms usually subside with supportive care including monitoring, pain control, and intravenous fluids. However, moderate or severe pancreatitis requires more extensive monitoring and supportive care.

Pain Control

Acute pancreatitis is intensely painful and unrelenting and requires pharmacological relief. In addition, pain management in acute pancreatitis diminishes further activation of the offending pancreatic enzymes.

Meperidine was most often the drug of choice for managing the pain associated with acute pancreatitis, as it was once believed that morphine caused increased pancreatic obstruction and pain spasms at the Sphincter of Oddi. However, research has shown that all types of opioids (including meperidine) can precipitate increased pain and spasms. In addition, the repeated administration of meperidine can result in the accumulation of its metabolite, normeperidine, which may lead to serotonin syndrome and the possibility of seizures (Forrett, 2006).

Given this information, it is likely that patients may benefit from receiving medications such as buprenorphine (a narcotic analgesic) and transdermal fentanyl, which are very effective in treating pancreatic pain (Holcomb, S. 2007).

Pain relieving narcotics should be administered via frequent, round the clock intravenous injections or by patient-controlled anesthesia (PCA). The patient’s pain should be regularly assessed using a valid and reliable pain intensity rating scale.

Test Yourself

The best way to control pain in acute pancreatitis is through the use of:

A. PCA pumps.
B. Oral opioids.
C. Fentanyl epidurals.

The correct answer is: A. PCA pumps.

Pain relieving narcotics should be administered via frequent, round the clock intravenous injections or by patient-controlled anesthesia (PCA).

Hemodynamic and Electrolyte Stabilization

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Fluid resuscitation for shock is of utmost priority in managing pancreatitis. Crystalloid solutions such as normal saline are often used to replace volume, followed by packed red blood cells as needed, based on the patient's hemoglobin and hematocrit.

Along with fluid resuscitation, electrolyte balance must be restored. Potassium may be low related to persistent vomiting associated with pancreatitis and calcium can be low due to excessive binding with pancreatic fatty acids.

Nutritional Support

Nutritional support is important to aid in healing. Usually total parenteral nutrition or enteral feedings are given within 24 hours of bowel rest.

Recent research supports combining N.P.O (nothing by mouth) status with early enteral feeding via the jejunum, which prevents pancreatic enzyme release. Early enteral feeding, starting 24 to 48 hours after illness onset, enhances immune system functioning and may help prevent complications such as GI atrophy and worsening inflammation (Holcomb, S. 2007).

Enteral feeding is preferable to parenteral feeding because it's more physiologic, less costly, and less likely to cause complications (Holcomb, S. 2007).

Total parenteral nutrition is now reserved for those patients who cannot tolerate enteral feedings (Vege & Chari, 2006).

Maintenance of Pancreatic Rest

Treatment to induce pancreatic rest can involve NPO status and NG (nasogastric) suction. NPO status is often ordered initially so not to stimulate the pancreatic enzymes from being produced and released. Often NJ (nasojejunal) feeding begin within 24 to 48 hours of admission.

The use of NG suction is controversial. Some authorities believe that NG suction will reduce the amount of gastrin leaving the stomach and entering the duodenum, but controlled trials have not supported this. However, many facilities no longer advocate the use of a nasogastric tube with suction as a routine therapeutic measure in acute pancreatitis because it has not been shown to decrease symptoms, mortality or hospital stay (Gardner, 2013). However, a nasogastric tube may be used when the patient has protracted vomiting or if obstruction is seen on the abdominal radiograph.

Complications

Regardless of the cause, the auto-digestion of the pancreas leads to severe inflammation with some very serious sequelae, and the consequences of pancreatitis on the body as a whole can be devastating. Most complications of acute pancreatitis and subsequent deaths occur within two weeks of onset of pain. Secondary pancreatic infection is the most common cause of death in acute pancreatitis, accounting for 70% to 80% of deaths (Gardner, 2013).

When the pancreas is damaged and begins the process of auto-digestion, the digestive enzymes and the body's own cytokines (extracellular hormones that mediate the immune response) begin to leak out of the pancreas into the peritoneal space surrounding the pancreas.

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Initial areas of damage from digestive enzymes and cytokines usually occur to the lining of the peritoneum. Irritation and inflammation to the tissues can lead to pleural effusions and damage to the organs of the abdominal cavity.

Once the digestive enzymes begin to be absorbed by the lymph system and the circulatory system, their toxic effect is unbound. Virtually every capillary that comes into contact with these enzymes can be affected.

Cardiac Complications

Once in the circulatory system, pancreatic enzymes are responsible for the release of myocardial depressant factor. Myocardial depressant factor acts to depress myocardial contractility.

Other factors that lead to severe circulatory problems include the leakage of plasma proteins from damaged endothelial cells, and the release of kinins which lead to vasodilation.

The physiologic responses from pancreatic enzymes in the circulatory system lead to edema formation, abdominal pseudocysts, pulmonary infiltrates and edema.

Management of Cardiac Complications

Drug therapy includes the use of inotropic agents such as digoxin and IV vasopressors such as dopamine and dobutamine.

Systemic Hypoperfusion

Once heart function becomes depressed, hypoperfusion becomes evident, and may be compounded by bleeding and dehydration. Hypoperfusion is evident in all organ systems as evidenced by an altered level of consciousness, oliguria, pallor, delayed capillary refill, and decreased pO2 and pulse oximetry readings.

This net effect of reduced cardiac output and decreased blood flow is sympathetic nervous system activation, and often shock. As the sympathetic nervous system is activated, catecholamines cause shock-like symptoms, such as cold, clammy skin and diaphoresis.

Renal ischemia is another result of hypovolemia, leading to acute tubular necrosis and acute renal failure.

Management of Systemic Hypoperfusion

Infusion of fluids and blood products is extremely important. Monitor intake and output closely and include nasogastric drainage in fluid balance calculations.

Renal complications often arise as a result of hypoperfusion and should be managed proactively to prevent full blown renal failure. Restoring volume to the kidneys is the best way to increase perfusion, and is achieved through fluid and blood administration, and medications that improve cardiac output.

Temporary dialysis is sometimes necessary to remove unwanted toxins that can accumulate during

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acute renal failure. The most critically ill pancreatitis patients do much better with continuous renal replacement therapy (CRRT) instead of the standard one to two hour hemodialysis treatment. Since fluid is removed and filtered slowly, the hemodynamically unstable patient tends to tolerate this type of dialysis much better than conventional dialysis.

Pulmonary Complications

Pulmonary complications of acute pancreatitis include pleural effusions, atelectasis, and Acute Respiratory Distress Syndrome (ARDS). ARDS results from direct lung injury from the circulating pancreatic enzymes in the vascular system. These enzymes damage the lung tissue itself, and can lead to the need for temporary mechanical ventilation.

Pleural effusions and atelectesis are the result of an enlarged abdomen and inflamed diaphragm that does not move as it normally would.

Management of Pulmonary Complications

Ventilatory support is often needed in the most severe forms of pancreatitis. Arterial blood gases are monitored frequently and ventilatory settings adapted to meet the body's demand.

A common complication for many patients following an episode of acute pancreatitis with intubation is weaning from the ventilator. Due to the damage that can occur in the pulmonary tissues, weaning can often be a long and difficult process. Patients can be left with many months of pulmonary rehabilitation to return to pre-pancreatitis lung function post extubation.

Vascular Complications

Disseminated intravascular coagulation (DIC) is another complication of the premature release of pancreatic enzymes into the vasculature.

Trypsin activates the clotting enzymes prothrombin and plasminogen. Once these clotting enzymes are activated, the patient begins to produce microthrombi.

These microthrombi are responsible for CVAs, pulmonary emboli, and acute tubular necrosis. Once the body's clotting factors are depleted, spontaneous hemorrhage can result.

Management of Vascular Complications

DIC is managed by administering whole blood or blood products to normalize the clotting factor level. Platelets and packed red blood cells (PRBC's) are given to replace those lost during hemorrhage. Cryoprecipitate or fresh frozen plasma is given to normalize clotting factor levels (White, L, 2005).

Heparin may be used to prevent the further development of microemboli, but the use of this drug is controversial in DIC, due to the risk of hemorrhage (White, L, 2005).

Infectious Complications

Sepsis is another deadly complication of acute pancreatitis. Bacteria in the gastrointestinal tract tend

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to move into the circulatory system during times of hypoperfusion and reduced bowel motility. These organisms are usually gram negative and often difficult to eradicate.

Other sources of infection stem from pancreatic pseudocysts and pancreatic abscesses that develop from the local effects of enzyme leakage into the pertioneum.

Pseudocysts

Pseudocysts form when the cavity surrounding the outside of the pancreas fills with necrotic products and liquid secretions. This results in a palpable epigastric mass which causes considerable abdominal pain, with nausea and vomiting. Serum amylase levels are usually significantly elevated. The pseudocyst may resolve spontaneously within a few weeks or may perforate, resulting in a peritonitis. Internal drainage is the recommended procedure to avoid further complications.

Pancreatic Abscess

Pancreatic abscess is a large, fluid-filled cavity within the pancreas that result from extensive necrosis of the pancreatic tissue. It causes a palpable abdominal mass and upper abdominal pain. There is usually an accompanying high fever and leukocytosis. A pancreatic abscess requires surgical drainage.

Management of Infectious Complications

In prospective, randomized clinical trials, administration of broad-spectrum antibiotics as prophylaxis against infection in severe, acute pancreatitis has shown no benefit in terms of preventing late infection in pancreatic necrosis.

Alternatively, these reports show that administration of early enteral nutrition with various formulas and supplements, including probiotics, may offer some clinical advantage in terms of morbidity and mortality (Tellado, 2007).

Localized Complications

The local complications of acute pancreatitis are precipitated by the release of pancreatic enzymes into the peritoneal cavity. Once these enzymes are released, their toxic effect is delivered to all tissues they come in contact with.

Pertitonitis, ileus, diaphragmatic inflammation, pleuritis, pleural effusions, and atelectasis are the most common results of this infiltration.

In response to the leakage of these toxic enzymes, edema formation and calcium adhesion to pancreatic fat occurs.

Other local complications may include:

- Fistula formation
- GI hemorrhage
- Pseudoaneurysm

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Management of Localized Complications

Necrosis, pseudocysts, and abscesses are treated primarily via surgery where drainage and debridement of affected areas can be successfully performed. Fine needle aspiration of the suspected infectious tissue is often obtained initially, to confirm the need for surgical debridement.

Pseudoaneurysms may be treated with angioembolism or conventional surgery. Angioembolization is now gaining favor over traditional surgery with ligation of the bleeding vessels. Angioembolization is associated with a 95% success rate and with a significantly lower need for total blood transfusions and length of hospital stay.

Additionally, the combination of angioembolization and later endoscopic drainage of pseudocysts via ERCP is safe and effective in the majority of the cases of pseudoaneurysms in chronic pancreatitis (Udd et al. 2007).

Note!

Embolization is a nonsurgical, minimally invasive procedure that involves the selective occlusion of blood vessels by purposely introducing emboli, to deliberately block a blood vessel.

Other localized complications are treated individually according to type. Ileus is treated with bowel rest and decompression. Fistulization and GI hemorrhage are usually treated surgically or via emergent endoscopic procedures to cauterize site of bleeding.

Case Study One

Introduction

Mr. X is a 45-year-old Caucasian male who presents to the Emergency Department with diffuse abdominal pain, guarding, diaphoresis, and tachycardia.

Presenting Signs and Symptoms

Pain:
The pain began two days ago and has not subsided at all during this time. When asked by the triage nurse to score his abdominal pain on a scale of 1 to 10, the patient states that his pain is about an 8, and is unrelenting. He also reports to the triage nurse that he has never felt pain like this before. It is sharp, stabbing, located in his mid-epigastric area, and radiates to his back. There is significant abdominal guarding present as well.

Nausea and Vomiting:
Mr. X has had both nausea and vomiting over the past 24 hours. His last meal was 4 hours earlier, and consisted only of chicken broth. The nausea is continuous and unrelenting, despite the consumption of Tums, Pepto-Bismol, and over-the-counter Zantac.

Cardiopulmonary symptoms:
The patient is diaphoretic (sweaty) and tachycardic.

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Think About It

What may be causing the diaphoresis and tachycardia in a patient with the above signs and symptoms?

The diaphoresis and tachycardia may be a manifestation of sympathetic nervous system stimulation brought about by severe pain or by dehydration (or possibly bleeding).

Assessment Findings

On physical examination, Mr. X's blood pressure is 152/92. His temperature is 38.1°C which indicates infection and/or acute inflammation. The patient's respirations are elevated at 24 and his pulse oximetry is 96%. Oxygen is started at 4 liters by nasal cannula. Since he is tachypneic, an arterial blood gas (ABG) is drawn.

The results of Mr. X's ABG are as follows:
pH = 7.25
pCO_2 = 53
HCO^3 = 26
pO_2 = 84%

What are these findings diagnostic of?

This is diagnostic of a respiratory acidosis with hypoxia. The patient most certainly has some type of pulmonary complication or primary pulmonary disorder.

His serum carbon dioxide (CO2) is normal at 20.2 mEq/L. This is consistent with his normal bicarbonate level identified in the ABG. It typically takes the kidneys 48 hours to begin compensating for a respiratory problem. Although Mr. X has had pain for a few days, it is unlikely that he has been this tachypneic for the past few days as well. Further history may be needed to confirm this.

On inspection of his abdomen, severe muscle guarding and rigidity is found in the upper abdominal area, with tenderness in the lower quadrants as well. His abdomen is swollen and the area around the umbilicus has a bluish tinge (Cullen's sign), and the flank area appears bruised (Grey Turner's sign).

Cullen's Sign

Cullen's sign is superficial edema and bruising in the subcutaneous fatty tissue around the umbilicus. It is named after Thomas Stephen Cullen, a gynecologist who first described the sign in 1916 (Mookadam & Cikes, 2005).

This sign takes 24 to 48 hours to appear and can predict acute pancreatitis. It may be accompanied by Grey Turner's sign (bruising of the flank), which may then be indicative of pancreatic necrosis with retroperitoneal or intra-abdominal bleeding.

Grey Turner's Sign

In 1919, Turner described a "dirty green" discoloration of the loin associated with acute pancreatitis.

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In acute pancreatitis, Cullen's or Turner's sign occurs in approximately 3% of patients and is associated with a mortality of 37% (Mookadam & Cikes, 2005).

Past Medical and Social History

Mr. X has a history of asthma, which is usually well controlled with Albuterol and Intal inhalers.

He has a positive smoking history of ½ pack per day for 20 years, and drinks socially about once a month with friends when watching sports.

His family history includes asthma, type 2 diabetes, and heart disease, including a father with a two vessel CABG at the age of 58, and a mother with a CVA at age 72.

Admission to the ER

The triage nurse in the Emergency Room is appropriately concerned, given Mr. X's symptoms. She admits him to the cardiac pod to rule out Acute Coronary Syndrome and Acute Abdomen. He is given oxygen, 4 liters nasal cannula and attached to a cardiac monitor which reveals sinus tachycardia. His blood pressure is 152/92, temperature is 38.1°C, and respirations are 24. Pulse oximetry is 96% on 4 L nasal cannula.

Fifteen minutes later, Mr. X's pain has not subsided even though he has chewed two baby aspirin, and has had one sublingual dose of nitroglycerin. He does complain of a new-onset headache at this point (probably from the nitroglycerin).

What orders may the ER nurse anticipate receiving next?

Further laboratory investigations

Laboratory Investigations

The Emergency Department's physician orders serial EKGs and cardiac enzymes. He also orders a CBC with differential, amylase, lipase, AST, ALT, LDH, PT, PTT, BUN, creatinine and electrolytes.

Mr. X's labs come back as follows:

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Patient Lab Values	Flag	Reference Range
WBC = 15,000 mm^3	H	4,500 – 11,000 mm^3
Hemoglobin (Hgb)= 10.2 g/dL	L	Male: 14 -18.5 g/dL
		Female: 12.2 – 16.2 g/dL
Hematocrit (Hct)= 30.6%	L	Male: 42 -56%
		Female: 36-48%
Erythrocytes (RBCs) =3.2	L	Male: 4.3 -5.9 $x10^6$
		Females: 3.5 – 5 $x10^6$
Platelets = 187 000/mm^3		150,000 – 450, 000/mm^3
PT = 11 sec.	L	11.5 – 14.5 seconds
Partial Thromboplastin Time (PTT) = 28 sec		24 – 37 seconds
Amylase = 869 U/L	H	30 – 119 U/L
Lipase = 685 U/L	H	23-300 U/L
AST = 110 U/L	H	Male: 13 – 56 IU/L
		Female: 12- 51 IU/L
ALT = 46 U/L		Male: 21 – 70 IU/L
		Female: 9- 61 IU/L
NA = 146 mEq/L		135 – 146 mmol/L
K = 4.6 mEq/L		3.5 – 6 mmol/L
CL = 94 mEq/L		95 – 110 mmol/L
CO_2 = 40 mEq/L	H	19 – 30 mmol/L
CA = 7.3 mEq/dl	L	8.5 – 10.1 mg/dL
PO_2 =74		65 – 75 mmHg
Mg = 2.1 mg/dl		1.7 – 2.3 mg/dL
BUN = 35 mg/dl	H	Male: 6 – 29 mg/dL
		Female: 6- 27 mg/dL
Fasting Glucose = 156 mg/dl	H	65 – 110 mg/dL
Creatinine = 0.6 mg/dl	L	Male: 0.7 – 1.4 mg/dL
		Female: 0.5- 1.2 mg/dL
Creatinine Phosphokinase (CPK)		Male: 0.7 – 1.4 mg/dL
		Female: 0.5- 1.2 mg/dL

Interpretation of Results

EKG and Cardiac Enzymes

The initial EKG is within normal limits with no ST changes, except for sinus tachycardia.

Cardiac enzymes are within normal limits.

What does this information tell us?

The absence of EKG changes and the fact that the pain is unrelieved by sublingual nitroglycerine indicates that Acute Coronary Syndrome is unlikely to be the correct diagnosis for Mr. X.

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Complete Blood Count (CBC) with Differential

Mr. X's WBC count is high. What does this imply?

An infection is likely.

Mr. X's hemoglobin and hematocrit (H&H) are low and continue to decrease throughout his hospitalization requiring a blood transfusion on day number seven.

What does a low H&H imply?
Mr. X is most likely hypovolemic and there may be some internal bleeding.

Interpretation of Results

Electrolytes and Creatinine

Mr. X's electrolytes are also out of normal range:

- Elevations in his BUN and potassium are most likely due to hemoconcentration.
- Decreased calcium may be due to the disease process of pancreatitis itself. In acute pancreatitis, lipase is activated and causes excessive necrosis of the fatty tissue in the pancreas. This fatty tissue binds circulating calcium, lowering the serum calcium level.

Fasting Blood Glucose

Mr. X's fasting blood glucose level is elevated. If the pancreas is damaged following an attack, the insulin producing function of the pancreas is also lost and diabetes can develop.

Serum Amylase and Lipase

Mr. X's serum amylase level greater than five times the normal level, and his lipase level is more than double the normal amount. These exceptionally high levels are diagnostic of acute pancreatitis, due to the escape of pancreatic enzymes into the blood from the surrounding damaged tissues. An exceptionally high level plus a good history makes the diagnosis quite likely.

Interpretation of Results

Imaging Studies

Mr. X had standard abdominal and chest films that revealed bilateral pleural effusions and a gas filled duodenum.

His abdominal ultrasound revealed two large pseudocysts in his abdomen. An ERCP is scheduled for Mr. X given the high likelihood that his pancreatitis is caused by a biliary stone. Many physicians will often schedule an ERCP to clear the ducts prior to a laparoscopy surgery to remove any pseudocysts, abscesses or necrosis that may be present.

Mr. X's CT scan revealed pancreatic enlargement and edema, consistent with acute pancreatitis.

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Plan of Care

Given Mr. X's elevated amylase and lipase levels and CT findings, a diagnosis of acute pancreatitis is made and he is admitted to a step-down unit.

Mr. X is treated with a Morphine PCA that keeps his pain to a 4/10 on a numeric scale. Mr. X is given 3 liters of normal saline in the emergency department and had a maintenance IV of normal saline at 125 milliliters/hour since he has been NPO. He was also transfused with 2 units of packed red blood cells.

Mr. X was made NPO in the emergency department and will remain NPO until at least 24 hours post his laparoscopic surgery. A nasojejunal tube is placed and continuous drip tube feedings are administered

According to Mr. X's symptoms (diaphoresis, tachycardia, elevated BUN, and elevations in HCT out of proportion with his HGB in week two), Mr. X may certainly be experiencing alterations in his cardiac output. These alterations can be caused by the activation of myocardial depressant factor, decreased blood volume, dehydration, sepsis, or any combination of these. Mr. X is initially stabilized with a 3-liter bolus of normal saline followed by a maintenance drip of 125 milliliters/hour. Eventually, in week two when Mr. X's HGB drops to 8.0 g/dL, he is transfused with 2 units of packed red blood cells. Hemodynamic stability is monitored via an arterial line at this point. Certainly, in more unstable patients a pulmonary artery line (Swan Ganz Catheter) may be necessary.

Mr. X was fortunate. Although his initial blood gases showed respiratory acidosis, ARDS did not ensue. His initial chest films showed bilateral pleural effusions which were treated with chest tubes and decompression of edema and pseudocysts in Mr. X's abdomen. Mr. X's oxygenation status was maintained and supported in the most extreme times of demand with Bi-Pap. Eventually a face mask, then nasal cannula did the trick. Upon discharge, Mr. X was weak and easily fatigued, but outpatient rehabilitation was initiated to rebuild his pre-pancreatitis stamina.

Plan of Care

Case Study One Conclusion
Mr. X's renal function was preserved via the direct administration of fluids and blood products which keep blood flowing to his kidneys. He never experienced acute tubular necrosis or renal ischemia to any lasting degree. If this was the case, CRRT would have been a safe option to excrete wastes while his kidneys were healing.

Fortunately, Mr. X only experienced surgery once to drain two large pseudocysts and relieve peri-pancreatic edema causing his pleural effusions. Abscess and infection did not follow, possibly due to the initiation of early enteral feeding via a J-tube with known, specific immunomodulating nutrients.

Case Study Two

Introduction

Mrs. Y, a 70-year-old female presents to the ED with nausea, vomiting and severe left upper quadrant and epigastric pain. She describes the pain as sharp and radiating towards her back. The pain began about 24 hours ago and is increasing in intensity. Her medical history is unremarkable, with the exception of her smoking habit of 2 packs per day for the past 20 years.

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Presenting Signs and Symptoms

On observation, her vital signs are as follows:

BP: 110/70 mmHg

HR: 100 bpm

RR: 36 breaths/min

Temp: 101^{O}F

Presenting signs and symptoms include:

- Slight discoloration of abdomen / flank with abdominal distention, tenderness and muscle guarding on the left.

Laboratory Investigations

A large bore IV line is started and blood drawn for analysis. After her lab work confirms an elevated serum amylase / lipase with a hypocalcemia, leukocytosis and hyperlipidemia, diagnostic imaging is ordered.

An abnormal CT scan and ERCP confirm the diagnosis of acute pancreatitis.

Plan of Care

Mrs. Y is diagnosed with acute pancreatitis and a nasogastric tube is inserted for abdominal decompression. She is admitted to a Medical-surgical Unit.

On admission to the MS Unit, what is her priority of care?

- Pain relief
- Frequent position changes: side-lying with HOB elevated 45 degrees, and knees drawn up to abdomen
- Nasogastric tube care and good oral and nasal hygiene
- Observation for signs of infection, paralytic ileus, renal failure or mental status changes
- Fluid replacement and maintenance
- Control of pancreatic exocrine and endocrine insufficiency

What labs are the most important to monitor in acute pancreatitis?

- Monitor for signs of hypocalcemia (tetany, numbness around lips / fingers, positive Chvostek or Trousseau sign) and hypomagnesemia

Management and Patient Education

Case Study Two Conclusion

Mrs. Y receives supportive care, including IV fluid replacement, pain management and close monitoring of her labs. She is kept nil by mouth for the first 72 hours, and maintained on intravenous

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hydration and nasogastric suction until she is no longer having significant abdominal pain, tenderness, nausea, or vomiting. On day 4 the nasogastric tube is removed and on day 5, oral feedings are initiated. Fortunately, Mrs. Y does not suffer from any cardiac, pulmonary, vascular or infectious complications and on day 8 of her hospital stay, the nurse is able to prepare her for discharge.

What patient education should be delivered?

The focus of patient education should be chronic health maintenance.

Items to discuss include:

- Healthy nutrition (high carbohydrate, low fat diet and avoidance of alcohol and caffeine)
- Importance of smoking cessation
- Medication adherence

Conclusion

In conclusion, acute pancreatitis can be either mild or severe, with severe cases often becoming deadly.

As a healthcare professional, it is important to understand the pathophysiology related to acute pancreatitis in order to develop an effective plan of care.

Many laboratory and imaging studies can assist in determining a diagnosis of acute pancreatitis as well as the extent of pancreatic complications.

It is our duty as healthcare professionals to understand these tests and their usefulness in this setting so that correct and successful management of this sometimes fatal disease can occur.

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