

The EM Educator Series

The EM Educator Series: The Critically Ill Pancreatitis Patient

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Case 1:

A 44-year-old male with known alcohol dependence presents to the ED for severe abdominal that radiates to the back pain for three days. He has had nausea with multiple episodes of non-bloody, non-bilious emesis. On examination his heart rate 110 bpm, but the rest of his vital signs are normal. His abdomen is diffusely tender but with no signs of peritonitis.

Case 2:

A 36-year-old female with known cholelithiasis presents with upper abdominal pain. Her vital signs include HR 115 bpm, temperature 100.5F, RR 22, BP 109/62 mm Hg, and saturation 98% on room air.

Questions for Learners:

1. What are the etiologies of pancreatitis?
2. How can patients with pancreatitis present?
3. What are other mimics of sepsis?
4. What is the ED evaluation for suspected pancreatitis?
5. How do you determine who is critically ill with pancreatitis, and what risk stratification tools are available?
6. What is the management of pancreatitis?
 - How much IV fluid resuscitation is necessary?
 - Which patients should receive antibiotics?
7. When should patients be allowed to eat?
8. Do all patients require inpatient admission?
9. What are the complications of pancreatitis?

Suggested Resources:

- Articles
 - emDOCs – [Pancreatitis Pearls and Pitfalls](#)
 - emDOCs – [Severe Pancreatitis](#)
 - emDOCs – [Pancreatitis Disposition](#)
 - EP Monthly – [Sepsis Mimics](#)
 - PulmCrit – [Pancreatitis](#)
 - REBEL EM – [Acute Pancreatitis](#)
- Journal Articles
 - JEM – [Acute Pancreatitis](#)
 - AGA Guideline – [Acute Pancreatitis](#)

Answers for Learners:

1. What are the etiologies of pancreatitis?

- Gallstones (~40%).
- Alcoholism (~30%).
- Metabolic abnormalities: hypertriglyceridemia, hypercalcemia.
- Medications include:
 - Antibiotics: tetracyclines, sulfonamides, pentamidine, HIV medications, isoniazid, metronidazole.
 - Immunosuppressives: azathioprine, sulfasalazine, aminosalicylates, 6-mercaptopurine.
 - Cardiac: amiodarone, losartan, furosemide, pravastatin, simvastatin.
 - Valproic acid.
 - All-trans-retinoic acid (ATRA).
 - Glucagon-like peptide-1 agonist therapy for diabetes.
- Posterior penetrating ulcer, trauma.
- Iatrogenic: ERCP, surgery, radiation therapy, post CABG.
- Pancreatic malignancy.
- Cystic fibrosis.

2. How can patients with pancreatitis present?

The diagnosis of acute pancreatitis is made by meeting 2/3 criteria: upper abdominal pain (clinical), serum lipase or amylase >3x upper limit of normal (laboratory), or imaging findings of pancreatic inflammation.

The classic presentation of pancreatitis is persistent upper abdominal pain. The pain may radiate to the back and may be associated with nausea, vomiting, anorexia, and decreased oral intake, as pain is frequently exacerbated with oral intake. Pain can be severe, but the level of pain does not correlate with clinical severity. Vital signs may be abnormal, with tachycardia, hypotension, or fever present.

Exam findings include epigastric or upper abdominal tenderness. Late stage, severe pancreatitis can result in skin discoloration around the umbilicus (Cullen's sign) or flank (Grey-Turner sign) from retroperitoneal hemorrhage.

3. What are other mimics of sepsis?

Table 2 – Common Sepsis Mimics

CLINICAL CONDITION	DIAGNOSIS	TREATMENT
Anaphylaxis	<ul style="list-style-type: none"> -Involvement of two organ systems (dermal, GI, respiratory, hemodynamic), or known allergen exposure with hypotension warrants consideration. -Fever unlikely to be present. -Inquire about allergic exposures, known allergies. -Patient may present with distributive shock (vasodilated and hypotensive). 	<ul style="list-style-type: none"> -Epipen or Epinephrine 1:1000 0.3-0.5ml IM in the lateral thigh. -If initial IM epinephrine insufficient, provide another dose of IM epinephrine and start IV drip.
Aspiration	<ul style="list-style-type: none"> -Due to chemical injury of the respiratory tract, resulting in cough, dyspnea, tachypnea. -Requires 20 ml of aspirated content, classically with pH < 2.5. -Evaluate for risk factors: stroke, periodontal disease, elderly, and on chronic proton pump inhibitor therapy. -May present with fever and infiltrate on chest radiograph. 	<ul style="list-style-type: none"> -If patient experiences symptoms, experiences a fever, and demonstrates chest radiograph with infiltrate, treat with antibiotics. -No fever with aspiration warrants observation.
Adrenal Insufficiency	<ul style="list-style-type: none"> -Abdominal pain, nausea/vomiting, diarrhea, weakness, fatigue, salt craving, and hypotension resistant to IV fluids/vasopressors. -Evaluate carefully for risk factors such as corticosteroid use over two weeks, cancer, and tuberculosis. -Laboratory findings often include hyponatremia, hyperkalemia, hypoglycemia, and hypercalcemia 	<ul style="list-style-type: none"> -Baseline cortisol testing can be completed to assist with further management, but is not mandatory. -Hydrocortisone in stress doses (100mg/IV X1) or dexamethasone 4 mg IV. -Appropriate fluid resuscitation with D5 NS for repletion of glucose is often needed.
Bowel Obstruction	<ul style="list-style-type: none"> -Episodic abdominal pain, nausea, vomiting (may have diarrhea due to gut-emptying). -CT of the abdomen and pelvis is the best test for diagnosis, as plain Xrays have sensitivities in the range of 60%. -US has recently demonstrated utility for diagnosis of bowel obstruction. 	<ul style="list-style-type: none"> -Provide fluid resuscitation, control pain and nausea, and consult the surgical team.
Diabetic Ketoacidosis	<ul style="list-style-type: none"> -May be tachypneic, hypotensive, dehydrated, and hypotensive from osmotic diuresis. -Elevated ketones and lactic acid with decreased pH is required. -Diagnosis requires ketosis, acidosis, and hyperglycemia. -Obtain ECG, metabolic profile, urinalysis, lipase, chest radiograph, and TSH. 	<ul style="list-style-type: none"> -Evaluate closely for cause of DKA: infection, ischemia, noncompliance, drug abuse, PE, pancreatitis, bleeding, and trauma. -Fluid resuscitation is required initially, with insulin following. -Closely monitor potassium.
Heat Emergency	<ul style="list-style-type: none"> -Presents as a continuum: heat cramps and edema to heat stroke, which presents with altered mental status and temperatures often greater than 40oC (104oF). -Heat stroke exists in exertional and nonexertional forms. -Obtain history on the scene, environment, patient activity, and medications. -Do not rely on peripheral temperature. Core temperature is necessary. 	<ul style="list-style-type: none"> -Remove the patient from the environment. -Provide cooled IV fluid resuscitation. -For heat stroke, use evaporative cooling or immersion cooling.

4. What is the ED evaluation for suspected pancreatitis?

Labs play an important role in the diagnosis, treatment, prognostication, and monitoring of patients with severe pancreatitis. Laboratory markers that can aid in the diagnosis of patients with abdominal pain include lipase, white blood cell count, bilirubin level, and transaminases. Acute phase reactants such as the C-reactive protein (CRP) and procalcitonin, as well as serum triglycerides, are less commonly ordered in the ED setting, but can assist in the continued workup and management of patients who are admitted.

Computed tomography (CT) of the abdomen with contrast is the best modality to visualize pancreatic inflammation in the ED, but most cases of uncomplicated, mild pancreatitis do not need CT imaging. CT should be obtained, however, when there is suspicion for necrotizing pancreatitis, as imaging will guide

surgical management and alter prognostication. However, the distinction between mild and severe pancreatitis, discussed above, is clinical. Pain out of proportion, failure to improve as expected, hemodynamic instability, an unclear diagnosis, and severe metabolic derangements are all factors which increase the utility of early imaging.

5. How do you determine who is critically ill with pancreatitis, and what risk stratification tools are available?

Gauging the severity of the patient's presentation is important in guiding management and disposition. There are two widely used sets of clinical criteria for making this assessment: Ranson's criteria and the bedside index of severity in acute pancreatitis (BISAP).

Ranson's score was developed in the 1970s. It primarily utilizes laboratory markers to predict the severity of acute pancreatitis. Points are assigned in a binary fashion — either the marker is above (+1) or below (+0) a threshold value. A score of greater than or equal to three suggests severe pancreatitis with a predicted mortality of 15% at 3 points and 40% at 5 points. A total score of 7 predicts 100% mortality. Notably, the score must be calculated at admission and 48 hours after admission.

In 2008, the BISAP score was developed and validated for use in the prediction of severe acute pancreatitis. It utilizes both clinical data (such as altered mental status and the presence of a pleural effusion) in addition to laboratory markers. It performed similarly to Ranson's criteria but is simpler and can be calculated at 24 hours after admission rather than at 48 hours. In the BISAP tool, a score of 0 is associated with <1% mortality, 2 with 1.9% mortality, and 5 with 22% mortality.

6. What is the management of pancreatitis?

○ How much IV fluid resuscitation is necessary?

Previously, aggressive fluid therapy was integral in the treatment of acute pancreatitis. Patients were not uncommonly given upwards of 10 liters of IV crystalloids over the first 24 hours of a hospital admission. However, abdominal compartment syndrome and acute respiratory distress syndrome (ARDS), once thought to be characteristic of acute pancreatitis may in fact be iatrogenic complications caused by the over-administration of intravenous fluid.

Nevertheless, volume expansion remains important; volume depletion is almost universal in patients with pancreatitis, due to gastrointestinal losses from vomiting, insensible losses from fever and tachypnea, and vasodilation from concomitant infection and a systemic inflammatory response. There is not strong evidence on the ideal amount of fluid for the initial resuscitation, but some studies demonstrate higher rates of ARDS and abdominal fluid collections in patients that receive more than about four liters in the first 24 hours. The International Association of Pancreatology released guidelines that suggest this as a reasonable limit as well. Additionally, this is similar as the amount of fluid recommended in patients with severe sepsis, which has a similar pathophysiology to severe pancreatitis. Therefore, we recommend a bolus of one or two liters of crystalloids (20-30 mL/kg) with transition to vasopressors as necessary. This approach may decrease the risk of iatrogenic volume overload. However, high-quality, randomized trials are still needed to determine the optimal volume of fluid administration in patients with severe acute pancreatitis.

- **Which patients should receive antibiotics?**

Patients with necrotizing pancreatitis are at high risk for superimposed infection, which can lead to multisystem organ failure and severe acute pancreatitis. Antibiotics should be avoided when features consistent with superimposed infection are not present (fever, abscess, peripancreatic gas, hemodynamic instability), as this may cause the development of multidrug resistant organisms. Superimposed infection is usually due to translocation from gut microbes and is often polymicrobial. The initial agents should be broad, cover anaerobic organisms, and be selected in conjunction with the hospital's antibiogram. The typical clinical course of a bacterial superimposed infection is improvement in symptoms or clinical status followed by an abrupt worsening, usually about a week after symptoms first began.

7. When should patients be allowed to eat?

For context, all patients with pancreatitis historically were made nothing by mouth (NPO) status until symptoms were essentially resolved. Since 2008-2010, this practice has shifted in patients with mild pancreatitis—early oral refeeding, if tolerated, is now encouraged, as some studies have shown a reduction in hospital length-of-stay with this approach. In patients with severe pancreatitis, this same paradigm shift has not occurred, as there are no studies in that population to support this practice. In patients with severe disease, this is a decision best left to the inpatient team that will be managing the patient; generally, patients with severe disease should be kept NPO in the ED.

8. Do all patients require inpatient admission?

The following criteria (several of which are major criteria from BISAP) are reasonable indicators that a patient is a high risk of decompensation and may require an ICU level of care:

- SIRS criteria
- Any evidence of pancreatic or peripancreatic necrosis on imaging
- Acute renal failure
- Signs of large volume third spacing:
 - Hemoconcentration on labs
 - Evidence of pleural effusions on CXR or abdominal CT (if obtained)
- Newly elevated blood urea nitrogen (BUN) > 25 mg/dl

This list is not all-inclusive. Any vital sign or laboratory derangement that would generally require ICU levels of care applies equally to acute pancreatitis.

9. What are the complications of pancreatitis?

- Necrotizing pancreatitis can cause acute necrotic collections, infected pancreatic necrosis, and walled-off necrosis. Alternatively, interstitial edematous pancreatitis may cause acute peripancreatic fluid collections and pseudocysts.
- Abdominal compartment syndrome is largely an iatrogenic complication, due to the use of excessive volumes of crystalloid. As we are moving away from large-volume resuscitation of pancreatitis, this seems to be less of a problem.
- Hemorrhage may result from erosion of arteries near the pancreas (especially the splenic or gastroduodenal arteries).
- ARDS

- Cholangitis
- Alcohol Withdrawal