Acute Pancreatitis

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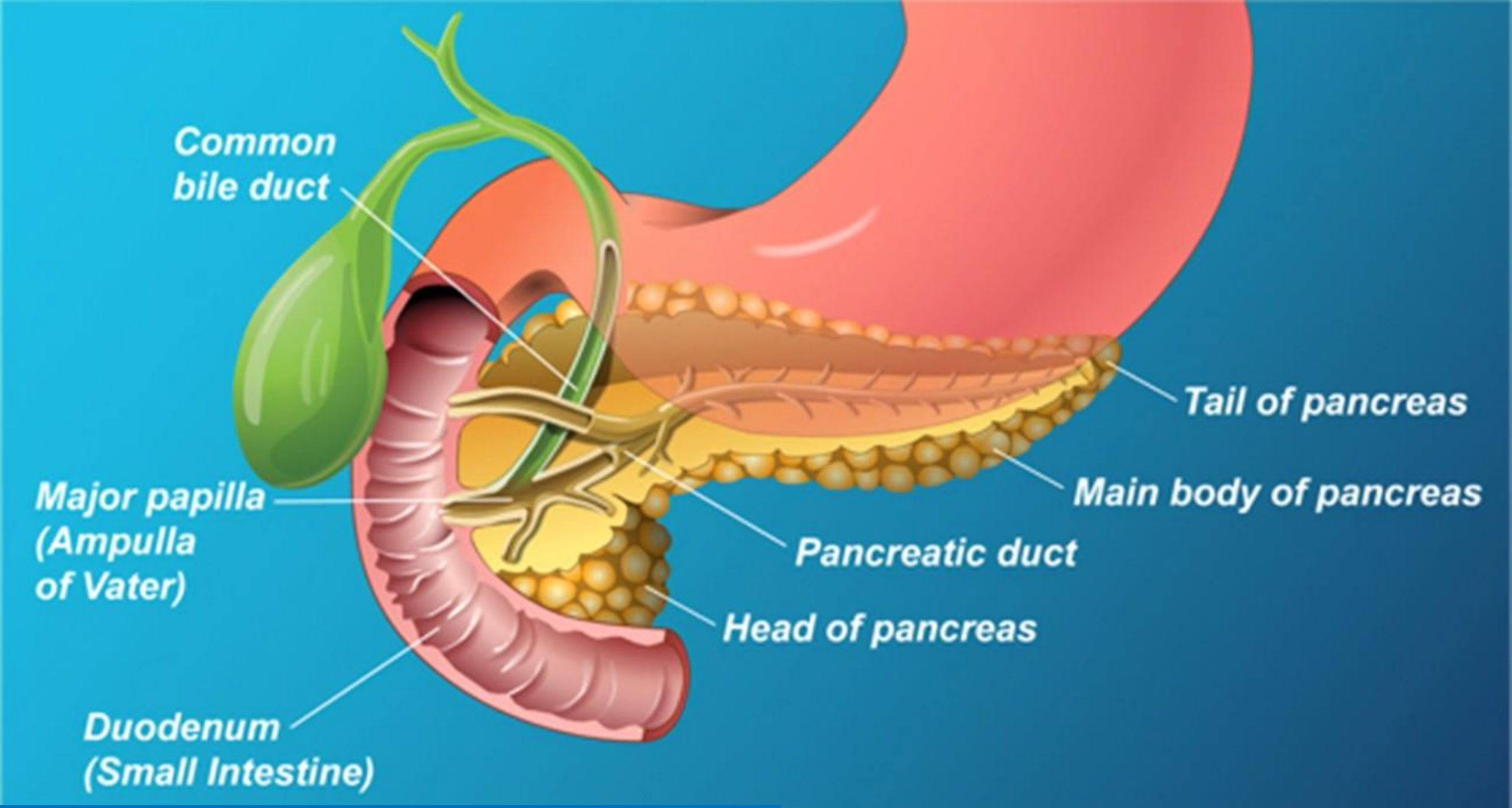
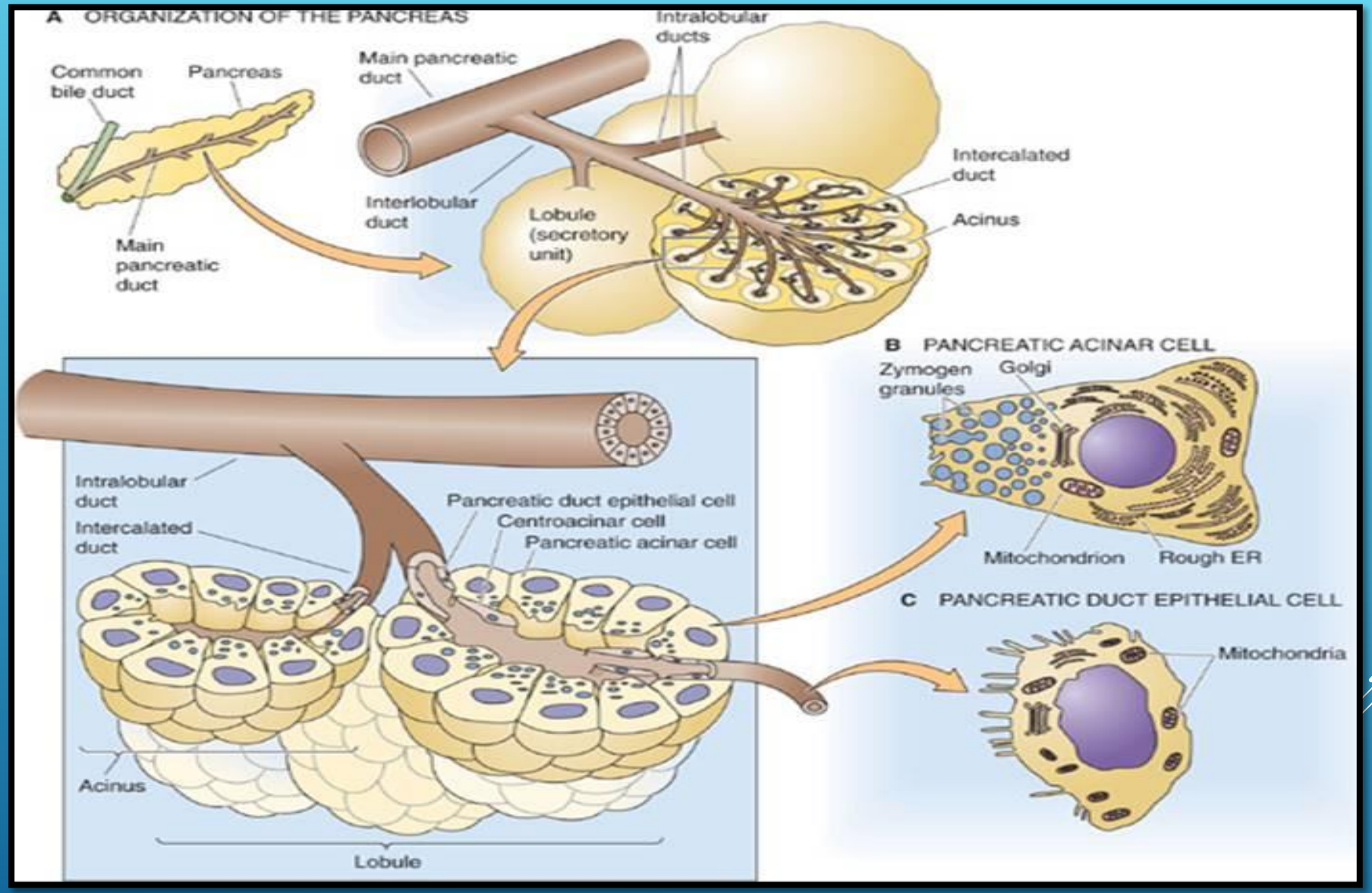
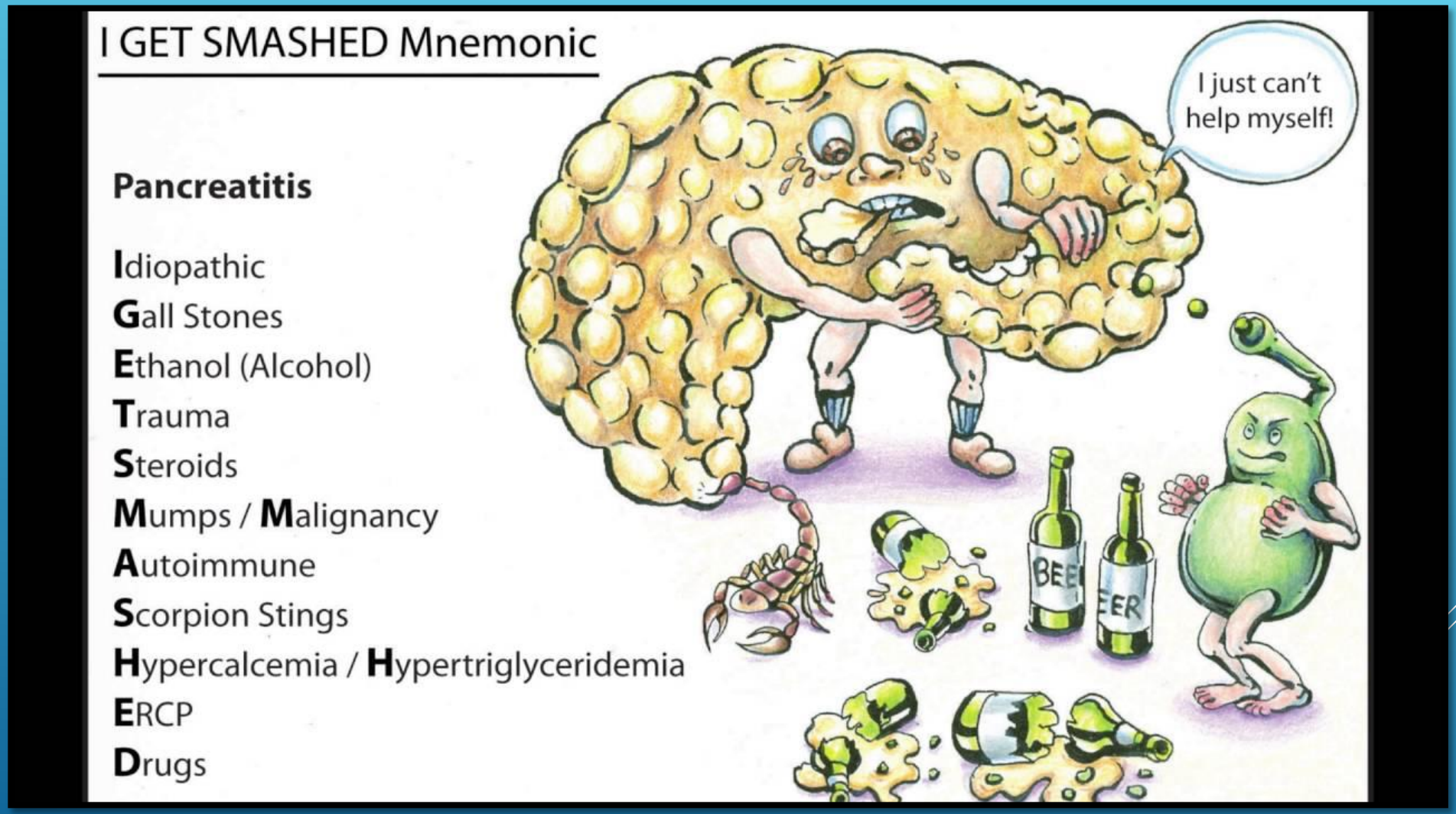


Gross anatomy of the pancreatic ductal system


A ORGANIZATION OF THE PANCREAS
Common bile duct
Pancreas
Main pancreatic duct
Main pancreatic duct
Interlobular duct
Intralobular ducts
Lobule (secretory unit)
Intercalated duct
Acinus
Intralobular duct
Intercalated duct
Pancreatic duct epithelial cell
Centroacinar cell
Pancreatic acinar cell
Acinus
Lobule
B PANCREATIC ACINAR CELL
Zymogen granules
Golgi
Mitochondrion
Rough ER
C PANCREATIC DUCT EPITHELIAL CELL
Mitochondria

Etiology


I GET SMASHED Mnemonic
Pancreatitis
Idiopathic
Gall Stones
Ethanol (Alcohol)
Trauma
Steroids
Mumps / Malignancy
Autoimmune
Scorpion Stings
Hypercalcemia / Hypertriglyceridemia
ERCP
Drugs
I just can't help myself!
BEE
EER

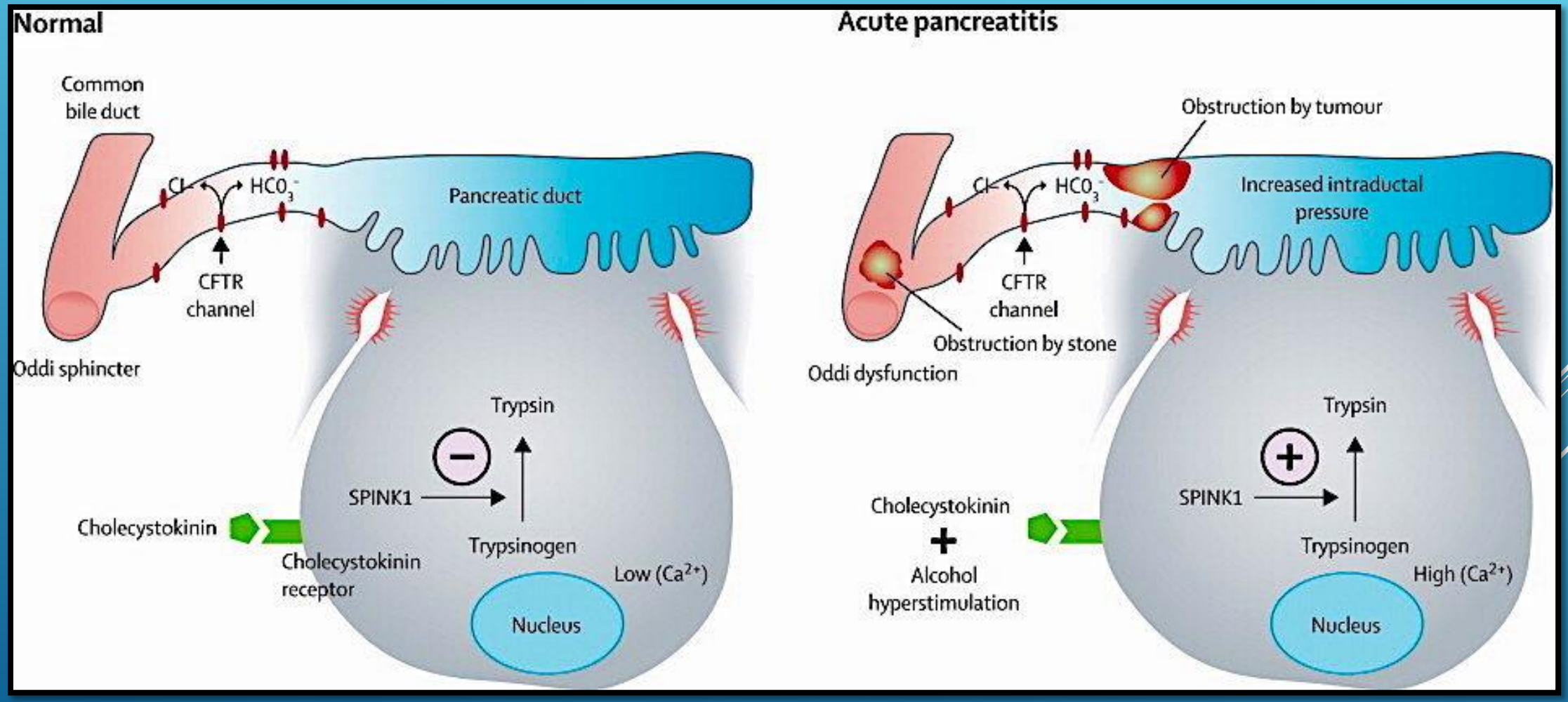
There are many causes of acute pancreatitis, which can be easily identified in 75%-85% of patients. In developed countries, obstruction of the common bile duct by stones (38%) and alcohol abuse (36%) are the most frequent causes of acute pancreatitis. Gallstone-induced pancreatitis is caused by duct obstruction by gallstone migration. Obstruction is localized in the bile duct and pancreatic duct, or both. Duct obstruction promotes pancreatitis by increasing duct pressure and subsequent unregulated activation of digestive enzymes. Alcohol abuse is the second most frequent cause of acute pancreatitis, but the correlation between alcohol and pancreatitis is not completely understood

The etiology and pathogenesis of acute pancreatitis have been intensively investigated, but the pathogenetic theories are controversial. The predominant theories of acute biliary pancreatitis are common pathway theory and gallstone migration theory, which consent that the key factor for acute biliary pancreatitis is *bile-pancreatic duct obstruction*, which *increases pancreatic duct pressure*, *bile reflux, trypsin activation and pancreatic auto-digestion.* Acute pancreatitis occurs when intracellular protective mechanisms to prevent trypsinogen activation or reduce trypsin activity are overwhelmed. However, these theories are controversial.

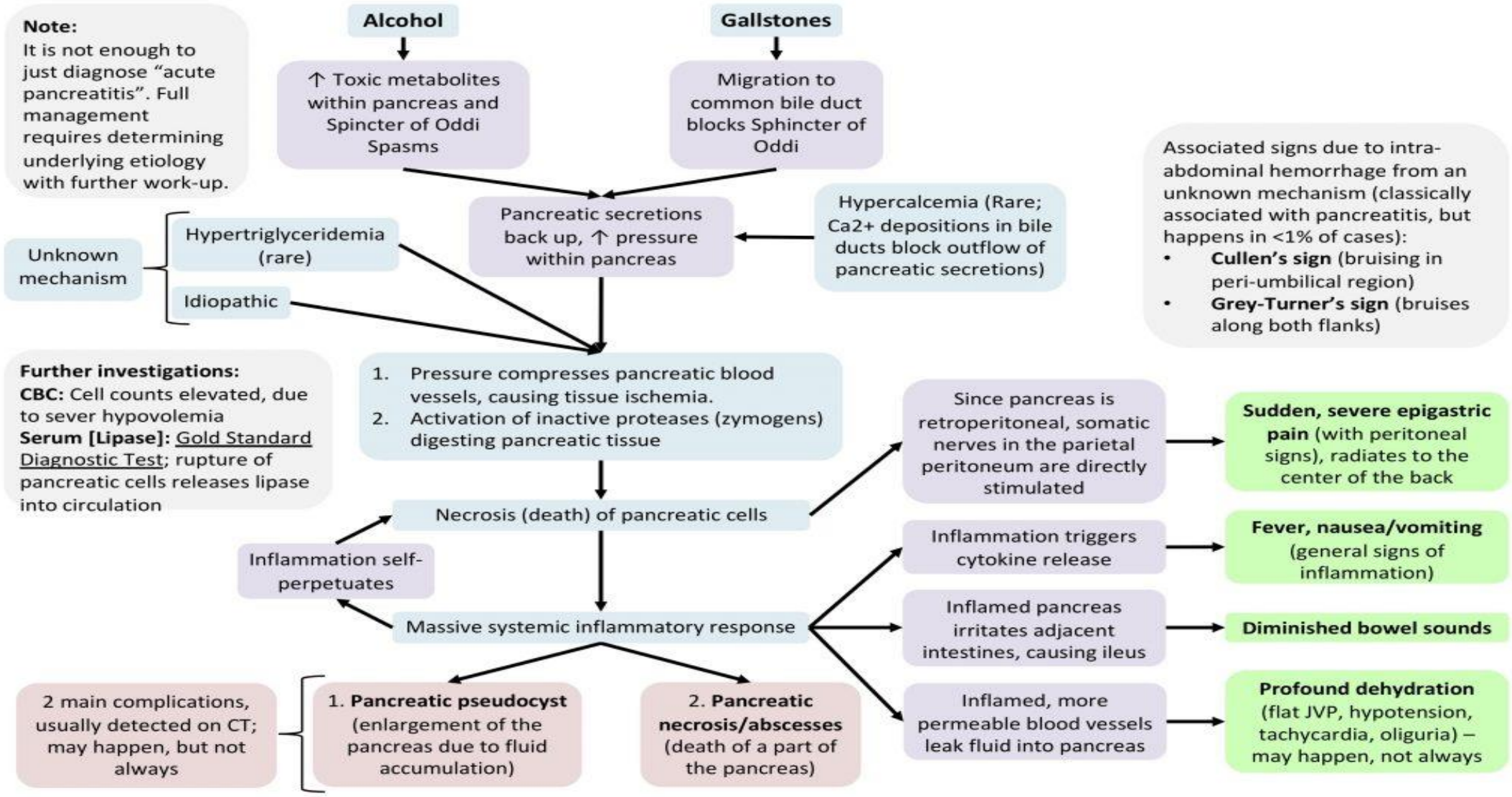
Reference:

Wang GJ, Gao CF, Wei D, Wang C, Ding SQ. Acute pancreatitis: etiology and common pathogenesis. *World J Gastroenterol.* 2009;15(12):1427-1430. doi:10.3748/wjg.15.1427

Pathophysiology of acute pancreatitis



Acute Pancreatitis: Pathogenesis and Clinical Findings



Legend: Pathophysiology | Mechanism | Sign/Symptom/Lab Finding | Complications

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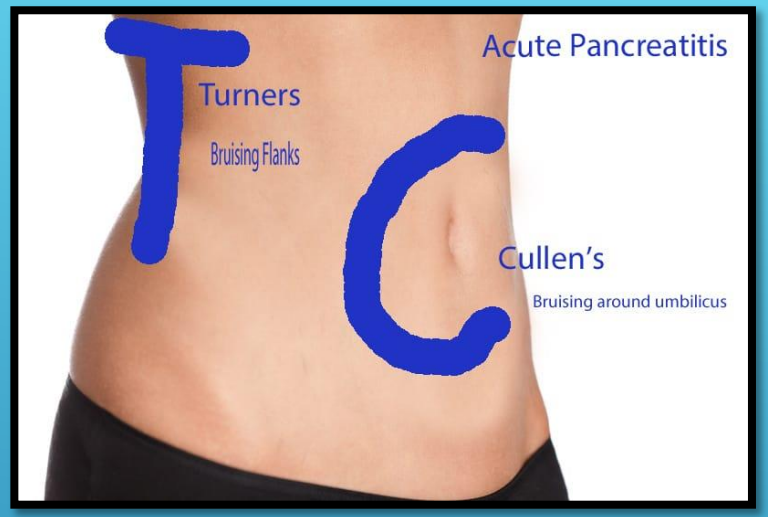
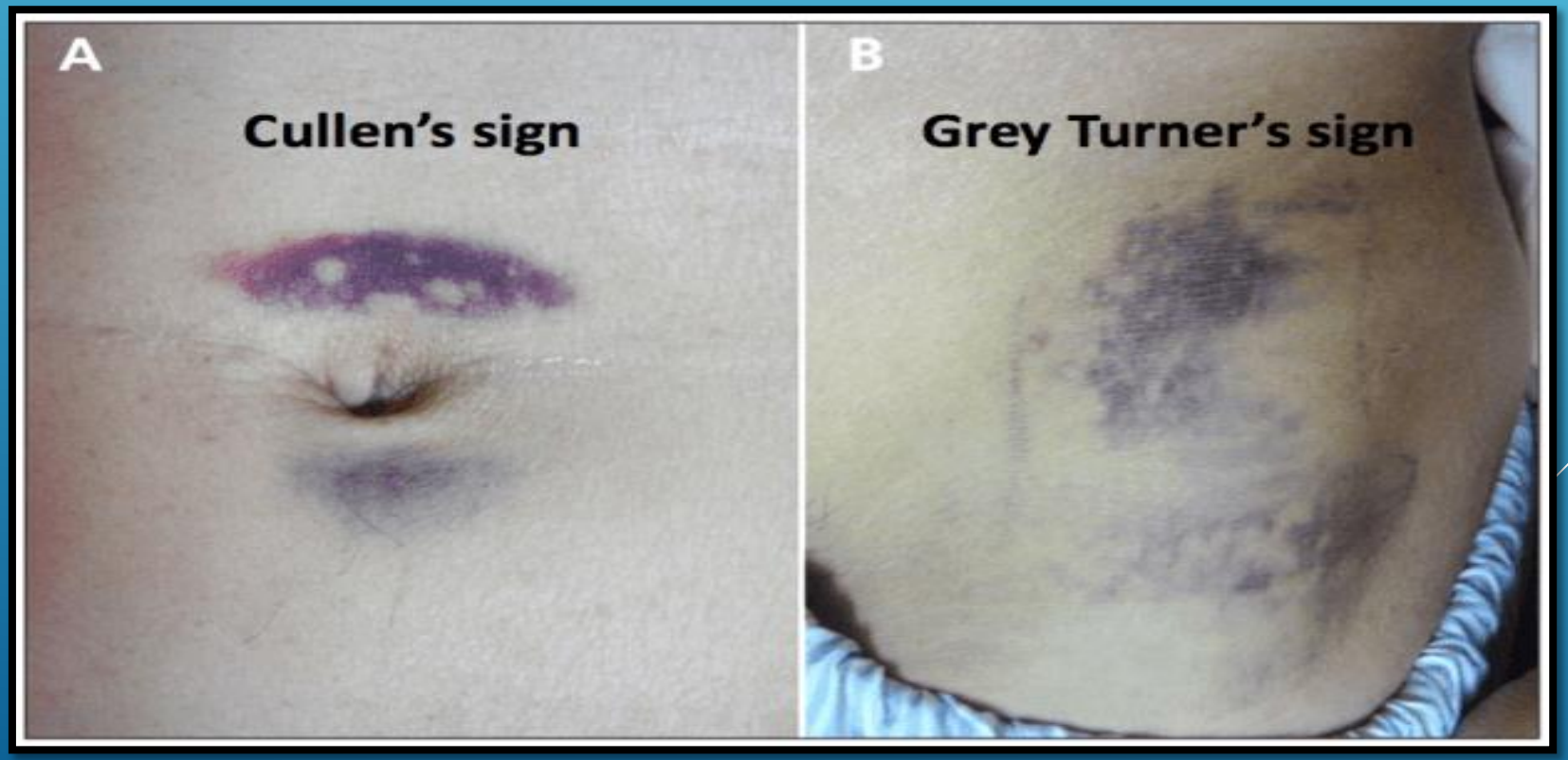


Cullen sign appears due to collection of blood in subcutaneous tissue around umbilicus. Cullen sign has been described in acute pancreatitis, rectus sheath hematoma, splenic rupture, perforated ulcer, intra-abdominal cancer, and acute ruptured ectopic pregnancy, and as a complication of anticoagulation . Cullen's sign may also be seen in any condition in which there is pigmented peritoneal fluid, such as cases of retroperitoneal bleeds in ectopic endometriosis or acute abdominal aortic aneurysmal rupture . Cullen sign is seen in approximately 3 per cent of patients and is associated with a mortality of 37%



Acute pancreatitis is difficult to diagnose due to several other disorders presenting with similar laboratory and clinical symptoms. **The primary diagnostic marker for acute pancreatitis is serum lipase**. Serum lipase levels increase within 4 to 8 hours of clinical symptom onset and decrease within 8 to 14 days. Serum amylase levels are elevated as well, but are ***not an indicator of severity or specificity of the disease***. Serum trypsin levels are very specific in indicating pancreatitis, but the test is not widely available. Severity can be predicted and measured by C-reactive protein, procalcitonin, blood urea nitrogen (BUN), and **Bedside Index of Severity in Acute Pancreatitis** (McCance & Huether, 2014)

Bedside Index for Severity of Acute Pancreatitis (BISAP)

- BUN > 25
- Impaired mental status
- SIRS (> 2 criteria)
- Age > 60 yrs
- Pleural effusion on CT scan
- 1 point for the presence of each finding.

BUN, blood urea nitrogen; SIRS, systemic inflammatory response syndrome

BISAP Score	Observed Mortlity
0	0.1%
1	0.4%
2	1.6%
3	3.6%
4	7.4%
5	9.5%

Adapted from: Wu BU, Johannes RS, Sun X, et al. The early prediction of mortality in acute pancreatitis: A large population-based study. *Gut* 2008;57:1698-1703.

Revised Atlanta Definitions and Classifications of Acute Pancreatitis

Diagnosis requires 2 out of the following 3

1. **Abdominal pain consistent with AP (Epigastric radiating to the back)**
2. **Serum Lipase or Amylase at least 3 times normal**
3. **Characteristic findings on imaging studies (contrast enhanced CT)**

**** If the diagnosis is made based on 1 and 2, CT is not required on admission.***

Markers used in clinical practice to assess the severity of AP in the 1st 3 days

1. Ranson's criteria
2. Glasgow criteria
3. APACHE II Score
4. CT severity index

All are of limited value in predicting persistent organ failure, the development of pancreatic necrosis and death

Amylase and Lipase are not severity markers

Severity markers that become elevated within 24hrs: serum neutrophila elastase, IL-6, and urinary product of trypsinogen activation (TAP). However, these assays are not widely available.

Management of AP

I. Correct Physiologic derangements and ameliorate symptoms

- **Fluid resuscitation**
- **Pain relief (use narcotics- no evidence that they exacerbate the attack by increasing pressure in sphincter of Oddi**
- **NG tube only if vomiting is prominent (not routine)**

II. Minimize the progression of pancreatic injury: NPO (rest the bowel to rest the pancreas)

III. Nutritional support; only in Acute Severe Pancreatitis

IV. No role for antibiotics in mild acute pancreatitis. However, some advocate it for biliary acute pancreatitis.

Main indication for the use of antibiotics is with infectious complications

V. Treat the underlying cause:

- **Gallstone pancreatitis- Lap Cholecystectomy**
- **Hypercalcemia induced- correct Hypercalcemia**
- **Drug induced- D/C Drug**
- **Hyperlipidemia induced- Plasmapheresis**

1. Which of the following is considered a “hallmark” symptom of pancreatitis?

A. Vomiting
B. Mid-epigastric pain that radiates to the back
C. Gradual onset of pain
D. Alcohol consumption

2. A 54-year-old patient admitted with diabetes mellitus, malnutrition, osteomyelitis, and alcohol abuse has a serum amylase level of 280 U/L and a serum lipase level of 310 U/L. To what diagnosis does the nurse attribute these findings?

A. Malnutrition
B. Osteomyelitis
C. Alcohol abuse
D. Diabetes mellitus

3. Which of the following can be used in the treatment of <u>acute</u> pancreatitis? *Select all that apply.*

A. Parenteral fluids
B. Corticosteroids
C. Nasogastric suctioning
D. H2 receptor antagonists
E. Narcotics or Demerol
F. Surgical resection of the pancreas

4. Which lab test result would indicate that Mr. Walker has acute pancreatitis?

A. Elevated bilirubin levels
B. Elevated serum lipase and amylase levels
C. Low white blood cell count
D. Elevated blood alcohol level

5. Why is the body more susceptible to secondary infections during cases of pancreatitis?

A. The production of anti-inflammatory cytokines and specific cytokine inhibitors
B. The formation of pseudocysts
C. The release of cortisol in response to stress
D. The activation of leukocytes in response to pro-inflammatory cytokine production

Quiz Answers with Rationale

1. Which of the following is considered a "hallmark" symptom of pancreatitis?

A. Vomiting – not necessarily indicative of anything specific

B. Mid-epigastric pain that radiates to the back – typical symptom of pancreatitis

C. Gradual onset of pain – pain is usually sudden and severe

D. Alcohol consumption – this is a risk factor, not a symptom

2. A 54-year-old patient admitted with diabetes mellitus, malnutrition, osteomyelitis, and alcohol abuse has a serum amylase level of 280 U/L and a serum lipase level of 310 U/L. To what diagnosis does the nurse attribute these findings?

A. Malnutrition

B. Osteomyelitis

C. Alcohol abuse

D. Diabetes mellitus

Rationale: *The patient with alcohol abuse could develop pancreatitis as a complication, which would increase the serum amylase (normal 30-122 U/L) and serum lipase (normal 31-186 U/L) levels as shown.*

3. Which of the following can be used in the treatment of acute pancreatitis? ***Select all that apply.***

A. Parenteral fluids – used to restore blood volume and prevent hypotension and shock in acute pancreatitis

B. Corticosteroids – used to treat autoimmune chronic pancreatitis, not acute

C. Nasogastric suctioning – helps with pain from acute pancreatitis and prevents paralytic ileus in patients who are nauseous and/or vomiting

D. H2 receptor antagonists – decreases gastric acid production and therefore decreases stimulation of pancreas in acute pancreatitis

E. Narcotics or Demerol – narcotics help with pain associated with acute pancreatitis and Demerol is used when the sphincter of Oddi is involved

F. Surgical resection of the pancreas – used as a treatment option for chronic pancreatitis, not acute

4.Which lab test result would indicate that Mr. Walker has acute pancreatitis?

A. Elevated bilirubin levels

B. Elevated serum lipase and amylase levels

C. Low white blood cell count

D. Elevated blood alcohol level

Rationale: The pancreas will continue to release the enzymes lipase and amylase even when the outflow of these enzymes becomes blocked. This will result in an elevated serum lipase and amylase level in the blood.

5. Why is the body more susceptible to secondary infections during cases of pancreatitis?

A. The production of anti-inflammatory cytokines and specific cytokine inhibitors

B. The formation of pseudocysts

C. The release of cortisol in response to stress

D. The activation of leukocytes in response to pro-inflammatory cytokine production

Rationale: As a result of the systemic inflammatory response during acute pancreatitis, anti-inflammatory cytokines and specific cytokine inhibitors are produced, increasing the body's risk for infection.