Devastating Pancreatitis: Getting Back to Basics

By Brenda J. Lane, RN, BScN, Dip Ad Ed, MN, CMSN(C)

People with pancreatitis are admitted to medical and surgical units, usually for four to five days for bowel rest with NPO and pain management orders. So why is it that 10-30% of patients develop severe pancreatitis and succumb to the devastating complications of hypovolemic shock, sepsis, and fatality? Let's take a look at a typical patient.

Picture this:

A slight woman, age 44 years, coming back from a week's vacation in Hawaii, presents to the emergency department at 0930 hours in acute epigastric pain radiating to the back. She is hunched forward with slight symmetrical abdominal distention. Pain is 8/10 and there is no nausea or vomiting. Her past medical history includes four previous admissions with pancreatitis and ETOH use.

In the emergency department she receives an IV bolus of 2 litres 0.9%NaCl, followed by a rate of 150ml/hr. Her vital signs are respectable with BP 126/80; HR 82. She is transferred to a medical unit at 1730 hours with a diagnosis of acute pancreatitis and nursing report indicates she had not voided since admission.

At 2130 hours, she voids 150 ml and she has a BP 94/74, HR 100, with complaints of dizziness, feeling faint and being very restless. Analgesics are administered Q3h for pain as the patient paces throughout the night and is up to the bathroom attempting to void. At 0230 hours the nurse performs a straight in and out catheterization for 30ml dark, concentrated urine. At 0630 hours the patient is found unresponsive with an unattainable BP. She is transferred to the ICU and suffers a cardiac arrest at 0730 hours, twenty-two hours after admission. Following aggressive resuscitative measures in ICU she succumbs to death at 1800 hours, 32 hours since admission.

What went wrong and what timely assessments and interventions could be implemented?

In reviewing the anatomy, recall the pancreas secretes digestive enzymes (trypsin, amylase, and lipase) into the duodenum upon ingestion of food. Patients with pancreatitis are NPO so that the pancreas is not stimulated to secrete enzymes. The gallbladder secretes bile into the duodenum and if gallstones (cholelithiasis) develop they can be released and block the cystic or the common bile duct (choledocholithiasis). Biliary tract disease can lead to pancreatic duct obstruction due to cholelithiasis with the accumulation and activation of pancreatic enzymes, which can cause auto digestion of the pancreas. The inflammatory process causes edema and necrosis. Additionally, it is believed that bile acids reflux through the opened sphincter of Oddi into the pancreatic duct also leading to inflammation and auto digestion. The two most common causes of pancreatitis are biliary tract disease

and chronic alcohol use, which account for 80% of pancreatitis. It is theorized the acinar cells metabolize ethanol and the toxic metabolites injure the acinar cells in the pancreas, which then activates enzyme release leading to auto digestion. Another theory suggests alcohol increases the production of digestive enzymes. 70% of chronic pancreatitis cases are associated with alcohol overuse; however, only 5-10% of people who overuse alcohol develop pancreatitis. Other causes can be trauma, steroids, mumps or infections, autoimmune (such as SLE), spider bites, hyperlipidemia, ERCP, or medications (corticosteroids, thiazides, azathioprine).

Predicting Severity

To predict severity, Ranson's Criteria, APACHE, or the Imrie scoring system may be used upon admission and throughout admission. Regardless of which system is used, consideration is given to elevated WBC (due to inflammatory process); hyperglycemia (due to destruction of pancreas and disruption with insulin production); elevated liver enzymes (AST, LDH); elevated pancreatic enzymes (amylase, lipase); and elevated C-reactive protein. Amylase is most accurate when at least twice the upper limit of normal; whereas, lipase has an increased sensitivity in alcohol-induced pancreatitis and is more specific than amylase. C-reactive protein is a late marker and high levels are associated with pancreatic necrosis. Elevation of Interleukins-6 and 8 are early indication of severity. Hypocalcemia occurs when calcium combines with fatty acid deposits, and although it is not well understood, it is an indication of severity. Severe pancreatitis can lead to hemorrhage as the pancreatic enzyme elastase breaks down the elastic fibers of the blood vessels. This can lead to a drop in hemoglobin and hematocrit. Hypovolemic shock is of major concern because of inadequate intravenous fluids, leading to decreasing circulating volume and the kidneys become hypoperfused causing prerenal failure evidenced by elevation of BUN. With decreased perfusion to organs the PaO_2 falls and patients become acidotic with elevation in base deficits. The greater the number of these indicators, the higher prediction of severity, leading to mortality.

Understanding Inflammation

Pancreatitis is an inflammatory process, which causes the release of mediators like histamine and bradykinin, increasing vascular permeability; this leads to fluid shifts from the vascular bed into surrounding tissues causing edema. Massive fluid shifts (third spacing) and inadequate fluid volume replacement can lead to sequestration of fluid. The sequestration of fluid greater than 6 liters within 48 hours is an indication of severity. In addition, patients may develop pseudocysts because the exudate that develops with the inflammatory process becomes encapsulated by granulation tissue. These pseudocysts can then rupture leading to peritonitis and sepsis.

Returning to the case study, this female had previous admissions with pancreatitis with weight loss, but did not have diabetes mellitus, both of which can occur with chronic pancreatitis. She had an ultrasound in the emergency department, which showed 3,500 ml of free fluid sequestered around her pancreas. In the emergency

department she had 3,800 ml of intravenous fluid without voiding. When she did void at 2130 hours the volume was only 150 ml.

At this point she was already in irreversible hypovolemic shock with output less than the 30 ml/hr of progressive shock (0.5ml X 60 kg (her wt) =30 ml/hr). Her oliguria was secondary to the compensatory mechanism of the renin-angiotensin-aldosterone system, which retains sodium and water to maintain BP. However, as this mechanism fails due to lack of fluid volume replacement, the BP will start to fall. She was symptomatic with tachycardia indicating the compensatory mechanism of increased heart rate to compensate for decreased stroke volume due to decreased circulating blood volume (cardiac output = stroke volume X heart rate). She complained of dizziness and feeling faint due to the hypovolemia, and her restlessness was a sign of hypoxemia. In addition her BP was down to 94/74 indicating hypovolemia.

Getting Back to Basics

Nurses can monitor for the early indicators of hypovolemia, by looking for tachycardia, concentrated urine, and oliguria. Additionally, symptoms of dizziness, faintness, and restlessness are early signs of hypovolemia and hypoxemia. Instructing the patient to save the urine for measurement is a key intervention. Urinary catheterization should be considered if the patient has not voided within 6 to 8 hours. Anticipate intravenous rates at 250 ml/hr or higher with boluses until urinary volume is adequate.

In summary, pancreatitis is an inflammatory process with massive fluid shifts that may result in hypovolemic shock, which can then lead to multiple organ dysfunction, sepsis, and death. Nurses need to assess frequently, attentively and report regularly so that aggressive fluid resuscitation may be provided.

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