

# Morning Report

7/3/18

TEAM 1

# HPI

- ▶ 38 year old female with history of hypertension and PTSD presents to the ED with c/o three weeks of intermittent epigastric abdominal pain described as similar to "hunger pains", worse with supine, better with upright. Today, it is burning and worse with spicy foods.
- ▶ Associated with two days of decreased appetite and one day of non-bloody, non-bilious vomiting. Also, endorses constipation X 5 days.
- ▶ Pertinent negatives: No fever, no chest pain, no diarrhea, no melena or hematochezia, no dysuria.

# Physical Exam

- ▶ Vitals: Temp 98.4, HR 72, RR 18, BP 154/98
- ▶ General: Awake, alert, well appearing
- ▶ CV: Normal rate, regular rhythm, no murmurs
- ▶ Lungs: CTAB, no wheezes or rales
- ▶ Abd: Soft, + BS, no guarding or rigidity, tender epigastric area
- ▶ Ext: No edema
- ▶ Skin: warm and dry

Differential?

# labs/studies

- ▶ WBC 9.8
- ▶ Hgb 12.2
- ▶ Hct 35.8
- ▶ Plt 375
- ▶ Creatinine 0.63
- ▶ BUN 8
- ▶ Lipase 100
- ▶ Calcium 9.5
- ▶ Alk phos 53, T bili 0.7, AST 36, ALT 22
- ▶ UA unremarkable
- ▶ LA 1.7
- ▶ Lipid: Chol 154, Trig 170, LDL 47, HDL 73
- ▶ CXR unremarkable
- ▶ EKG NSR without ischemic changes
- ▶ RUQ US
  - ▶ No cholelithiasis or biliary ductal dilatation
- ▶ CT abdomen/pelvis with IV contrast
  - ▶ Pancreatic inflammation (head, peripancreatic area), hypoattenuation suggestive of edema/early necrosis
  - ▶ Gallbladder mildly thickened without gallstones

# Acute Pancreatitis

# American College of Gastroenterology Guideline: Management of Acute Pancreatitis

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**This guideline presents recommendations for the management of patients with acute pancreatitis (AP). During the past decade, there have been new understandings and developments in the diagnosis, etiology, and early and late management of the disease. As the diagnosis of AP is most often established by clinical symptoms and laboratory testing, contrast-enhanced computed tomography (CECT) and/or magnetic resonance imaging (MRI) of the pancreas should be reserved for patients in whom the diagnosis is unclear or who fail to improve clinically. Hemodynamic status should be assessed immediately upon presentation and resuscitative measures begun as needed. Patients with organ failure and/or the systemic inflammatory response syndrome (SIRS) should be admitted to an intensive care unit or intermediary care setting whenever possible. Aggressive hydration should be provided to all patients, unless cardiovascular and/or renal comorbidities preclude it. Early aggressive intravenous hydration is most beneficial within the first 12–24 h, and may have little benefit beyond. Patients with AP and concurrent acute cholangitis should undergo endoscopic retrograde cholangiopancreatography (ERCP) within 24 h of admission. Pancreatic duct stents and/or postprocedure rectal nonsteroidal anti-inflammatory drug (NSAID) suppositories should be utilized to lower the risk of severe post-ERCP pancreatitis in high-risk patients. Routine use of prophylactic antibiotics in patients with severe AP and/or sterile necrosis is not recommended. In patients with infected necrosis, antibiotics known to penetrate pancreatic necrosis may be useful in delaying intervention, thus decreasing morbidity and mortality. In mild AP, oral feedings can be started immediately if there is no nausea and vomiting. In severe AP, enteral nutrition is recommended to prevent infectious complications, whereas parenteral nutrition should be avoided. Asymptomatic pancreatic and/or extrapancreatic necrosis and/or pseudocysts do not warrant intervention regardless of size, location, and/or extension. In stable patients with infected necrosis, surgical, radiologic, and/or endoscopic drainage should be delayed, preferably for 4 weeks, to allow the development of a wall around the necrosis.**

# Etiologies

- ▶ I – Idiopathic
- ▶ G – Gallstones
- ▶ E – Ethanol – 5 yrs, 50 g
- ▶ T – Trauma
- ▶ S – Steroids
- ▶ M – Mumps, malignancy
- ▶ A – Autoimmune
- ▶ S – Scorpion sting
- ▶ H – Hypercalcemia, Hypertriglyceridemia –1-4%
- ▶ E – ERCP
- ▶ D – Drugs (HCTZ, Sulfa)

**Table 1. Causes of Acute Pancreatitis.\***

Cause	Approximate Frequency	Diagnostic Clues	Comments
Gallstones	40%	Gallbladder stones or sludge, abnormal liver-enzyme levels	Endoscopic ultrasonography can reveal very small gallbladder or duct stones.
Alcohol	30%	Acute flares superimposed on underlying chronic pancreatitis	Diagnosis rests on history, obtained with CAGE questions.†
Hypertriglyceridemia	2–5%	Fasting triglycerides >1000 mg/dl (11.3 mmol per liter)	
Genetic causes	Not known	Recurrent acute pancreatitis and chronic pancreatitis	
Drugs	<5%	Other evidence of drug allergy (e.g., rash) only in rare cases	The condition is idiosyncratic and usually mild.
Autoimmune cause	<1%	Type 1: obstructive jaundice, elevated serum IgG4 levels, response to glucocorticoids; type 2: possible presentation as acute pancreatitis; occurrence in younger patients; no IgG4 elevation; response to glucocorticoids	Type 1 is a systemic disease affecting the pancreas, salivary glands, and kidneys; in type 2, only the pancreas is affected.
ERCP	5–10% (among patients undergoing ERCP)		The symptoms can be reduced with rectal NSAIDs (diclofenac or indomethacin) or temporary placement of a stent in the pancreatic duct.
Trauma	<1%	Blunt or penetrating trauma, particularly in midbody of pancreas as it crosses spine	
Infection	<1%	Viruses: CMV, mumps, and EBV most common; parasites: ascaris and clonorchis	
Surgical complication	5–10% (among patients undergoing cardiopulmonary bypass)		The condition is probably due to pancreatic ischemia; pancreatitis may be severe.
Obstruction	Rare	Celiac disease and Crohn's disease, pancreas divisum (controversial), and sphincter of Oddi dysfunction (very controversial)	On rare occasions, malignant pancreatic duct or ampullary obstruction is seen.
Associated conditions	Common	Diabetes, obesity, and smoking	

\* CMV denotes cytomegalovirus, EBV Epstein-Barr virus, ERCP endoscopic retrograde cholangiopancreatography, and NSAIDs nonsteroidal antiinflammatory drugs.

† CAGE is an acronym for the following questions: Have you ever felt you should cut down on your drinking? Have people annoyed you by criticizing your drinking? Have you ever felt bad or guilty about your drinking? Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (eye opener)?



# Classification of acute pancreatitis

**Table 3.** Definitions of severity in acute pancreatitis: comparison of Atlanta and recent revision

<b>Atlanta criteria (1993)</b>	<b>Atlanta Revision (2013)</b>
<b>Mild acute pancreatitis</b>	<b>Mild acute pancreatitis</b>
Absence of organ failure	Absence of organ failure
Absence of local complications	Absence of local complications
<b>Severe acute pancreatitis</b>	Moderately severe acute pancreatitis
1. Local complications <b>AND/OR</b>	1. Local complications <b>AND/OR</b>
2. Organ failure	2. Transient organ failure (< 48 h)
GI bleeding (> 500 cc/24 hr)	<b>Severe acute pancreatitis</b>
Shock – SBP ≤ 90 mm Hg	Persistent organ failure > 48 h <sup>a</sup>
PaO <sub>2</sub> ≤ 60%	
Creatinine ≥ 2 mg/dl	
GI, gastrointestinal; SBP, systolic blood pressure.	
<sup>a</sup> Persistent organ failure is now defined by a Modified Marshal Score (6,8)	

# Diagnosis

- ▶ Presence of two of the three: (Strong recommendation, moderate quality of evidence)
  - ▶ 1. Abdominal pain consistent with the disease
  - ▶ 2. Serum amylase/lipase > 3X ULN
  - ▶ 3. Characteristic findings on abdominal imaging
- ▶ Contrast enhanced CT and MRI of the pancreas should be reserved for patients where the diagnosis is unclear or those who fail to improve after 48-72 hours (strong recommendation, low quality of evidence)

# Diagnosis

- ▶ Transabdominal ultrasound should be performed in all patients with acute pancreatitis (Strong recommendation, low quality)
- ▶ Obtain triglycerides in the absence of gallstones and significant alcohol use. (conditional recommendation)
  - ▶ Considered etiology if  $> 1000$  mg/dl
- ▶ Consider pancreatic tumor if  $> 40$  years of age -CECT/MRI
- ▶ Limit endoscopic investigation in patients with acute idiopathic pancreatitis
- ▶ Consider genetic testing in young ( $<30$ ) if no obvious cause and if family hx pancreatic disease

**Table 4.** Clinical findings associated with a severe course for initial risk assessment<sup>a</sup>

<i>Patient characteristics</i>
Age >55 years (53,57)
Obesity (BMI >30 kg/m <sup>2</sup> ) (68)
Altered mental status (69)
Comorbid disease (53)
<i>The systemic inflammatory response syndrome (SIRS) (6,53,54,70,71) Presence of &gt;2 of the following criteria:</i>
– pulse >90 beats/min
– respirations >20/min or PaCO <sub>2</sub> >32 mm Hg
– temperature >38 °C or <36 °C
– WBC count >12,000 or <4,000 cells/mm <sup>3</sup> or >10% immature neutrophils (bands)
<i>Laboratory findings</i>
BUN >20 mg/dl (63)
Rising BUN (63)
HCT >44% (62)
Rising HCT (62)
Elevated creatinine (72)
<i>Radiology findings</i>
Pleural effusions (73)
Pulmonary infiltrates (53)
Multiple or extensive extrapancreatic collections (67)
BMI, body mass index; BUN, blood urea nitrogen; HCT, hematocrit; WBC, white blood cell.
<sup>a</sup> The presence of organ failure and/or pancreatic necrosis defines severe acute pancreatitis.

# Initial management

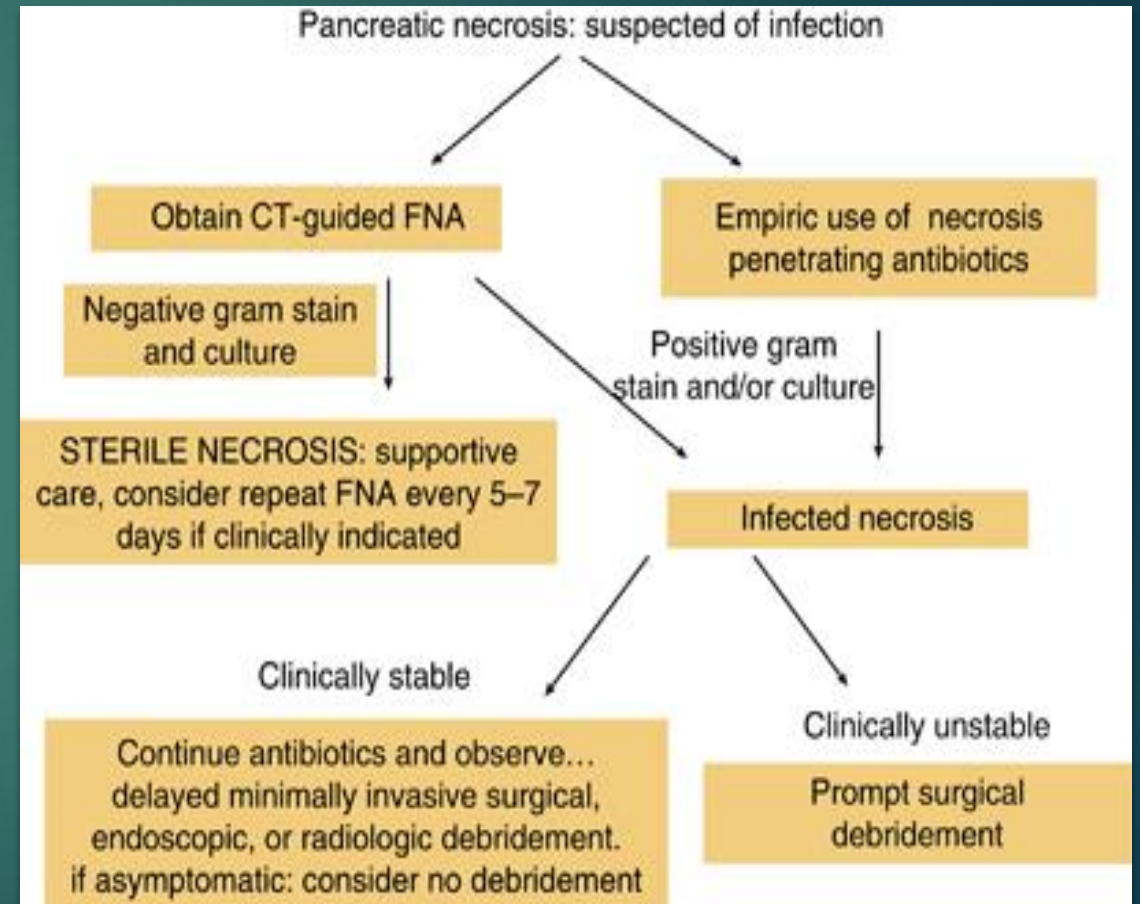
- ▶ Aggressive IV hydration 250-500 cc/hr of isotonic crystalloid soln for all pts unless limited by cardiovascular/renal issues (Strong recommendation, moderate quality)
  - ▶ Most beneficial for first 12-24 hours
  - ▶ Lactated ringer's may be preferred
  - ▶ Goal is decrease in BUN
  - ▶ Assess frequently in the first 6 hours
  - ▶ If volume depleted, needs bolus
- ▶ Nutrition
- ▶ Antibiotics
- ▶ Surgery

# Nutrition in Acute Pancreatitis

- ▶ In mild AP, oral feedings can be started immediately if there is no N/V - shorter hospital stay
- ▶ In mild AP, oral feedings with low-fat solid diet appears as safe as a clear liquid diet
- ▶ In severe AP, Enteral route preferred to prevent infectious complications. Parenteral route should be avoided unless no other options

# Role of antibiotics

- ▶ Should be given for extrapancreatic infections:
  - ▶ Cholangitis, catheter-related infections, bacteremia, UTI, PNA
- ▶ Should not be given routinely in severe acute pancreatitis
- ▶ Should not be given in sterile necrosis to prevent development of infected necrosis
- ▶ Consider infected necrosis:
  - ▶ Pts who deteriorate or fail to improve after 7-10 days hospitalization
  - ▶ CT guided FNA with gram stain + culture to guide ABX or empiric ABX
- ▶ In patients with infected necrosis, antibiotics known to penetrate pancreatic necrosis, such as carbapenems, quinolones, and metronidazole, may be useful in delaying or sometimes totally avoiding intervention



# Role of surgery

- ▶ In mild AP, found to have gallstones, need cholecystectomy prior to discharge to prevent recurrence
- ▶ In necrotizing biliary AP, defer cholecystectomy until inflammation resolves to prevent infection
- ▶ Asymptomatic pseudocysts and pancreatic and/or extrapancreatic necrosis do not warrant intervention regardless of size, location, and/or extension
- ▶ In stable pts with infected necrosis, delay for 4 weeks to allow for walled off necrosis
- ▶ In symptomatic infected necrosis, minimally invasive preferred to open-invasive



# Key teaching points

- ▶ Need two of three for diagnosis (clinical picture, Lipase > 3X ULN, Imaging findings)
- ▶ Transabdominal ultrasound should be performed in all patients with acute pancreatitis
- ▶ Obtain triglycerides in the absence of gallstones and significant alcohol use.
- ▶ In mild AP, consider not delaying oral feedings if there is no N/V
- ▶ In mild AP, found to have gallstones, need cholecystectomy prior to discharge to prevent recurrence
- ▶ In necrotizing biliary AP, defer cholecystectomy until inflammation resolves to prevent infection

# References

- ▶ American College of Gastroenterology. Management of Acute Pancreatitis. Accessed July 1st, 2018. [http://gi.org/guideline/acute-pancreatitis/?utm\\_medium=referral&utm\\_source=r360&utm\\_campaign=gastroenterology](http://gi.org/guideline/acute-pancreatitis/?utm_medium=referral&utm_source=r360&utm_campaign=gastroenterology)
- ▶ New England Journal of Medicine