



Introduction

Acute pancreatitis is inflammation and destruction of pancreatic tissue. It is a laboratory diagnosis, and a common cause of abdominal pain in patients, occurring in ~20 per 100,000 patients in the USA. There are a multitude of board-relevant causes, 75% of cases are due to gallstones and alcohol. This review covers the presentation, symptoms, diagnosis, and management. Mortality was once 12% several years ago, now 2%.

Pathophysiology

Transient obstruction of pancreatic ducts either by toxic metabolites, stones, or faulty membrane channels leads to stasis and accumulation of deadly pancreatic enzymes released from acinar cells, which eventually activate and cause harm to nearby tissues.

Causes ("GET SMASHED", a mnemonic which we do not take credit for)

Gallstones: Most common cause of acute pancreatitis. only ~5% of patients with gallstones get pancreatitis. Ironically, stones <5mm are much higher risk than larger ones.

EtOH: 2nd most common cause of pancreatitis.

Trauma: handlebar trauma or any direct blunt trauma to the epigastrium. Classically in children.

Steroids

Malignancy/Mumps: cancer, in particular pancreatic, can easily block ducts and cause inflammation. Mumps is super rare in the United States.

Autoimmune: lupus (of course, along with others you don't need to memorize.)

Scorpions: the one everyone remembers but never sees. We will pay you serious cash-money if you email us with a case.

Hypertriglyceridemia: levels typically >1000 mg/dL.

ERCP: a frequent complication of this procedure. Estimates are hard to pinpoint, some 2-15%.

Drugs: tetracyclines, azathioprine, thiazides, valproate, didanosine (HAART NRTI).

Presentation

Most patients present with acute, sudden onset of central abdominal pain, mainly epigastric. RUQ pain may occur, associated with gallstone disease. Pain reaches maximum intensity within the first hour.

50% have pain that radiates to the back. Some have pain that is worse with sitting up, this is not quantified.

90% have nausea/vomiting.

On physical exam, the epigastrium is typically tender to palpation, this can be altered by body habitus. Patients with severe cases can have scleral icterus, fever, tachycardia, tachypnea, and even hypotension. Cullen's Sign (periumbilical bruising) may also be seen, which is nonspecific but suggestive of necrotizing pancreatitis and retroperitoneal bleeding.

Diagnosis

Despite the high association of location of pain associated with nausea/vomiting, labs are needed for confirmation, mainly, just a lipase.

Lipase: sensitivity 82-100%. Needs to be >3x the upper limit elevated. It rises >6 hours after symptom onset, peaks at 24 hours. Lipase rises earlier and lasts longer than amylase.

Bottom line: stop ordering amylases. If you see providers ordering them, call the Pancreatitis Police, 1-800-WASTAGE

Why not amylase?

Amylase historically was used, but it is not as specific for pancreatitis as lipase.

Amylase may be normal in 20% of alcoholic pancreatitis cases and 50% of hypertriglyceridemia cases. It has a short half-life as well, and many cases might be missed if presentation is delayed.

CBC, CMP, urine studies, urine pregnancy test should be ordered as well for general workup of abdominal pain.

Imaging:

RUQ US: doesn't help diagnose pancreatitis but helps diagnose the most common cause of it- gallstones.

Any patient who presents to the ED with first time pancreatitis should undergo a RUQ US. It's a relatively cheap test with no radiation to it and diagnoses a treatable cause of pancreatitis.

CT abd/pelvis with contrast: shows focal or diffuse enlargement of the pancreas with heterogeneous enhancement. *Lack of contrast enhancement is concerning for necrosis.*

MRI with or without contrast: higher sensitivity than CT for early acute pancreatitis. More expensive too.

Patients with recurrent episodes should undergo EUS (endoscopic ultrasound) to evaluate for pancreatic ductal abnormalities, tumors, or microlithiasis in the gallbladder. This is an inpatient test, not ED.

Confirming the diagnosis of pancreatitis:

Requires 2 of the following: *acute onset of persistent epigastric pain, elevated lipase, or CT/MRI imaging confirmation.*

When do we need CT?

Imaging is *not* required in cases where the former two criteria are present. In standard cases with obvious symptoms and the patient is not critically ill, CT does not often lead to changes in decision making.

If patient is in clear shock or if there is diagnostic uncertainty you can consider it. There is no evidence that CT improves clinical outcomes, and most importantly we don't know the full damage extent until often >72 hours.

DDx: cholecystitis, gastritis, choledocholithiasis or cholangitis, peptic ulcer disease, small bowel obstruction, hepatitis

Management

85% of patients with acute pancreatitis have an enlarged, inflamed pancreas with no necrosis. 15% have necrotizing pancreatitis and these people will have a bad day.

Acute interstitial pancreatitis: symptomatic management with aggressive fluid hydration

NPO at first with aggressive IV lactated Ringers and nausea medications. IV fluids reduce mortality. Inadequate hydration leads to kidney injury, worsening vascular leak and hemoconcentration causing ischemic pain and lactic acidosis.

Advance a low-fat diet early on within 24 hours if patient wishes to eat and is non-toxic appearing.

Recovery in 3-5 days depending on cause.

20% progress to more severe organ failure.

Prophylactic antibiotics are *not* indicated.

Gallstone pancreatitis: ERCP in 24 hours if obvious CBD obstruction. If it isn't obvious, EUS or MRCP needs to happen.

Complications

Peripancreatic fluid collection: no defined wall and are often asymptomatic. Rare need for drainage.

Pseudocyst collection: encapsulated fluid collection outside the pancreas with minimal necrosis. Occurs >4 weeks after. Most are observed, some need endoscopic drainage.

Acute necrosis and walled-off necrosis: usually with polymicrobial infections.

- Antibiotics to consider: carbapenem, quinolone, ceftazidime, cefepime + metronidazole.

Prognosis

Overall mortality is 5% for all patients, 3% for acute interstitial and 17% for necrotizing.

Recurrent attacks increase the risk for chronic pancreatitis.

Predicting severity is something we've been trying to do for years, as some patients will recover and go home in days, others will be in the ICU and die in days.

There are many scoring criteria, but none are perfect. Ranson's and APACHE II are the most common, only Ranson's is on EM boards.

Ranson's Criteria: assess 5 factors at admission (see table to the right), reassess 6 factors at the next 48 hours. Meta-analysis has shown this score to suck big time. It's a poor predictor of severity.

The bad news is you *need* to know the first part of Ranson's Criteria for boards. The good news is you do not need to know the 48-hour reassessment part (we've never seen a board question on that). Memorize the table to the right for boards. We don't care if you use it clinically or not (we don't).

Age	>55
WBC	>16k
Glucose	>200 mg/dL
LDH	>350 U/L
AST	>250 U/L

APACHE II (Acute Physiology and Chronic Health Examination): developed for critically ill patients in the ICU, *not ED*. Good negative predictive value and modest positive predictive value. It can be performed daily.

References

1. Opie EL. The etiology of acute hemorrhagic pancreatitis. Bull Johns Hopkins Hosp 1901; 12:182.
2. Steer ML. Pathogenesis of acute pancreatitis. Digestion 1997; 58 Suppl 1:46.
3. Singh VK, Bollen TL, Wu BU, et al. An assessment of the severity of interstitial pancreatitis. Clin Gastroenterol Hepatol 2011; 9:1098.
4. van Santvoort HC, Bakker OJ, Bollen TL, et al. A conservative and minimally invasive approach to necrotizing pancreatitis improves outcome. Gastroenterology 2011; 141:1254.
5. Working Group IAP/APA Acute Pancreatitis Guidelines. IAP/APA evidence-based guidelines for the management of acute pancreatitis. Pancreatology 2013; 13:e1.
6. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. Gut 2013; 62:102.
7. Eckerwall GE, Tingstedt BB, Bergenzaun PE, Andersson RG. Immediate oral feeding in patients with mild acute pancreatitis is safe and may accelerate recovery--a randomized clinical study. Clin Nutr 2007; 26:758.
8. Li J, Xue GJ, Liu YL, et al. Early oral refeeding wisdom in patients with mild acute pancreatitis. Pancreas 2013; 42:88.
9. Banks PA, Freeman ML, Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. Am J Gastroenterol 2006; 101:2379.

Super Smashed: Guide to Pancreatitis

Twitter/Instagram: [@emboardbombs](#)

10. Beger HG, Bittner R, Block S, Büchler M. Bacterial contamination of pancreatic necrosis. A prospective clinical study. *Gastroenterology* 1986; 91:433.
11. Bradley EL 3rd, Allen K. A prospective longitudinal study of observation versus surgical intervention in the management of necrotizing pancreatitis. *Am J Surg* 1991; 161:19.
12. Banks PA, Freeman ML, Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. *Am J Gastroenterol* 2006; 101:2379.
13. Larvin M. Assessment of clinical severity and prognosis. In: *The Pancreas*, Beger HG, Warshaw AL, Buchler MW, et al (Eds), Blackwell Science, Oxford 1998. p.489.
14. Bollen TL, Singh VK, Maurer R, et al. A comparative evaluation of radiologic and clinical scoring systems in the early prediction of severity in acute pancreatitis. *Am J Gastroenterol* 2012; 107:612.
15. Lecesne R, Taourel P, Bret PM, et al. Acute pancreatitis: interobserver agreement and correlation of CT and MR cholangiopancreatography with outcome. *Radiology* 1999; 211:727.
16. Ranson JH, Rifkind KM, Roses DF, et al. Prognostic signs and the role of operative management in acute pancreatitis. *Surg Gynecol Obstet* 1974; 139:69.
17. Ranson JH. The timing of biliary surgery in acute pancreatitis. *Ann Surg* 1979; 189:654.
18. De Bernardinis M, Violi V, Roncoroni L, et al. Discriminant power and information content of Ranson's prognostic signs in acute pancreatitis: a meta-analytic study. *Crit Care Med* 1999; 27:2272.
19. Forsmark CE, Baillie J, AGA Institute Clinical Practice and Economics Committee, AGA Institute Governing Board. AGA Institute technical review on acute pancreatitis. *Gastroenterology* 2007; 132:2022.
20. Riela A, Zinsmeister AR, Melton LJ, DiMugno EP. Etiology, incidence, and survival of acute pancreatitis in Olmsted County, Minnesota. *Gastroenterology* 1991; 100:A296.
21. Venneman NG, Renooij W, Rehfeld JF, et al. Small gallstones, preserved gallbladder motility, and fast crystallization are associated with pancreatitis. *Hepatology* 2005; 41:738.
22. Yang AL, Vadavakar S, Singh G, Omary MB. Epidemiology of alcohol-related liver and pancreatic disease in the United States. *Arch Intern Med* 2008; 168:649.
23. Nawaz H, Koutroumpakis E, Easler J, et al. Elevated serum triglycerides are independently associated with persistent organ failure in acute pancreatitis. *Am J Gastroenterol* 2015; 110:1497.
24. Kahaleh M, Freeman M. Prevention and management of post-endoscopic retrograde cholangiopancreatography complications. *Clin Endosc* 2012; 45:305.
25. Sadr-Azodi O, Mattsson F, Bexlius TS, et al. Association of oral glucocorticoid use with an increased risk of acute pancreatitis: a population-based nested case-control study. *JAMA Intern Med* 2013; 173:444.
26. Singh S, Chang HY, Richards TM, et al. Glucagonlike peptide 1-based therapies and risk of hospitalization for acute pancreatitis in type 2 diabetes mellitus: a population-based matched case-control study. *JAMA Intern Med* 2013; 173:534.
27. Swaroop VS, Chari ST, Clain JE. Severe acute pancreatitis. *JAMA* 2004; 291:2865.
28. Banks PA, Freeman ML, Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. *Am J Gastroenterol* 2006; 101:2379.
29. Mookadam F, Cikes M. Images in clinical medicine. Cullen's and Turner's signs. *N Engl J Med* 2005; 353:1386.
30. Yadav D, Agarwal N, Pitchumoni CS. A critical evaluation of laboratory tests in acute pancreatitis. *Am J Gastroenterol* 2002; 97:1309.
31. Fortson MR, Freedman SN, Webster PD 3rd. Clinical assessment of hyperlipidemic pancreatitis. *Am J Gastroenterol* 1995; 90:2134.
32. Frank B, Gottlieb K. Amylase normal, lipase elevated: is it pancreatitis? A case series and review of the literature. *Am J Gastroenterol* 1999; 94:463.
33. Gwozdz GP, Steinberg WM, Werner M, et al. Comparative evaluation of the diagnosis of acute pancreatitis based on serum and urine enzyme assays. *Clin Chim Acta* 1990; 187:243.
34. Lecesne R, Taourel P, Bret PM, et al. Acute pancreatitis: interobserver agreement and correlation of CT and MR cholangiopancreatography with outcome. *Radiology* 1999; 211:727.