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

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.
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).

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