**CLINICAL PRACTICE DOCUMENT** 

PLEASE NOTE: UNDER REVIEW

D-00-07-30245

### **Acute Pancreatitis**

## **Site Applicability**

**VGH** 

#### **Practice Level**

RN, LPN

### **Need to Know**

Acute pancreatitis (AP) results from pancreatic inflammation and may manifest as a mild transient illness or a severe and rapidly fatal disease. This inflammation process can compromise direct pancreatic tissue or remote organ systems. Auto digestion is a leading factor contributing to this tissue injury and is initiated when activated digestive enzymes are unable to be released out of the pancreas because of reflux, obstruction, spasm or inflammation. The most common causes are alcoholism and gall stones. Other causes include biliary tract disease, pancreatic cancer, trauma, hypercalcemia, hyperlipidemia, drugs, tumour, ERCP, bacterial or viral infection. In mild or early stages of acute pancreatitis there is interstitial edema and necrosis of pancreatic fat. Severe acute pancreatitis often progresses to organ failure or complications such as infection, necrosis, abscesses, pseudocysts, fistulas or hemorrhage.

Signs and symptoms will vary depending on the severity and stage of the disease process, age and other associated illnesses. Pain is the only universal symptom that usually involves an acute onset of continuous and severe epigastric pain. It is often poorly localized and is generally described as 'boring through' to the back and radiating up between the shoulder blades. It is usually worse in the supine position and may be somewhat relieved by sitting and leaning forward. The pain rapidly intensifies, often within an hour and can last for several hours or even days. Nausea and vomiting are commonly experienced along with diaphoresis, fever and tachycardia. Abdominal distension and tenderness are common along with decreased or absent bowel sounds. Other complications include renal failure, respiratory failure and acute diabetes.

Serum amylase, lipase, CBC, lytes, LFTs and blood glucose are afew tests which assist with diagnosis. Abdominal ultrasound can be useful especially if gall stones are present. CT scan is the 'gold standard' for diagnosis.

Interstitial (or edematous) pancreatitis is the process of digestive enzymes escaping into surrounding tissues causing sterile inflammation and localized edema. Treatment is conservative. In most patients the disease is self-limiting. Supportive care includes intravenous fluids; pain management; maintenance of cardiovascular system; observation and management of possible complications; and antibiotics (generally used in severe episodes of pancreatitis). Nutritional status is monitored closely. In some cases nutritional support is required. Refer to <a href="Nutritional Care of Patients with Acute Pancreatitis">Nutritional Care of Patients with Acute Pancreatitis</a>.

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# PROCEDURE / PROTOCOL / PRACTICE GUIDELINE:

Problems	Interventions
Pain	<ul> <li>position semi to high fowler's (pain may worsen when supine; try having the pt flex their knees towards their chest as this may decrease the pain)</li> <li>Parenteral Non Steroidal Anti-inflammatory Drugs such as Ketorolac and diclofenac were found to be at least as effective as parenteral opioids and more effective than hyoscine in providing analgesia for biliary colic and may also prevent progression to colecystitis<sup>1</sup></li> <li>Therefore NSAIDs ought to be considered in first line options for pain control for suspected 'ductal spasm'<sup>1</sup></li> <li>ALL opioids increase Sphincter of Oddi tone and bile duct pressures (not just morphine) - there are NO studies comparing opioids in the treatment of pain associated with biliary spasm or acute pancreatitis). <sup>1</sup></li> <li>assess signs &amp; symptoms of peritonitis (swelling &amp; tenderness in the abdomen; fever &amp; chills; nausea &amp; vomiting; limited</li> <li>urine output; inability to pass gas or stool) &amp; abdominal hemorrhage</li> </ul>
Impaired oxygenation	<ul> <li>monitor for signs &amp; symptoms of pulmonary complications, including pleural effusion, atelectasis &amp; acute respiratory distress</li> <li>syndrome; monitor vital signs closely</li> <li>assess for adequate ventilation &amp; oxygenation (SpO2) and administer oxygen as ordered/prn</li> <li>manage abdominal pain</li> <li>monitor acid base balance/ABG's as ordered</li> <li>encourage DB&amp;C as tolerated; incentive spirometry</li> <li>chest X-ray as ordered</li> <li>assess &amp; document fluid balance to rule out fluid overload</li> <li>consider outreach team or ICU consult if fio2 requirements are increasing</li> </ul>
Fluid balance	<ul> <li>monitor for intravascular hypovolemia – STRICT in &amp; output monitoring as early fluid resuscitation is extremely important both in acute pancreatitis &amp; early sepsis</li> <li>monitor IV fluids &amp; assess for signs of fluid overload, including dyspnea, edema &amp; abnormal breath sounds (i.e. crackles) replace NG losses as per doctor's orders &amp; maintain fluid balance</li> </ul>

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	<ul> <li>monitor lab work closely such as serum calcium, potassium, magnesium, albumin and sodium levels etc.</li> </ul>
Coagulation deficits (DVT, DIC, bleed)	<ul> <li>assess for signs &amp; symptoms of hemorrhage, i.e., blue discoloration around umbilicus "Cullen's sign" &amp; flank discoloration - "Turners sign" - which indicates hemorrhagic pancreatitis</li> <li>monitor vital signs closely</li> <li>monitor lab work including INR, PTT, platelets as ordered calf compressors as ordered ambulate as appropriate and encourage leg exercises</li> </ul>
Sepsis	<ul> <li>for a temperature greater than 38.5o C, blood cultures, urine for C &amp; S, and sputum as ordered report signs and symptoms of sepsis to MD</li> <li>antibiotic therapy as ordered</li> <li>monitor tissue &amp; organ perfusion – specifically assess pulse, BP, capillary refill, general cognitive state &amp; urine output assess for hypovolemia, i.e., increase Hct levels, poor skin turgor, dry oral/mucal membranes, etc. &amp; urine output</li> </ul>
Paralytic ileus	<ul> <li>monitor degree and characteristics of pain, i.e., increased abdominal rigidity and distention; report any changes to MD</li> <li>monitor bowel sounds and activity</li> <li>maintain NG suction</li> <li>administer stool softeners as ordered/needed to relieve constipation caused by immobility &amp; opioid use</li> </ul>
Decreased nutritional status	<ul> <li>consult dietitian</li> <li>assess for nausea &amp; vomiting and administer antiemetics as ordered weight daily/weekly as ordered</li> <li>monitor lab values &amp; blood sugar levels as ordered strict intake &amp; output</li> <li>refer to Nutritional Care of Patients with Acute Pancreatitis</li> </ul>
Acute Renal Failure	<ul> <li>monitor creatinine &amp; urea levels</li> <li>indwelling catheter as ordered &amp; monitor urine output</li> </ul>

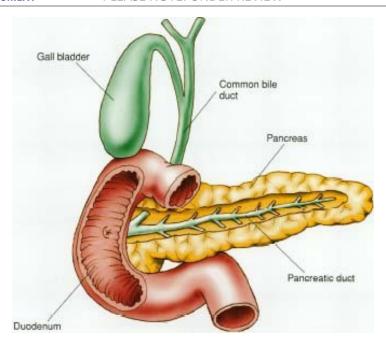
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## **PATIENT / CLIENT / RESIDENT EDUCATION:**

The patient and/or family will be offered education about:

- basic pathophysiology of the pancreatitis and potential complications.
- the importance of nutrition and diabetic counselling with follow-up of required.
- community resources (homemaking, PT/OT/RD, and counseling about alcohol).
- the gradual progression in the level of activity post discharge.
- techniques for pain management.
- how to access help if recurrence of pancreatitis.
- follow-up appointments.

#### **Related Documents**

- Parenteral Nutrition, Care and Management (Adult) [D-00-12-30069]
- Nutritional Care of Patients with Acute Pancreatitis[BD-00-07-40046]
- Septsis [in Development]
- Enteral feeds [In development]

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### References

Amerine, E. (2005). Get optimum outcomes for acute pancreatitis patients. The Nurse Practitioner. 32 (6), 44-48.

1. Australia & New Zealand College of Anaesthetists & Faculty of Pain Medicine Acute Pain Management: Scientific Evidence (2005) NHMRC www.anzca.edu.au

Banks, P., & Freeman, M. (2006). Practice guidelines in acute pancreatitis. American Journal of Gastroenterology. 101:2379-2400.

Berkley, T. & Klamut, K. (2009). Nursing. Acute pancreatitis. Vol. 39(6), June, p. 64.

Brenner, Z. (2010). Nursing Volume: 40 Issue: 1. p. 32-38.

Burruss, N., & Holz, S. (2005). Understanding acute pancreatitis. Nursing 2005, 35(3).

Fawcett, T., & Smith, G. (2005). Acute pancreatitis: Pathophysiology and patient care. Gastrointestinal Nursing, 3 (8), 31-39.

Holcomb, S. (2007). Stopping the destruction of acute pancreatitis. Nursing 2006, June.

Marchiondo, K. (2010). Acute pancreatitis. Medsurg nuring: Jan/Feb2010, Vol. 19, Issue 1, 54-55.

Mayerle, J., Hlouschek, V., & Lerch, M. (2005). Current management of acute pancreatitis. Nature Clinical Practice Gastroenterology & Hepatology, 2(10), 473-483.

Phillips, R. (2006). Acute pancreatitis: inflammation gone wild. Nursing made incredibly easy! September/October 2006, 18-28.

Sargent, S. (2006). Pathophysiology, diagnosis and management of acute pancreatitis. Journal of Nursing (15), 18, 999-1005.

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SharePoint 2nd Reading – Final for Endorsement (PSMs & Affected Council Chairs)

## **Approved for Posting**

SharePoint Final for Sign-Off by VA Operations Directors

Professional Practice Director, Nursing VA

### **Date of Revision**

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