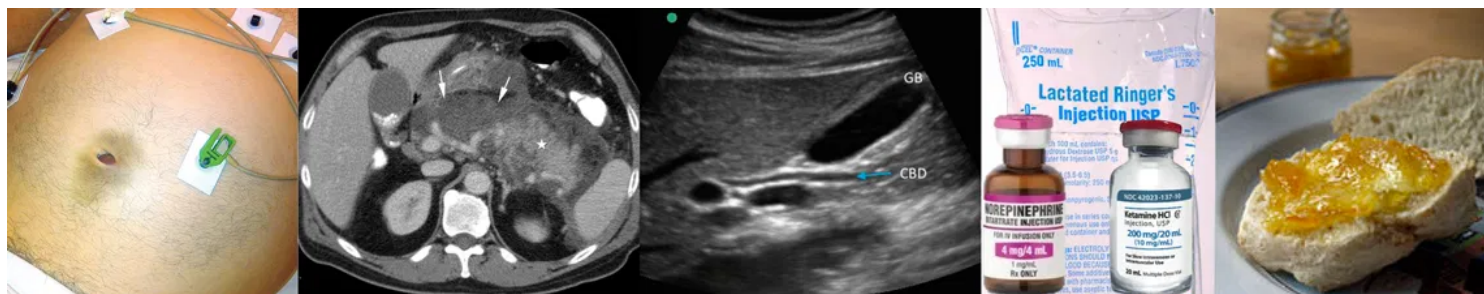


# The Internet Book of Critical Care

## Acute Pancreatitis

November 23, 2016 by [Josh Farkas](#)



(<https://io.wp.com/emcrit.org/wp-content/uploads/2016/11/pancbanner.jpg>)

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## diagnosis

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### diagnostic criteria for acute pancreatitis

- At least two of the following are required:

- (1) Elevation of lipase >3 times upper limit normal (1)
- (2) Characteristic abdominal pain
- (3) Imaging evidence of pancreatitis on CT, MRI, or ultrasound.
- Patients not meeting these criteria don't have pancreatitis and should not be treated for it.

### clinical findings

- Pain:
  - Typically in epigastrium or left upper-quadrant, may radiate to back.
  - Epigastric tenderness on exam is usually present.
- Persistent nausea/vomiting
- Hemorrhagic pancreatitis may cause Cullen Sign & Grey Turner Sign:



Several signs of pancreatitis are shown here. Most obvious are ecchymosis of the umbilicus (Cullen sign) and flanks (Grey Turner Sign) due to retroperitoneal bleeding. Jaundice (corresponding to a bilirubin of 4.2 mg/dL here) and abdominal distension can also be seen. Valette X & du Cheyron D 2015 NEJM PMID 26650175

(<https://io.wp.com/emcrit.org/wp-content/uploads/2016/11/turnerssign2.jpg>) **lipase**

- Sensitivity and specificity ~90% for acute pancreatitis.
- Causes of elevated lipase include:
  - Pancreatic disease of any sort (pancreatitis, pseudocyst, cancer)
  - Intestinal obstruction/pseudo-obstruction, perforation, duodenal ulceration, ischemia
  - Biliary disease (cholecystitis, cholangitis, choledocholithiasis)
  - Renal failure
- Elevations of lipase due to diseases other than pancreatitis tend to be under three times the upper-limit normal.
  - Very high lipase values are more *specific* for a diagnosis of pancreatitis.
  - Higher lipase values *don't* correlate with worse prognostic outcome. So severely elevated lipase values may seem scary, but they shouldn't actually be.
- Lipase has replaced amylase for the diagnosis of pancreatitis. There is no point in checking an amylase level.



**Gordon Johnson**  
@pdxfootball

Epigastric pain, elevated LFTs & lipase. #POCUS shows "wall echo shadow" of GB. Likely gallstone pancreatitis. Image - @PhilipsLumify. Confirmed with CT



14 4:48 PM - Dec 24, 2017 · Portland, OR

[See Gordon Johnson's other Tweets](#)

## CT scan

- Early CT if necessary to clarify the diagnosis
  - CT is sensitive and specific for pancreatitis, also providing information about severity.
  - If the patient definitely has pancreatitis (based on typical history, exam, and labs), then there is no reason to get an early CT scan (it won't affect management).
  - For patients in shock, CT scan is often sensible to exclude a focus of intra-abdominal sepsis.
- Late CT scan for complications
  - The real role of CT scan in pancreatitis is to look for complications if the patient deteriorates later on in their course (after several days). For example, CT scan may help evaluate for infected necrosis and pseudocyst.

## evaluating the cause of pancreatitis

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### common causes of acute pancreatitis

- Gallstones (~40%)<sup>1</sup>
- Alcoholism (~30%)
- Metabolic abnormalities: hypertriglyceridemia, hypercalcemia
- Medications include:<sup>2</sup>
  - 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)
- Cystic fibrosis
- Posterior penetrating ulcer, trauma
- Iatrogenic: ERCP, surgery, radiation therapy, post CABG
- Pancreatic malignancy

### labs

- Calcium
  - Hypercalcemia is a rare cause of pancreatitis
  - Hypocalcemia can occur due to pancreatitis itself, occasionally causing symptomatic hypocalcemia.
- Triglyceride level (>1,000 mg/dL or 11.2 mM suggests hypertriglyceridemic pancreatitis)
- Liver function tests
  - Significantly elevated bilirubin & alkaline phosphatase suggest obstruction, raising a possible concern of simultaneous ascending cholangitis.

|             |                               |  |
|-------------|-------------------------------|--|
| WBC         | 4.0 - 12.4 K/mm <sup>3</sup>  | 9.35   |
| RBC         | 3.86 - 5.04 M/mm <sup>3</sup> | 3.62 (L)   |
| Hemoglobin  | 11.6 - 15.2 gm/dL             | Hemoglobin not reportable due to presence of lipemia   |
| HCT         | 34.9 - 44.4 %                 | 35.1   |
| MCV         | 81 - 98 fL                    | 97   |
| MCH         | 26.7 - 33.3 pg                | Could not be calculated due to unreportable hemoglobin |
| Hypochromia |                               | Could not be calculated due to unreportable hemoglobin |
| MCHC        | 32.1 - 35.9 gm/dL             | Could not be calculated due to unreportable hemoglobin |

If the lab can't run tests because the blood is lipemic, your patient probably has hypertriglyceridemic pancreatitis.

*The Internet Book of Critical Care, by @PaulMcrit*

<https://iz.wp.com/emcrit.org/wp-content/uploads/2016/11/hypertag.jpg> **right upper-quadrant ultrasonography**

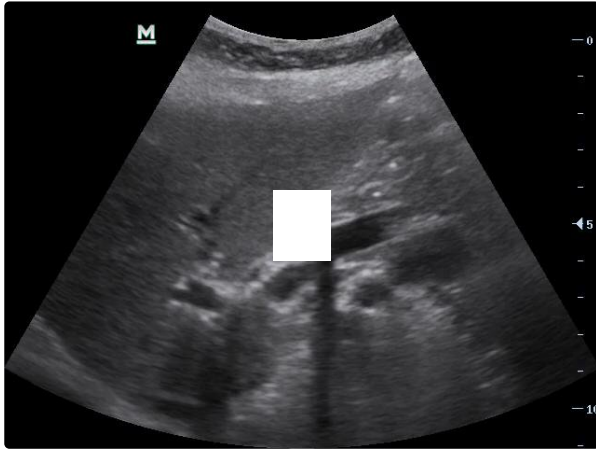
- Should be obtained on all pancreatitis patients.<sup>3</sup>

- Gallstones may suggest gallstone pancreatitis.
- Most important finding is size of the common bile duct.



**Mohammed Alshamsi**  
@EmergOmBur

When CBD is dilated, no one should miss it. Just anterior to PV.  
Use color or power if in doubt. #POCUS #FOAMed



14 1:58 PM - Jan 9, 2016

23 people are talking about this

## risk stratification- who needs ICU?

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Most pancreatitis patients have mild disease and can be admitted to the ward, but some require ICU admission. This is tricky because pancreatitis patients may look OK initially, but deteriorate later.

### classification: edematous vs. necrotizing pancreatitis

- Pancreatitis can be divided into two categories:
  - Interstitial edematous pancreatitis (90%) – diffuse inflammation of the pancreas, tissue remains viable.
  - Necrotizing pancreatitis (10%) – areas of pancreatic tissue become necrotic.
- The diagnosis of necrotizing pancreatitis is generally made based on contrast CT scan, which shows a lack of blood flow to necrotic areas. Note, however, that CT scan shouldn't be obtained solely for this purpose.
- Necrotizing pancreatitis is more worrisome, as these patients are at risk for developing multiorgan failure or superinfection of the devitalized pancreatic tissue (infected pancreatic necrosis). The mortality rate of necrotizing pancreatitis is 17%, much higher than the mortality of interstitial edematous pancreatitis at 3%.<sup>2</sup>

### scoring systems?

- About a dozen scoring systems are available to predict outcomes. However, it's unclear what the role of scoring systems should be, compared to clinician judgement. I'm unaware of prospective evidence that any score is superior to clinical judgement.
- APACHE-II score seems to be the best scoring system, with scores >7 predicting severe acute pancreatitis. Unfortunately its performance is far from perfect, with sensitivity of 65% and specificity of 76% for severe pancreatitis ([APACHE-II calculator \(http://www.mdcalc.com/apache-ii-score/\)](http://www.mdcalc.com/apache-ii-score/)).
- The Ranson score can't be fully calculated for 48 hours, so it plays no meaningful role in up-front risk stratification.

### neutrophil-lymphocyte ratio (NLR)

| ROUTINE HEMATOLOGY   |                    |
|----------------------|--------------------|
| WBC                  | 8.93               |
| RBC                  | 4.45               |
| Hemoglobin           | 13.0               |
| HCT                  | 39.0               |
| MCV                  | 88                 |
| MCH                  | 29.2               |
| MCHC                 | 33.3               |
| RDW-CV               | 15.2               |
| RDW-SD               | 48.7               |
| PLT                  | 89                 |
| MPV                  | 11.0               |
| Neutrophils          | 84.0               |
| Bands                | 6.0                |
| Lymphocytes          | 3.0                |
| Monocytes            | 6.0                |
| Metamyelocytes       | 1.0                |
| ABS Neutrophils      | 7.49               |
| ABS Bands            | 0.54               |
| ABS Lymphs           | 0.27               |
| ABS Monocytes        | 0.54               |
| ABS Metamyelocytes   | 0.09               |
| Differential Comment | Reviewed by Dr.... |
| Type of Diff:        | Manual             |
| WBC MORPHOLOGY       |                    |
| Vacuolization        | Present            |

$$\text{NLR} = 84/3 = 28$$

$$\text{NLR} = 7.49/0.27 = 28$$

This patient with pancreatitis and ascending cholangitis was admitted to ICU due to tenuous hemodynamics and gram-negative bacteremia. The neutrophil/lymphocyte ratio is 28 (markedly elevated) despite a normal WBC. The patient did fine with an ICU stay of only one day.

*The Internet Book of Critical Care, by @PulmCrit*

(<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/calculnrl.jpg>).

- NLR is easily calculated from an admission hemogram as shown above, so it's immediately available on all of your patients for free.
- NLR measures physiologic stress. Under stress the neutrophil count tends to rise, lymphocyte count tends to fall, so the NLR increases.
- Higher NLR values predict severe pancreatitis (area under ROC curve of ~0.75)<sup>4 5 6 7</sup> and mortality (area under ROC curve ~0.8).<sup>4 8</sup> NLR seems more accurate than C-reactive protein, a lab test which is occasionally recommended for prognostication in pancreatitis.<sup>8 9</sup>
- The table below provides a rough guide to interpreting NLR.<sup>4</sup> NLR usually decreases over time as pancreatitis resolves. Studies have predominantly evaluated the prognostic value of NLR upon admission.
- Prognostication shouldn't be based solely on NLR, but NLR may provide another bit of information to contribute to the global clinical assessment.

#### interpretation of admission Neutrophil/Lymphocyte Ratio (NLR) in acute pancreatitis

| NLR   | Require ICU care for >1 week* | In-hospital mortality* | Overall interpretation                        |
|-------|-------------------------------|------------------------|---|
| <6    | 4%                            | 0.8%                   | Reassuring                                    |
| 6-10  | 14%                           | 1.6%                   | Grey zone – provides little added information |
| 10-15 | 26%                           | 7%                     | Worrisome                                     |
| >15   | 39%                           | 14%                    | More worrisome                                |

\*Numbers are from a single-center study (PMID 27631223). Exact rates will vary between different hospitals with varying patient populations.

*The Internet Book of Critical Care, by @PulmCrit*

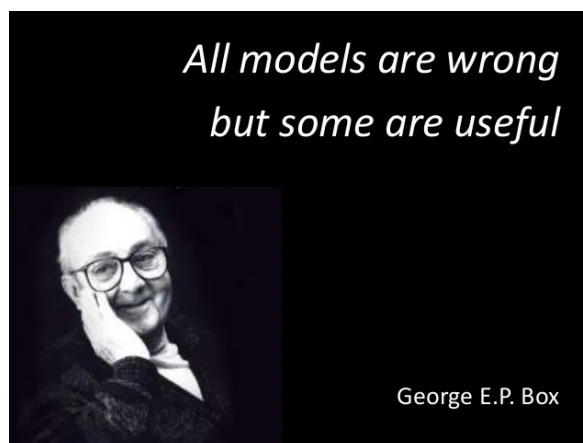
(<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/admitnrl.jpg>) possible indications for ICU admission?

- Any patient felt to require aggressive fluid resuscitation (e.g. >1-2 liters total). As discussed below, fluid resuscitation should be used only as needed to support perfusion. If the patient is so sick that they require large-volume resuscitation, then they should be admitted to an ICU.
- Respiratory insufficiency (e.g. significant tachypnea or increased work of breathing).
- Other organ failures (e.g. poor urine output, kidney injury, delirium, hypotension).
- Hypertriglyceridemic pancreatitis (triglyceride >1,000 mg/dL or 11.2 mM) should be considered for insulin infusion in the ICU.

## general principle- pancreatoseptic equivalence

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Historically, pancreatitis has been treated as a *unique* disease. It was feared by intensivists, who skittishly deferred management to gastroenterologists and surgeons. Pancreatitis was treated with vast quantities of fluid, bowel rest, nasogastric tube drainage, parenteral nutrition, and prophylactic antibiotics – a wholly weird potpourri of therapies inconsistent with basic principles of critical care. Only recently have we begun the hard work of dispelling these harmful treatments.



(<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/test2.jpg>)

### principle of pancreatoseptic equivalence

- Severe pancreatitis and septic shock are extremely similar processes (both vasodilatory shock states involving profound systemic inflammation and endothelial dysfunction).
- The treatment for severe pancreatitis should follow the same principles as the treatment of septic shock.
- Evidence gaps regarding how to manage severe pancreatitis can be filled with experience gained from the treatment of septic shock.

This may not be 100% accurate, but it's a huge advance compared to the bizarre ways in which pancreatitis has been treated over the past few decades.

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## treatment of causative factors

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### hypertriglyceridemic pancreatitis

- Definition of hypertriglyceridemic pancreatitis: Pancreatitis with triglyceride level over ~1,000 mg/dL (11.2 mM) in the absence of another obvious cause of pancreatitis.
- Triglyceride level should be lowered, usually with an insulin infusion. However, before admitting the patient to ICU for an insulin infusion make sure that they truly have pancreatitis as defined above (elevated triglyceride level in the *absence* of pancreatitis doesn't require an insulin infusion).
- More on this [here](https://emcrit.org/pulmcrit/hypertriglyceridemic-pancreatitis/) (<https://emcrit.org/pulmcrit/hypertriglyceridemic-pancreatitis/>).

### other causes

- **Medication-induced:** stop potentially offending medications
- **Hypercalcemia:** treat this aggressively (e.g. with bisphosphonate & procalcitonin).
- **Gallstone-induced:** ideally cholecystectomy during admission for acute pancreatitis

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## ERCP?

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- The American Gastroenterological Association recommends *against* routine urgent ERCP in patients with acute biliary pancreatitis without cholangitis. Most studies of ERCP have failed to show benefit.



- The strongest indication for ERCP is definite or suspected *ascending cholangitis* (which sometimes occurs simultaneously with pancreatitis, serving as a focus of septic shock). Evidence of cholangitis may include:
  - Dilation of the common bile duct
  - Significantly elevated & rising bilirubin
- ERCP may also be indicated for patients with evidence of choledocholithiasis (e.g. persistently elevated bilirubin, impacted stone visualized radiographically).
- When in doubt about the need for ERCP, possible approaches are:
  - Follow the patient clinically, with serial monitoring of liver function tests and overall picture.
  - Magnetic Resonance Cholangiopancreatography (MRCP) – a noninvasive imaging modality to better define the anatomy of the biliary tract.

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## resuscitation

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### traditional dogma: large volume fluid resuscitation

- Traditionally patients have been treated with massive fluid resuscitation (e.g. 250-500 ml/hr, resulting in ~8-14 liters fluid administration over the first day). This is insanity.
- There is *no evidence* to support massive volume administration. Available prospective studies show that more aggressive fluid administration increases rates of infection, abdominal compartment syndrome, ARDS, and death.<sup>10 11</sup>

### reasonable approach?

- Nobody knows the best approach, there is little high-quality prospective data to guide this.
- A reasonable approach to resuscitation is probably similar to a septic shock resuscitation:
  - Give fluid based on hemodynamic assessment (e.g. with ultrasonography). Most patients may benefit from a *moderate* amount of fluid initially (e.g. 2-4 liters total over the first day).
  - Don't give much fluid after the initial resuscitation (e.g. beyond 12-24 hours). Following stabilization, it's generally wise to target an *even* fluid balance (inputs = outputs).
  - Use vasopressors (e.g. norepinephrine) early, as needed to maintain an adequate MAP. This may reduce the amount of fluid given, thereby reducing the risk of abdominal compartment syndrome (more on this below).
- Be careful about the use of *fluid-responsiveness* in these patients. Even if the patient is fluid-responsive, administered fluid will rapidly leak out of the vascular space. Pancreatitis patients will often be fluid-responsive regardless of how much fluid they are given.
- Patients will often develop renal failure due to acute tubular necrosis. This doesn't respond to additional fluid administration.

### lactated ringers (LR) is preferred

- Lactated Ringers appears to be the fluid of choice, given one RCT in pancreatitis that found reduced inflammation when using LR compared to normal saline.<sup>12</sup>
- LR is an [excellent](https://emcrit.org/pulmcrit/smart/) resuscitative fluid anyway, so it shouldn't take a lot of evidence to convince us to use it here.

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## nutrition

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### the concept of "pancreatic rest" is dangerously misleading dogma

- Traditionally there was a concept that nutrition would stimulate pancreatic secretions and thereby worsen pancreatitis.
- Not only is this wrong, it's probably backwards.<sup>13</sup> Early enteral nutritional support (ideally within 24 hours) may improve outcomes, for example
  - Improved intestinal function (reduced rate of ileus, decreased bacterial translocation into the bloodstream)
  - Reduced risk of infected pancreatic necrosis.
  - Reduced hospital length of stay.
  - Decreased gastrointestinal symptoms.<sup>1</sup>

### nutritional support for the non-intubated patient

- Oral diet may be started immediately.
- It's fine to start with a low-fat diet (there is no need to start with a clear-liquid diet).
- Some patients are unable to tolerate food (e.g. due to pain or emesis). This may be observed for a couple days. If the patient still isn't eating after 3-5 days, a small-bore nasal feeding tube may be placed to provide nutrition.<sup>14</sup> It's ideal to place this in a post-pyloric location, but gastric feeding is also fine.

### nutritional support for the intubated patient

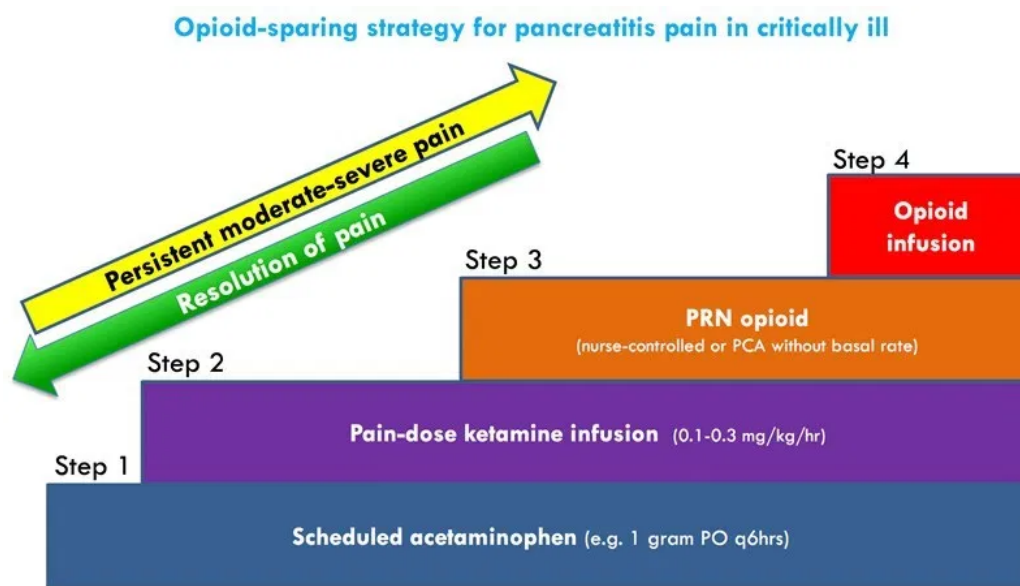
- Enteral tube feeding should be started immediately after the initial resuscitation. Start feeds at a trickle level (10-20 ml/hr) and advance as tolerated.
- It is controversial whether to feed the stomach (e.g. via nasogastric/orogastric tube) or to use a post-pyloric tube.
  - RCTs show no differences in outcome: either route is fine.
  - Among intubated patients it's usually easier to place an orogastric tube, so this route is often used initially.
  - If the patient has problems with gastroparesis or vomiting, then switching to a post-pyloric tube may be helpful.

### total parenteral nutrition should be avoided

- RCTs in pancreatitis have shown harm from parenteral nutrition. This has been shown to increase the risk of infected pancreatic necrosis and multi-organ failure.<sup>13</sup>
- Parenteral nutrition should be used only as a last resort, when enteral nutrition is impossible.

## analgesia

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Most patients with pancreatitis will require scheduled acetaminophen and a pain-dose ketamine infusion. Opioids may be needed for refractory pain (Step #3-4). Limitation of opioids may theoretically reduce the risk of ileus, nausea/vomiting, abdominal compartment syndrome, and failure of enteral nutrition. However, there isn't any strong evidence to support this strategy.

*The Internet Book of Critical Care, by @PulmCrit*

<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/pancpain.jpg>

- Opioids may promote ileus, interfering with nutrition and potentially increasing the risk of abdominal compartment syndrome. Most patients will need some amount of opioid, but this should be kept to a minimum.
- Pain-dose ketamine infusions (e.g. 0.1-0.3 mg/kg/hr) may be helpful to control pain while avoiding opioids.<sup>15 16 17</sup>
- Scheduled acetaminophen shouldn't be forgotten, as this may also be opioid-sparing (e.g. acetaminophen 1 gram every six hours). Among patients with cirrhosis or severe alcoholism, this should probably be reduced to a dose of two grams daily (e.g. 650 mg every eight hours).<sup>18</sup>
- Non-steroidal anti-inflammatory agents should be avoided in critically ill patients (and especially among patients with pancreatitis), because these patients are at a higher risk of acute kidney injury.



- Epidural analgesia may be considered if available.

## antibiotics & infected pancreatic necrosis

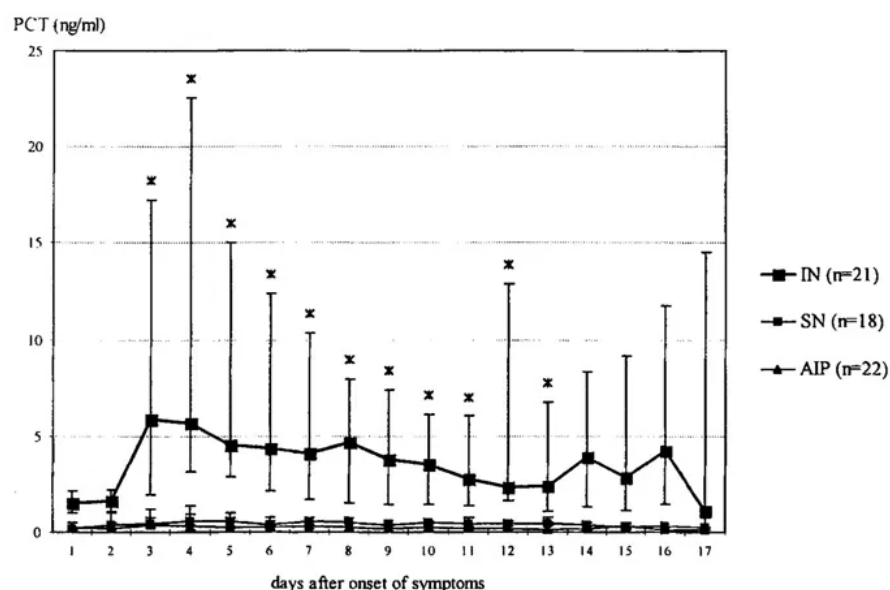
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### avoid antibiotics in the first week

- As discussed above, there are many parallels between sepsis and pancreatitis. These will cause the pancreatitis patient to *look* infected upon arrival (e.g. pancreatitis commonly causes fever, leukocytosis, hypotension, and vasodilatory shock). However, this is generally a reflection of sterile inflammation rather than true infection.
- Historically there was a concept that *prophylactic antibiotics* could prevent the development of infected pancreatic necrosis. This has been debunked and should not be used. Up-front antibiotics will select out resistant organisms, which cause problems later on (when true infection actually does occur).
- Antibiotics should generally be avoided during the first week, with the following exceptions:
  - (a) The diagnosis of pancreatitis is unclear and there is concern for septic shock with a focus of infection elsewhere.
  - (b) The patient has coexisting ascending cholangitis (which is a true bacterial infection and requires decompression & antibiotics).
- Infectious complications of pancreatitis (e.g. infected necrosis) are rare during the first week. During this time frame, inflammatory symptoms (e.g. fever, leukocytosis) likely reflect sterile pancreatic inflammation.

### infected pancreatic necrosis

- This peaks about 10-14 days after the onset of pancreatitis. The classic presentation would be a patient who initially improves, but subsequently deteriorates with worsening sepsis.
- Investigation typically begins with repeat CT scan. Occasionally, radiologic features may be diagnostic (e.g. air within pancreatic tissue implies infection).
- Fine-needle aspiration to determine whether infection is present is routinely used at some centers and recommended in the Canadian guidelines for acute pancreatitis.<sup>19</sup> However, empiric antibiotics are favored at some centers due to fear of introducing infection into the pancreas during fine-needle aspiration.<sup>20</sup>
- Traditionally a carbapenem (e.g. meropenem) as used for improved penetration of the pancreas. However, other antibiotics also penetrate the pancreas well (e.g. cefepime/metronidazole, piperacillin-tazobactam).<sup>21 22</sup> Given that these patients often remain in the ICU for some weeks, using piperacillin-tazobactam initially (instead of a carbapenem) could limit the selection of resistant pathogens.
- A team approach is required for these stubborn problems, including pancreatic surgeons, interventional radiologists, and invasive gastroenterologists. Ideally this should be managed at a large center which offers a range of minimally invasive debridement techniques.<sup>2</sup>



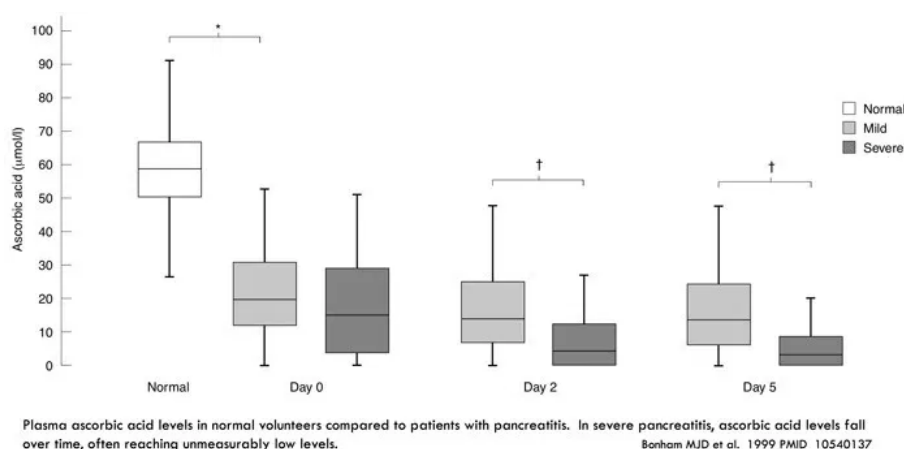
Procalcitonin trends among patients who develop infected necrosis (IN), sterile necrosis (SN), or acute interstitial edematous pancreatitis (AIP). Infected necrosis is associated with an early elevation in procalcitonin values (e.g. on day 3-4) as well as re-elevation of procalcitonin (e.g. on days 14-16). Rao et al. 2000 (PMID 18470712).

<https://i.wp.com/emcrit.org/wp-content/uploads/2016/11/pcttrends.jpg> **procalcitonin use in pancreatitis?**

- Although procalcitonin is often conceptualized as a test for bacterial sepsis, it can be elevated in pancreatitis as well (as might be expected based on similarities between these two conditions). Procalcitonin may potentially be used for two purposes:
- (1) Risk stratification**
  - Greater procalcitonin elevation reflects more severe inflammation, which may predict a more severe disease course.
  - Elevation of procalcitonin >0.5 ng/mL predicts severe pancreatitis with moderate reliability (sensitivity 73%, specificity 87%).<sup>23</sup>
- (2) Diagnosis of infected pancreatic necrosis**
  - Pancreatitis alone generally doesn't cause profound elevation in procalcitonin. Therefore, a markedly elevated procalcitonin level (e.g. >3.5 ng/ml) is suggestive of infected pancreatic necrosis.<sup>24 25</sup>
  - Other causes of procalcitonin elevation include renal failure and other foci of nosocomial infection (e.g. line infection, pneumonia).
  - The value of procalcitonin for infected pancreatic necrosis is likely as a *rule-out* test (e.g. a low procalcitonin argues against infected necrosis, whereas an elevated value is nonspecific). This might be useful in avoiding unnecessary antibiotic courses or invasive procedures in patients at low risk for true infection. Further prospective evidence is needed to validate this.

## metabolic resuscitation

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### <https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/vitcpanc.jpg> rationale for ascorbic acid (vitamin C)

- Ascorbic acid is required for a variety of processes, including endothelial integrity and catecholamine synthesis.
- Severe pancreatitis causes a drop in ascorbic acid levels, often to undetectably low levels.<sup>26 27</sup>
- Animal models of pancreatitis show benefit from ascorbic acid administration.<sup>28 29</sup>
- Intravenous ascorbic acid has been tested in a single RCT for the treatment of acute pancreatitis. Administration of 10 grams daily improved symptoms and reduced the rate of clinical deterioration.<sup>30</sup>
- Small RCTs have suggested benefit from ascorbic acid in septic shock, which as discussed above is closely related to severe pancreatitis.<sup>31 32</sup>

### rationale for thiamine (vitamin B1)

- Alcoholism is the second leading cause of pancreatitis. Patients with pancreatitis due to alcoholism are likely to have pre-existing thiamine deficiency.
- The body has minimal thiamine reserves. Consequently, it's not uncommon for thiamine deficiency to develop among critically ill patients with poor oral intake.<sup>33 34</sup>
- Some studies have suggested benefit from thiamine in sepsis, a closely related disorder.<sup>35 36</sup>

### metabolic resuscitation cocktail (hydrocortisone / ascorbic acid / thiamine)

- Recently some biochemical and clinical [evidence \(https://emcrit.org/pulmcrit/metabolic-sepsis-resuscitation/\)](https://emcrit.org/pulmcrit/metabolic-sepsis-resuscitation/) has emerged to support a cocktail of hydrocortisone, ascorbic acid, and thiamine in septic shock.<sup>37</sup> Specifically, hydrocortisone and ascorbic acid function synergistically to improve endothelial function.<sup>38</sup> However, this remains extremely controversial.
- There is some limited evidence to support a role of steroid in acute pancreatitis.<sup>39</sup> Together with borrowed evidence from septic shock, this suggests possible benefit from metabolic resuscitation cocktail in pancreatitis.

- If there are concerns about potential side effects from steroid, a combination of ascorbic acid and thiamine may be considered.

## abdominal compartment syndrome

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- Compartment syndrome can cause deterioration and multi-organ failure.
- This 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.

## algorithm

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## Pancreatitis management checklist

### ☐ Evaluation to guide etiology & management

- ☐ RUQ ultrasound
- ☐ Calcium level
- ☐ Triglyceride level
- ☐ Liver function test panel
- ☐ Review of medication list for potentially causative drugs



### ☐ Resuscitation

- ☐ Use same strategy as for septic shock (e.g. moderate amount fluid, vasopressors if needed).
- ☐ Avoid large-volume resuscitation (e.g. fluid balance >3-4 liters positive) as this may increase the risk of abdominal compartment syndrome.

### ☐ ERCP

- ☐ Not routinely indicated, but may be considered if evidence of ascending cholangitis or choledolithiasis (e.g., markedly elevated bilirubin, dilation of the common bile duct).

### ☐ Nutrition

- ☐ Non-intubated: Low-fat diet
- ☐ Intubated: Initiate enteral nutrition as soon as hemodynamically stabilized (via either nasogastric or postpyloric feeding tube).

### ☐ Pain control

- ☐ Start with scheduled acetaminophen (e.g. 1 gram Q6hr) and pain-dose ketamine infusion (0.1-0.3 mg/kg/hr).
- ☐ Opioids may worsen ileus, limit them as able.

### ☐ May consider metabolic resuscitation

- ☐ Ascorbic acid 1.5g IV q6hr, thiamine 200 mg IV q12hr, +/- hydrocortisone 50 mg IV q6hr

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## podcast

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## questions & discussion

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To keep this page small and fast, questions & discussion about this post can be found on another page [here \(https://emcrit.org/pulmcrit/pancreatitis/\)](https://emcrit.org/pulmcrit/pancreatitis/).



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- #1 most common error is administration of excessive volumes of fluid, causing ARDS and abdominal compartment syndrome. Unfortunately, this strategy continues to be recommended by many sources.
- Delayed initiation of nutrition, due to a desire to “rest” the pancreas.
- Fear-induced initiation of antibiotics during the first week of therapy, when superinfection is uncommon.
- Meropenem isn't required to penetrate pancreatic tissue, piperacillin-tazobactam also has good penetration.

### Going further:

- Pancreatitis overview
  - [Acute pancreatitis \(http://rebelem.com/acute-pancreatitis/\)](http://rebelem.com/acute-pancreatitis/) (Anand Swaminathan, RebelEM)
  - [Pancreatitis \(https://lifeinthefastlane.com/ccp/pancreatitis/\)](https://lifeinthefastlane.com/ccp/pancreatitis/) (Chris Nickson, LITFL)
- Resus & abdominal compartment syndrome
  - [Pancreatitis: please stop the drowning \(https://emcrit.org/pulmcrit/the-myth-of-large-volume-resuscitation-in-acute-pancreatitis/\)](https://emcrit.org/pulmcrit/the-myth-of-large-volume-resuscitation-in-acute-pancreatitis/) (PulmCrit)
  - [Killer resuscitation: Abdominal hypertension as a cause of multiorgan failure \(https://emcrit.org/pulmcrit/abdominal-hypertension/\)](https://emcrit.org/pulmcrit/abdominal-hypertension/) (PulmCrit)
- [Hypertriglyceridemic pancreatitis \(https://emcrit.org/pulmcrit/hypertriglyceridemic-pancreatitis/\)](https://emcrit.org/pulmcrit/hypertriglyceridemic-pancreatitis/) (PulmCrit)
- [New guidelines simplify ICU nutrition \(https://emcrit.org/pulmcrit/enteral-nutrition-intubated/\)](https://emcrit.org/pulmcrit/enteral-nutrition-intubated/) (PulmCrit)
- [Analgesic ladder for pain control in critically ill patients \(https://emcrit.org/pulmcrit/analgesic-ladder/\)](https://emcrit.org/pulmcrit/analgesic-ladder/) (PulmCrit)

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