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Acute Pancreatitis

Noah Prebish
Otterbein University, prebish1@otterbein.edu

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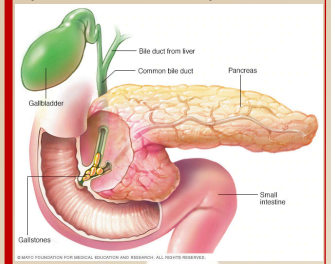
Noah Prebish BSN, RN, CCRN, SRNA
Otterbein University, Westerville, Ohio

Introduction

- Acute pancreatitis (AP) is a medical disorder that involves inflammation of the pancreas.
- There are 15 cases of acute pancreatitis for every 100,000 Americans. Only 20% of cases require hospitalization.
- Risk Factors for acute pancreatitis include: smoking, alcoholism, cholelithiasis, abdominal trauma, peptic ulcers, certain drugs and genetic factors.

(Mccance & Huether, 2019)



(Mayo Clinic Staff, n.d.)

Given that such few cases of AP require hospitalization, the general public is usually unaware of how devastating severe AP can be. I was personally unaware of the severity of acute pancreatitis until I began working as a registered nurse in the surgical intensive care unit (SICU). Once I began caring for AP patients, I quickly saw the numerous setbacks, long road to recovery and sometimes mortality associated with this disorder.

Over 25% of patients with severe AP end up staying in intensive care for over two weeks (Portelli & Jones, 2017). Long SICU stays are usually due to the acute respiratory distress seen in patients with AP, eventually requiring the need for a tracheostomy and long term ventilatory support. In addition, patients that progress to necrotizing pancreatitis have 72% chance of being readmitted an average of 2.3 times after their original discharge from the hospital (Maatman et al., 2019). My goal in choosing this poster topic is to provide information on the pathophysiology of AP and to increase awareness on how serious acute pancreatitis can be.

Signs and Symptoms

Many of the signs and symptoms of acute pancreatitis associated with the inflammatory effects AP has on the pancreas. Signs and symptoms of AP include the following:

- Mild to severe acute pain that starts in the upper abdomen and sometimes radiates to the back.
- Fever
- Nausea and vomiting
- Tachycardia
- Abdominal bloating and tenderness

(Mccance & Huether, 2019)

Diagnostic Testing

In order to diagnose acute pancreatitis patients must meet at least two of the following criteria:

- Abdominal pain that may or may not radiate to the back
- Serum amylase or serum lipase are three or more times normal limits
- Characteristics of acute pancreatitis as noted on CT, MRI, or ultrasound

(Garber et al., 2018)

Laboratory findings

Laboratory diagnosis of AP involves the measurement of serum amylase or serum lipase levels. Results are considered indicative of AP when serum amylase or serum lipase are three or more times the normal limits. There are several emerging laboratory tests that may be early predictors of the severity and potential mortality of AP. Elevated serum laboratory levels such as red cell distribution width (RDW), serum creatinine, albumin, and serum glucose, have been shown be an early predictor of mortality from AP (Hassan et al., 2018). In some Cases, laboratory tests are not a reliable diagnostic tool for AP and require the use of noninvasive imaging.

Imaging

There are three different types of imaging used to diagnose acute pancreatitis. Those methods include:

- Transabdominal ultrasound
- Magnetic resonance cholangiopancreatography (MRCP)
- Computerized tomography (CT)

(The National Pancreas Foundation, n.d.)

The severity of AP is be classified using the Balthazar score featured in table 1 below.

- A score of 0-3 indicates mild acute pancreatitis
- A score of 4-6 indicates moderate acute pancreatitis
- A score of 7-10 indicates severe acute pancreatitis

(Taydas et al., 2017)

Table 1. Components of the CT severity index.

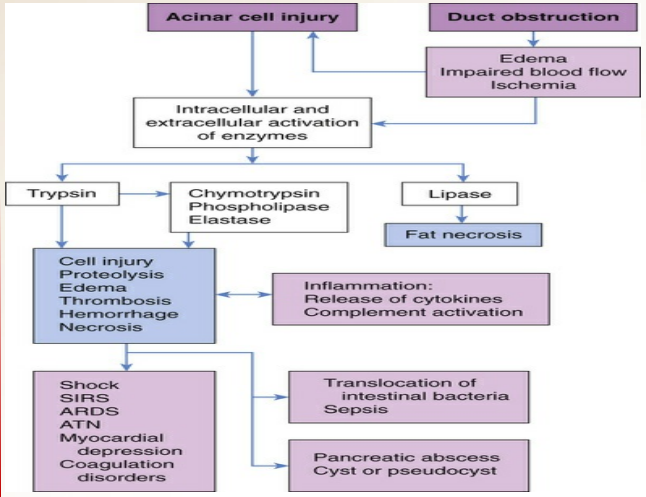
Pancreatic inflammation	
Normal pancreas	0
Focal or diffuse enlargement of the pancreas	1
Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2
Single, ill-defined fluid collection or phlegmon	3
Two or more poorly defined collections or presence of gas in or adjacent to the pancreas	4
Pancreatic necrosis	
None	0
Less than or equal to 30%	2
Greater than 30 and less than, or equal to, 50%	4
Greater than 50%	6

(Taydas et al., 2017)

Pathophysiology

The most common cause of AP is due to an obstruction that forms in the bile and/or pancreatic ducts. When an obstruction occurs in theses ducts, pancreatic digestive enzymes begin to back up within the pancreas. The backup of pancreatic enzymes within the pancreas causes additional enzymes to be released within pancreatic acinar cells. When the enzymes trypsin, chymotrypsin, lipase and elastase are released within acinar cells, autodigestion of the pancreatic cells and tissues begins to occur. Destruction of pancreatic cells and tissues leads to inflammation of the pancreas and the release of cytokines. Release of cytokines leads to the activation of the systemic immune responses commonly associated with severe AP (Mccance & Huether, 2019).

Another common cause of AP is chronic alcohol abuse. When pancreatic acinar cells metabolize excessive amounts of ethanol it leads to the development of toxic metabolites. The build-up of these toxic metabolites eventually lead to acinar cell injury. Much like obstructive pathology, once acinar cell injury occurs, pancreatic enzymes are released causing an inflammatory response. The inflammatory response causes autodigestion of the pancreatic tissues leading to the release of cytokines and the systemic inflammatory response (Mccance & Huether, 2019).



(Mccance & Huether, 2019)

Systemic Response

The destruction acinar cells causes the release of cytokines and vasoactive peptides into systemic circulation. Cytokines and vasoactive peptides lead to the development of systemic inflammatory response syndrome (SIRS) (Mccance & Huether, 2019). The SIRS response can cause vessel wall damage, hypotension and shock. The inflammatory response seen with severe AP can also lead to the development of acute respiratory distress syndrome (ADRS), often requiring the need for mechanical ventilation (Mccance & Huether, 2019).

The inflammation and edema caused by the autodigestion of the pancreas can also lead to a condition known as abdominal compartment syndrome (ACS). ACS is the result of severely increased intraabdominal pressures. Severely increased intraabdominal pressures leads to decreased lung volumes, reduction of cardiac preload, acute renal failure and ischemia to abdominal organs due to hypoperfusion (Bezmarevic, 2018).

Implications for Nursing Care

- Assessment of hemodynamic status - AP patients are at risk for hemodynamic instability as a result of the SIRS response commonly seen with AP. Frequent vital signs must be obtained and monitored for signs of hypotension and tachycardia.
- Management of symptoms – Patients with AP often present with severe abdominal pain. Nurses must be diligent with their pain assessment techniques and administer PRN pain medications when warranted (Johnstone, 2018).
- Intravenous (IV) fluid administration – Adequate fluid administration is a hallmark of AP management, especially in patients with hemodynamic instability. Nurses need to maintain adequate IV access and monitor for signs of hypo and hypervolemia (Johnstone, 2018).
- Glycemic control- Patients with AP have could become hyperglycemic due to the inability of the damaged pancreas to produce insulin. Nurses must be diligent in checking patient's glucose level throughout their hospital stay. Patients with uncontrolled hyperglycemia may require the administration of a continuous insulin infusion (Johnstone, 2018).
- Assessment for adnominal compartment syndrome – AP can cause severe abdominal bloating and tenderness. Nurses must frequently assess their patient's abdomen and report any rapidly increasing abdominal bloating to the attending provider.
- Management and assessment of drains – Patients that develop necrotizing AP often require the placement of abdominal drains to remove excess fluid build-up around the pancreas. Nurses must frequently monitor the amount and characteristics of the drainage in the collection canisters. Purulent drainage in the cannister may be indicative of the development of an infection. Lack of drainage may be indicative of a clogged drain. Clogged drains can lead to the build-up of fluid in the abdominal compartment and potentially ACS.
- Patient and family education – Nurses have a major role in proving patients and their families with education on what to expect when diagnosed with AP. Long stays in the intensive care unit and frequent need for surgical intervention can have a demoralizing mental effect on patients and their family members. Nurses need to inform patients and their families of the potential complications that can occur with AP.

Conclusion

Although rare, severe acute pancreatitis can have devastating clinical effects on patients. The intense systemic pathophysiologic response seen with severe AP can quickly lead to patient mortality if providers are not diligent in the diagnosis and management of AP. Research efforts are continually being made to identify ways to quickly identify and a treat pancreatitis in its early stages before it transitions to severe pancreatitis. I hope this poster helps educate the general public on the pathophysiology of AP and how truly destructive this disorder can be.

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