Gastrointestinal Alterations

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ACUTE GASTROINTESTINAL BLEEDING

GI bleeding results in high patient morbidity and medical care costs. Many causes of acute GI bleeding necessitate admission of a patient to the critical care unit.

CAUSES OF GASTROINTESTINAL BLEEDING

Causes of Upper Gastrointestinal Bleeding

- Duodenal ulcer
- Gastric ulcer
- Esophageal or gastric varices
- Mallory-Weiss tear

Causes of Lower Gastrointestinal Bleeding

- Polyps
- Inflammatory disease
- Diverticulosis, Cancer ,Vascular ectasias
- Hemorrhoids

DUODENAL VS GASTRIC

Duodenal Ulcers

- Pain relieved by meal.
- Occurs 2-3 hrs after meal.
- Most common type.
- Dark, Tarry Stools (Melena) Occur

Gastric Ulcers

- Pain increased by meal.
- Occurs 30m to 1hr after meal.
- Not as common.
- Vomiting occurs.

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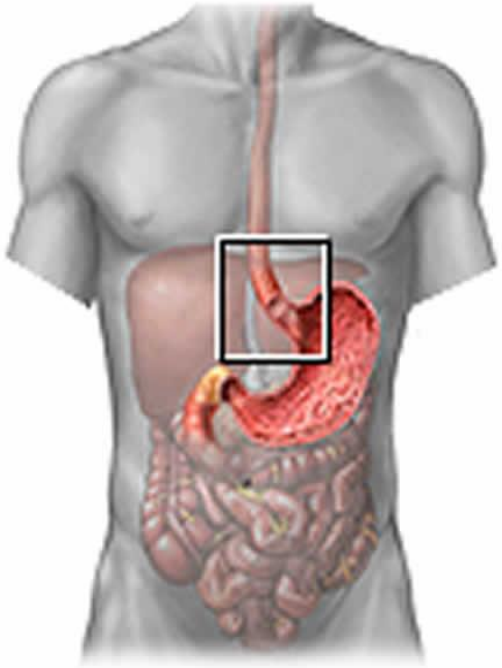
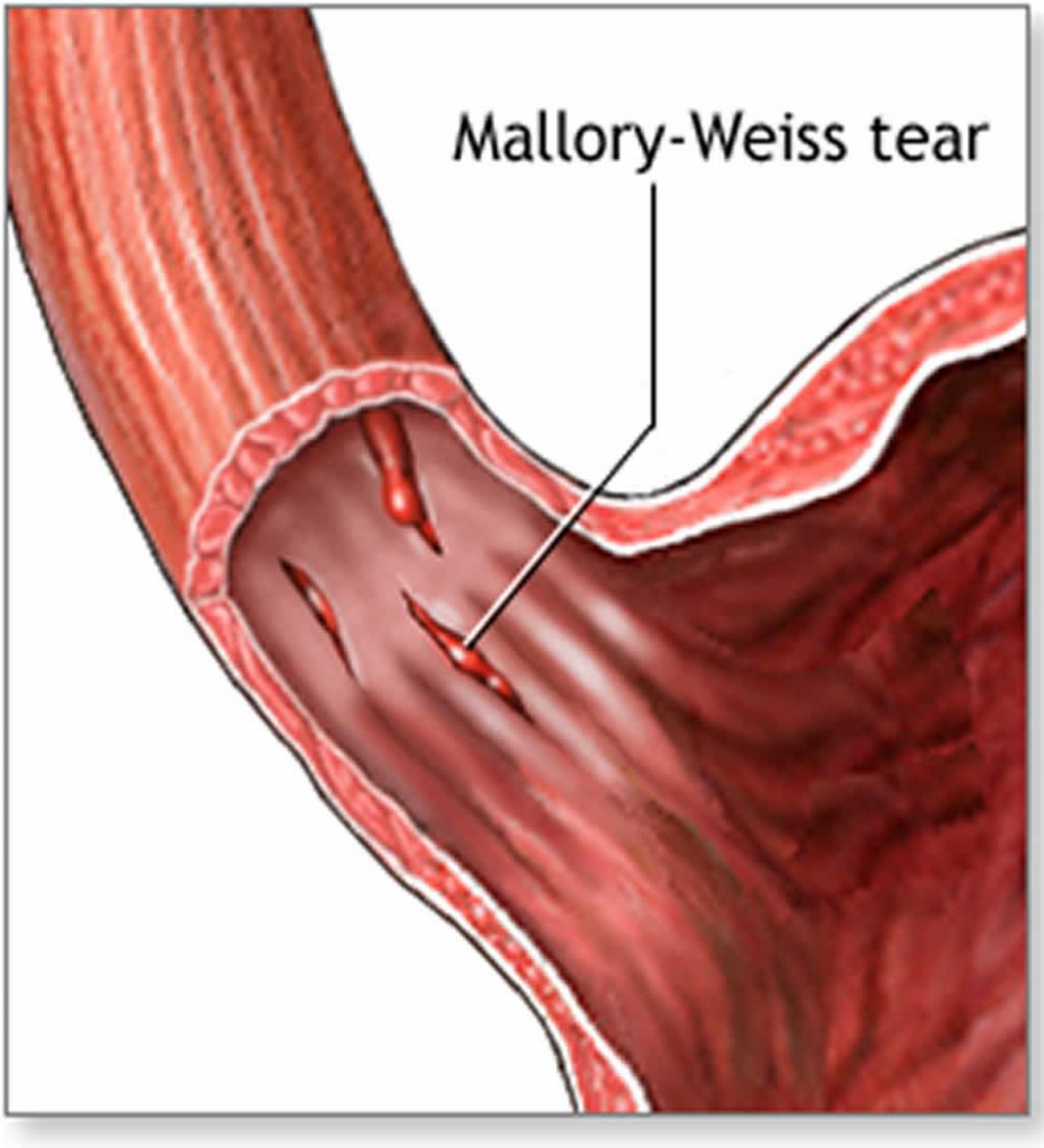


Mallory-Weiss tear is a tear in the mucosal layer at the junction of the esophagus and stomach



Peptic Ulcer Disease

-Peptic ulcer disease is characterized by a break in the mucosa that extends through the entire mucosa and into the muscle layers, damaging blood vessels and causing hemorrhage or perforation into the GI wall

-Duodenal and gastric ulcers are the most common cause of peptic ulcer disease and the most common cause of upper GI bleeding

The secretion of acid is important in the pathogenesis of ulcer disease. Acetylcholine (a neurotransmitter), gastrin (a hormone), and secretin (a hormone) stimulate the chief cells, which stimulate acid secretion.

Parietal cell mass in people with peptic ulcer disease is 1.5 to 2 times greater than in persons without disease.

RISK FACTORS FOR PEPTIC ULCER DISEASE

- Smoking: stimulates acid secretion
- Helicobacter pylori infection: elevates levels of gastrin and pepsinogen, and releases toxins and enzymes promoting inflammation and ulceration
- Habitual use of nonsteroidal anti inflammatory drugs: inhibits prostaglandins
- Alcohol consumption

CONTRIBUTING FACTORS TO ULCER FORMATION

- Increased number of parietal cells in the gastric mucosa
- Gastrin levels remain higher longer after eating
- Gastrin levels continue to stimulate secretion of acid and pepsin
- Feedback mechanism fails
- Rapid gastric emptying overwhelms buffering capacity
- Association of Helicobacter pylori with mucosal epithelial cell necrosis
- Decreased mucosal bicarbonate secretion

Stress Ulcers

Ischemia is the prior etiology associated with stress ulcer formation. Ischemic ulcers develop within hours of an event such as hemorrhage, multisystem trauma, severe burns, heart failure, or sepsis. The shock, anoxia, and sympathetic responses decrease mucosal blood flow leading to ischemia

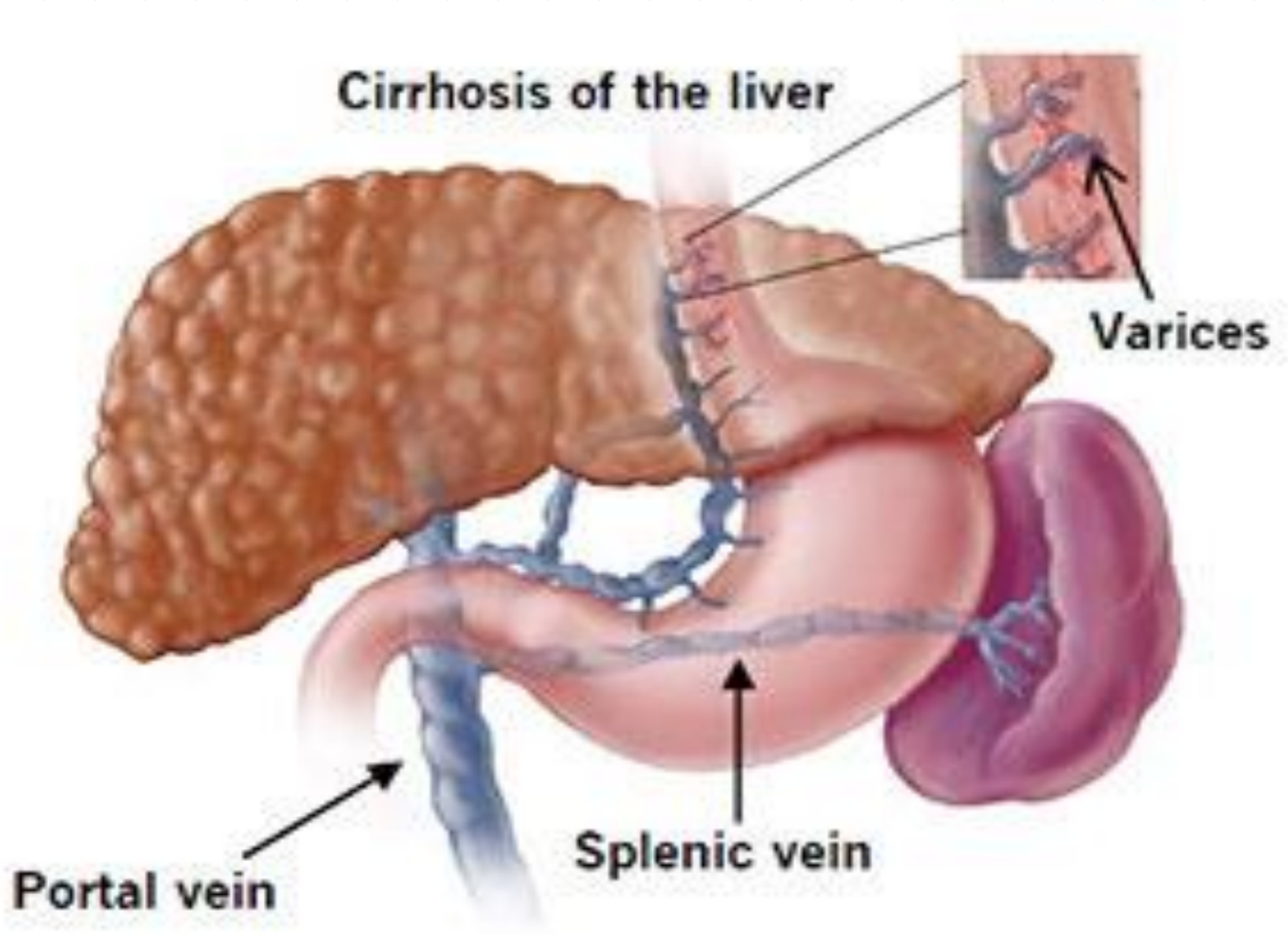
Mallory-Weiss Tear

Arterial hemorrhage from an acute longitudinal tear in the gastroesophageal mucosa and accounts for 10% to 15% of upper GI bleeding episodes. It is associated with long-term nonsteroidal anti-inflammatory drug or aspirin ingestion and with excessive alcohol intake.

The upper GI bleeding usually occurs after episodes of forceful retching. Bleeding usually resolves spontaneously; however, lacerations of the esophagogastric junction may cause massive GI bleeding, requiring surgical repair

Esophageal Varices

In chronic liver failure, liver cell structure and function are impaired, resulting in increased portal venous pressure, called portal hypertension


Cirrhosis of the liver
Varices
Portal vein
Splenic vein

Pathophysiology of UGI bleeding

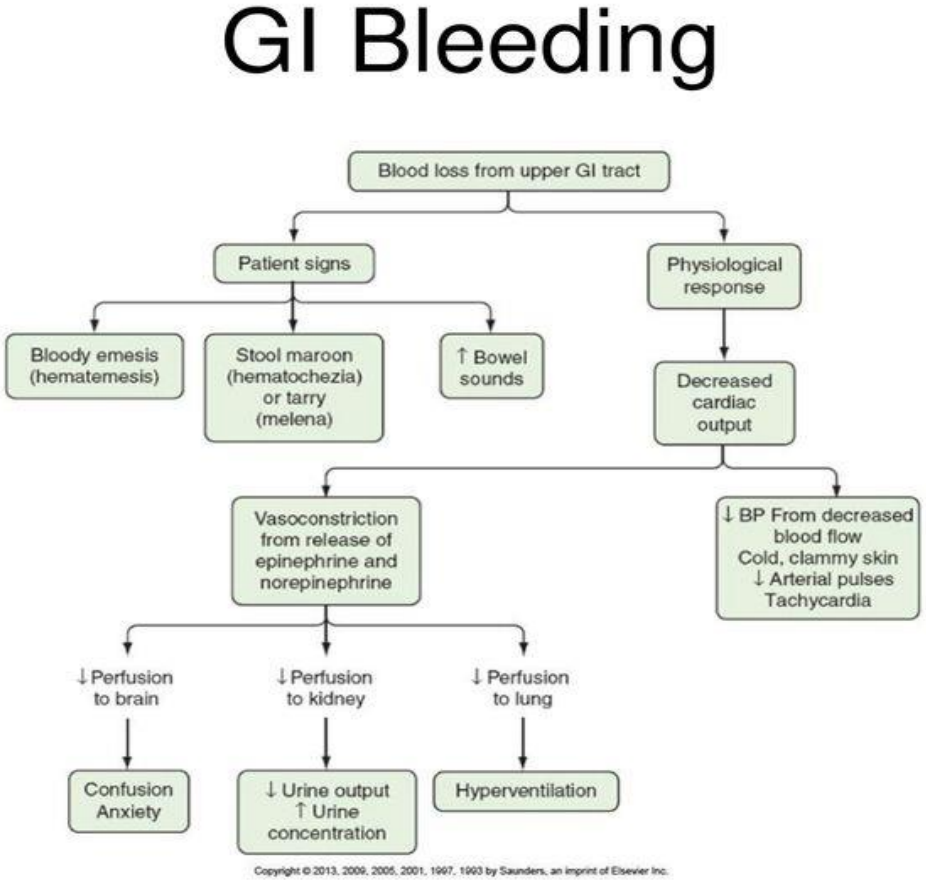


Figure 17-8. Pathophysiology flow diagram of acute upper gastrointestinal (GI) bleeding. *BP,* Blood pressure.

Signs and Symptoms of Upper Gastrointestinal Bleeding

Hematemesis

Melena

Hematochezia

Abdominal discomfort

Signs and symptoms of hypovolemic shock

- Hypotension
- Tachycardia
- Cool, clammy skin
- Change in level of consciousness
- Decreased urine output
- Decreased gastric motility

LABORATORY

- Hemoglobin: Normal, then decrease
- Hematocrit: Normal, then decrease
- White blood cell count increase
- Platelet count decrease
- Potassium: decrease then increase
- Sodium: D.
- Calcium: Normal or D.
- Blood urea nitrogen, creatinine:I.
- Hyperglycemia
- Prothrombin time, partial thromboplastin time: I.
- Respiratory alkalosis/metabolic acidosis

MANAGEMENT OF UGI BLEEDING

- Hemodynamic Stabilization

Colloids ,Crystalloids, Blood or blood products

Definitive and Supportive Therapies

- Gastric lavage

- Pharmacological therapies

Antacids

Antibiotics-Triple-agent therapy with a proton pump inhibitor and two antibiotics for 14 days

Definitive and Supportive Therapies—cont'd

- H2-histamine blockers
- Proton pump inhibitors
- Mucosal barrier enhancers

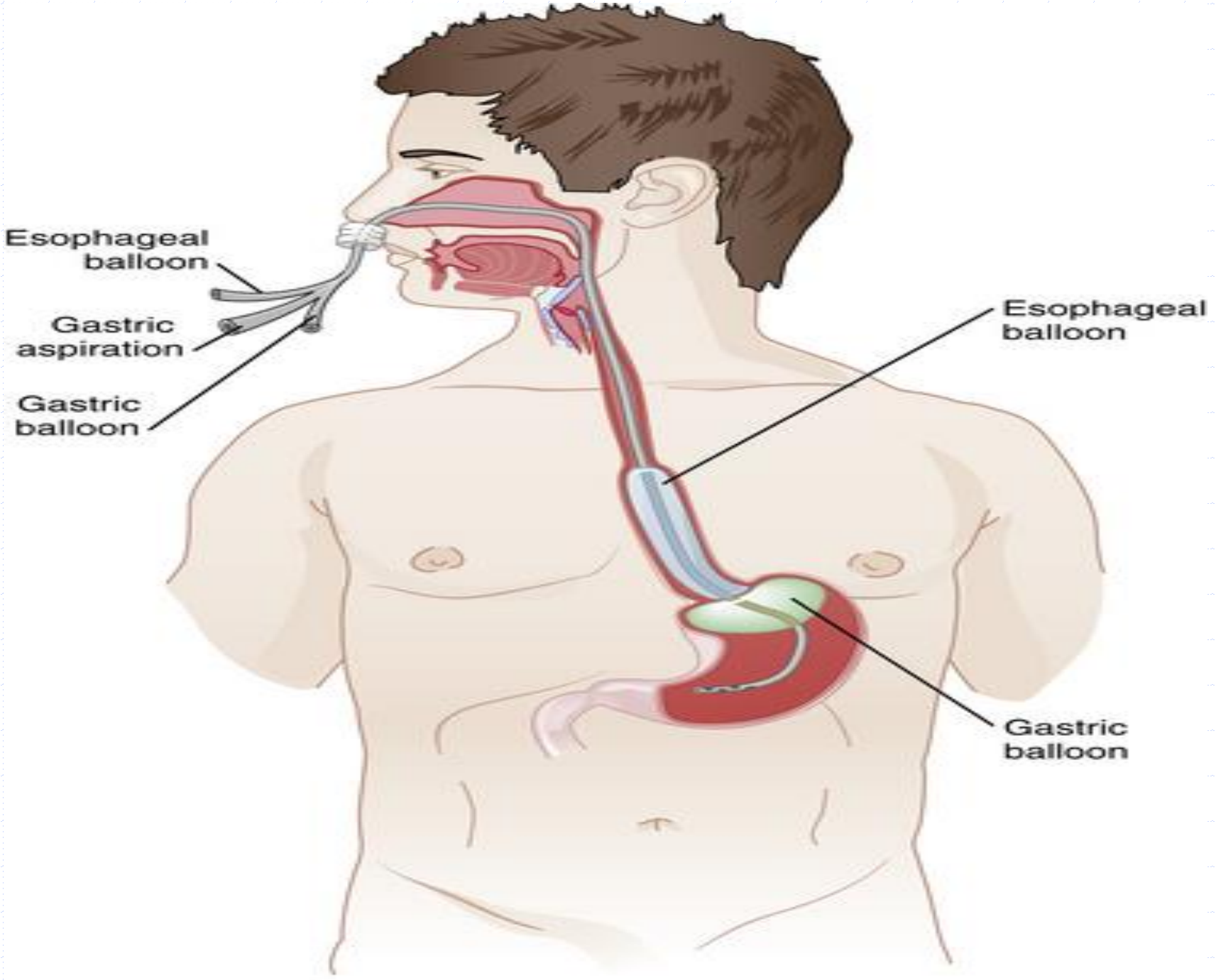
Endoscopic therapies

- Sclerotherapy
- Heater probe
- Laser

Surgical therapies

Treatment of Variceal Bleeding

- Bleeding esophageal or gastric varices are usually a medical emergency because they cause massive upper GI bleeding.
- Somatostatin or octreotide is commonly ordered to slow or stop bleeding by decrease splanchnic blood flow and reduce portal pressure
- Vasopressin lowers portal pressure
- Esophagogastric Tamponade


Esophageal balloon
Gastric aspiration
Gastric balloon
Esophageal balloon
Gastric balloon

ACUTE PANCREATITIS

An acute inflammatory disease of the pancreas. The intensity of the disease ranges from mild, in which the patient has abdominal pain and elevated blood amylase and lipase levels, to extremely severe, which results in multiple organ failure.

In 85% to 90% of patients, the disease is self-limiting (mild acute pancreatitis), and patients recover rapidly

CAUSES OF ACUTE PANCREATITIS

-Biliary disease

- Gallstones
- Common bile duct obstruction
- Post ERCP procedure

-Alcohol

-Traumatic injury of the pancreas

-Tumors of pancreatic ductal system

-Medications

-Heredity

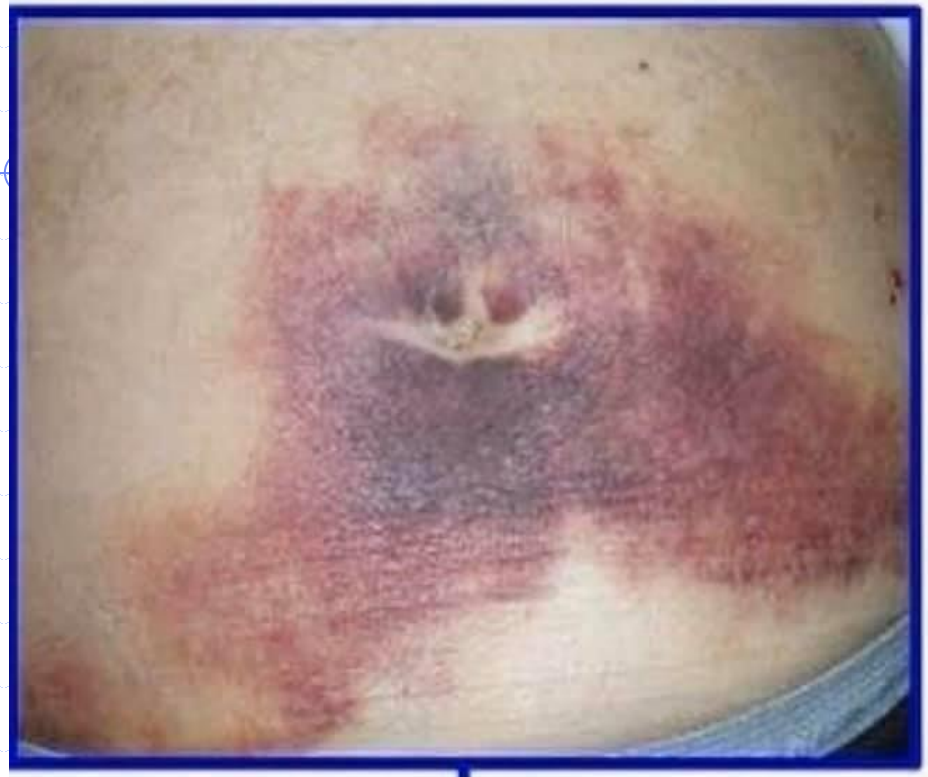
- Hypercalcemia

-Hypertriglyceridemia

- Infections and Idiopathic

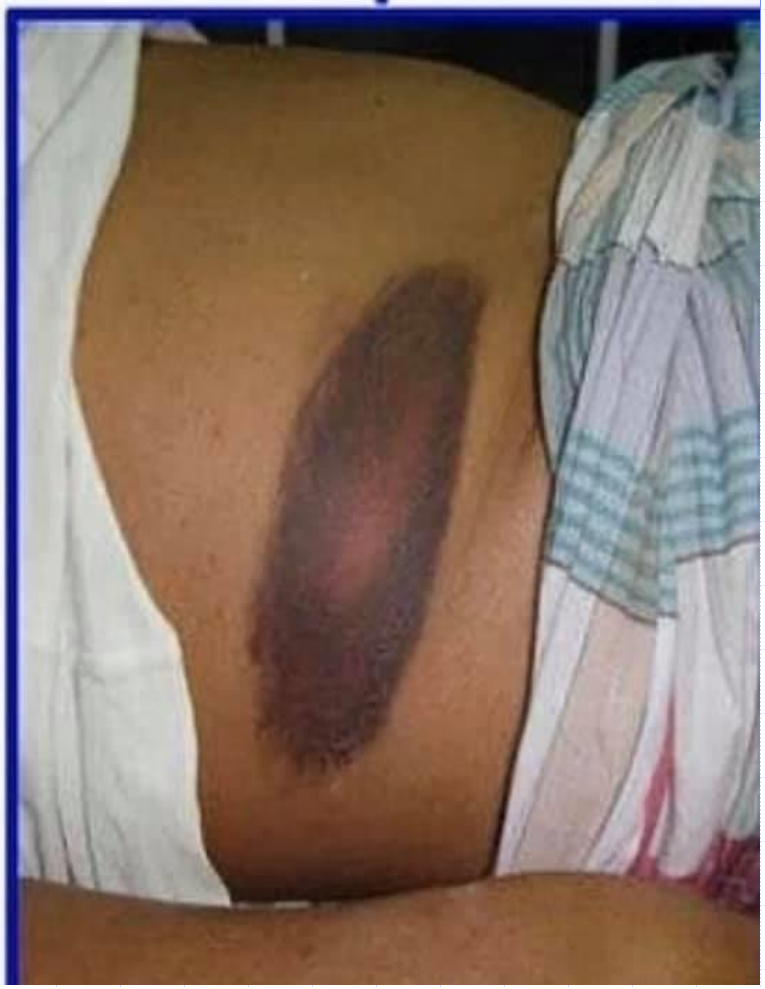
Signs and Symptoms of Acute Pancreatitis

- Pain
- Nausea and vomiting
- Fever
- Dehydration
- Abdominal guarding, distention
- Grey Turner's sign
- Cullen's sign



Cullen's Sign

Turner's Sign



LABORATORY

- Serum and urine amylase: I
- Serum lipase: I
- White blood cell count: I
- Calcium: D
- Potassium: D
- Albumin: D
- Glucose: I with islet cell damage
- Bilirubin, AST, LDH: I
- Alkaline phosphatase: I with biliary disease

SYSTEMIC COMPLICATIONS OF ACUTE PANCREATITIS

- Pulmonary :Hypoxemia ,Atelectasis, pneumonia, pleural effusion,ARDS
- Cardiovascular : Hypovolemic shock ,Myocardial depression, Cardiac dysrhythmias
- Hematological: Coagulation abnormalities

Disseminated intravascular coagulation

- Gastrointestinal :Gastrointestinal bleeding

Pancreatic pseudocyst ,Pancreatic abscess

- Renal :Azotemia ,Oliguria ,Acute renal failure
- Metabolic :Hypocalcemia ,Hyperlipidemia

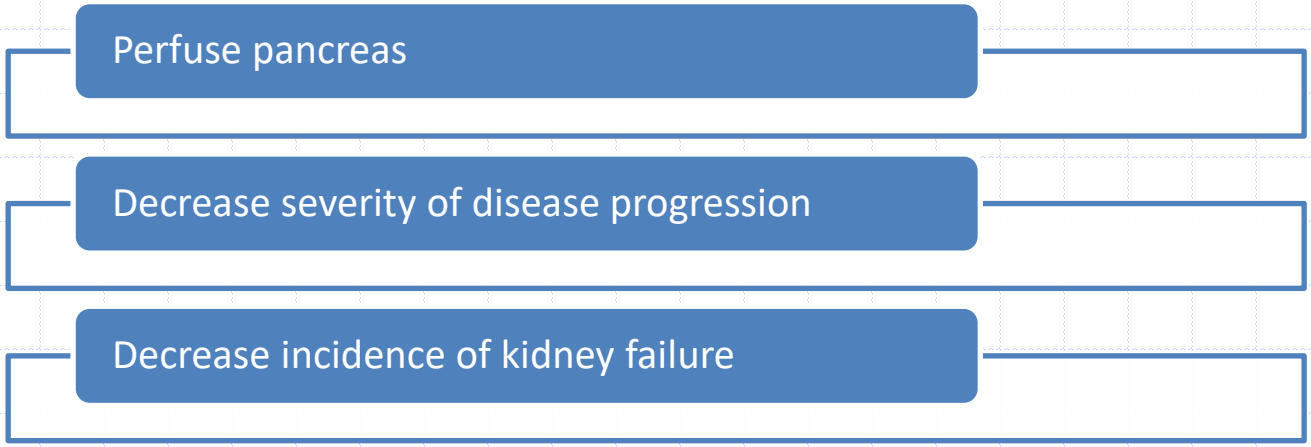
Hyperglycemia , Metabolic acidosis

Diagnosis

- Diagnosis is based on labs, radiologic tests, clinical history, presenting signs and symptoms and physical findings
- Serum amylase & lipase most specific indicator (24-72 hours)
- Serum amylase and urine amylase must be measured
- Decreased albumin and serum protein due to extracellular shift
- Abdominal x-ray, CT & MRI

Management

- Aim of therapy is fluid and electrolyte replacement, analgesic and pain control
- Fluids usually colloids or lactated Ringers solution
- FFP and albumin may be used



- Correction of electrolyte imbalance

-Hypocalcemia leading to ECG changes and tetany

-Serum albumin level important for correct calcium levels

-Hypomagnesemia

-Hypokalemia

-Hyperglycemia

- Nasogastric suction is needed to suppress pancreatic secretions and decrease nausea, vomiting and abdominal pain
- Keep NPO until pain decreases and serum pancreatic enzymes return to normal
- In mild cases restart oral fluids intake within 3-7 days
- TPN with/without lipids
- Bed rest

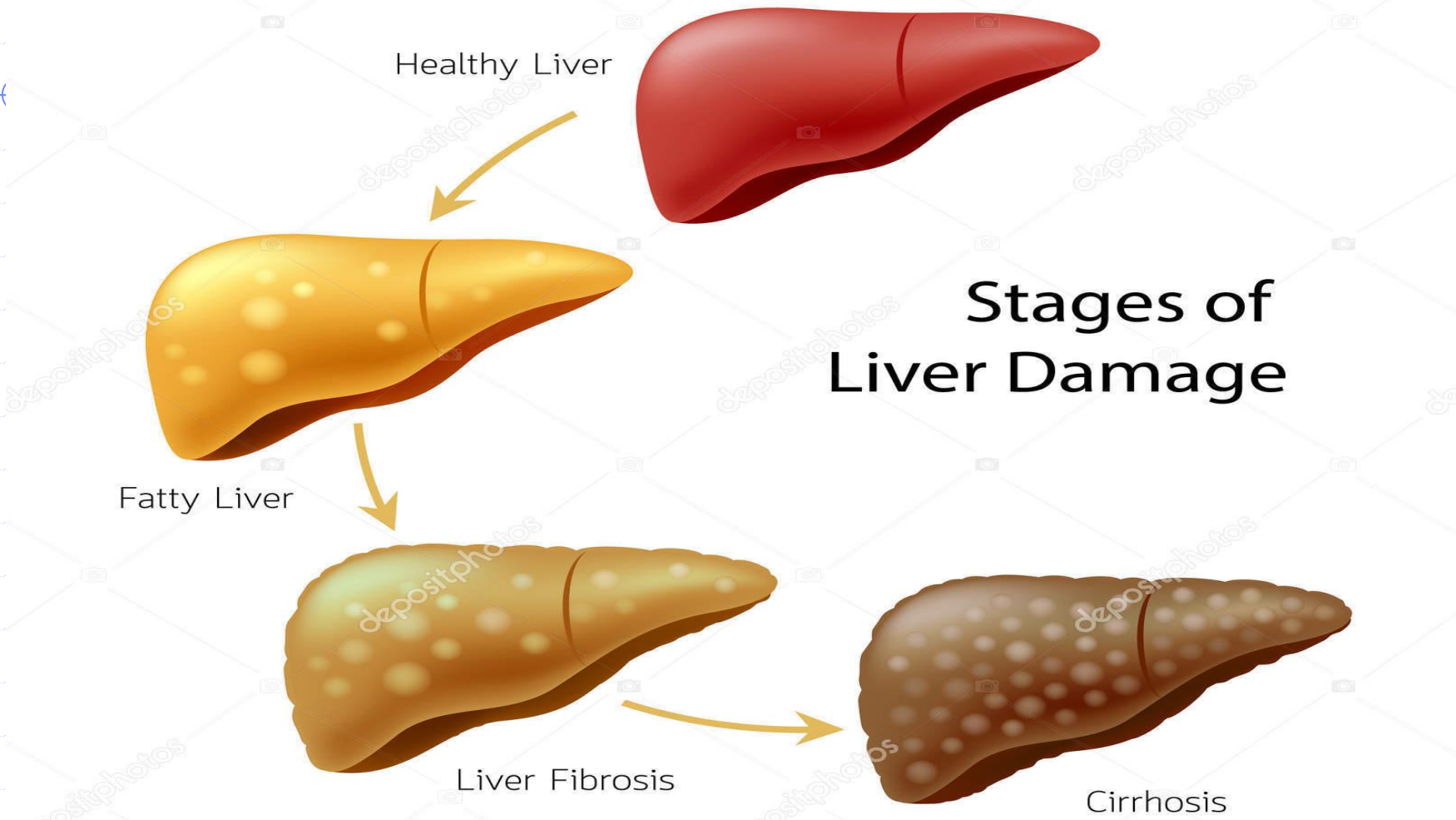
- Pain control
- Opiate analgesic
- Surgical resection of necrotic part
- Treatment of Systemic Complications

Pharmacological Intervention

- Drugs given to rest the pancreas specifically anticholinergics, glucagon, somatostatin, cimetidine, and calcitonin
- Prevention of stress ulcers is achieved through the use of histamine blockers and antacids
- prophylactic systemic antibiotics

HEPATIC FAILURE

- Liver unable to perform function
- Necrosis or a decrease in blood supply
- Cirrhosis: inflammation and necrosis- focal or diffuse
- Fat deposition, portal hypertension and enlarged liver cells
- Alcohol



Signs and symptoms

- Weakness, fatigue, weight loss, loss of appetite, Nausea and vomiting, changes in bowel habits
- Structural changes will lead to portal hypertension and development of varices
- Signs and symptoms of heart failure
- Altered metabolic processes: carbohydrates, fat, & protein

- Decreased clotting factors synthesis: micro-emboli
- Decreased detoxification level: ammonia level
- Elevated blood sugar
- Change in serum osmotic pressure: low albumin
- Kupffer's cells: low immunity- severe gram-negative sepsis
- Jaundice & spider angioma

- Ascites
- Encephalopathy
- Hepatorenal syndrome: Aldosterone system

LABORATORY

- Albumin D
- Ammonia I
- Total bilirubin I
- Direct or conjugated bilirubin I
- Cholesterol I
- Coagulation Tests ,Prolonged
- APT ,AST ,ALT : I
- Urine Bilirubin ,Urobilinogen :I

Management

- Maintain hemodynamic stability
- Administer drugs & fluids
- Hypoglycemia at later stage
- Increased risk of bleeding
- Drug metabolism must be restricted
- Bed rest, fluid restriction and diuretics for ascites
- Use of lactulose to decrease ammonia level

Management of Fulminant Hepatic Failure

- **Drugs:**
  - A. Vitamin K
  - B. H2 antagonist
  - C. Antacids
  - D. Lactulose
  - E. N-acetylcysteine for acetaminophen toxicity
  - F. Broad-spectrum antibiotics
  - G. Antifungals

Systemic Manifestations of Acute Liver Failure