CERTIFICATION REVIEW: GASTROINTESTINAL SYSTEM

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Content Description

There are many acute and critical illnesses which develop within the gastrointestinal tract. This session will provide the critical care and progressive care nurse with the necessary background knowledge needed regarding these aberrant conditions within the organ systems of the GI tract. This session will provide a review of the most common GI disorders and GI surgeries seen in critically ill patients. This information will serve to prepare the participant for the CCRN or PCCN certification examination. This session will explore the following:

| GI hemorrhage                          | Bowel infarction/Obstruction/perforation |
| Pancreatitis                           | Malnutrition and malabsorption           |
| Hepatic failure/coma                   | GI surgeries (Whipple, esophagogastrectomy, gastric bypass) |
| **PCCN only**                          | ****                                    |
| GI infections                          | Acute abdominal trauma                  |
| GI motility disorders                  |                                         |

Learning Objectives

At the end of this session, the participant will be able to:

1. Discuss the incidence and etiology of gastrointestinal bleeding, GI obstruction and perforation, acute pancreatitis, hepatic failure and other acute illnesses and surgeries of the GI tract
2. Apply assessment and management strategies for acute and critical illnesses of the gastrointestinal system as well as for GI surgical problems
3. Review the significant symptoms of GI illness or catastrophe within the context of the related anatomy and pathophysiology

Outline

**CCRN and PCCN Blueprints**

- CCRN and PCCN
  - GI hemorrhage
  - Pancreatitis
- CCRN only
  - Bowel infarction, perforation
  - GI surgeries
– Hepatic failure/coma - Abdominal trauma
– Malnutrition
• PCCN only
  – GI infections
  – GI motility disorders

GI Test Plan
• 6% of CCRN – 9 questions
• 5% of PCCN – 6 questions

• This presentation includes discussions of pulmonary artery catheter measurements, administration of vasoactive medications, and advanced devices such as balloon tamponade and abdominal pressure measurement. These topics will not be tested in the PCCN exam.

Upper Gastrointestinal Bleeding
• Bleeding that originates from any gastrointestinal site proximal to ligament of Treitz (duodenal-jejunal juncture)

Etiology of UGI Bleeding
• Peptic ulcers
  – Duodenal 80%; gastric 20%
  – Erosive gastritis
  – Stress ulcer
  – Colonization with helicobacter pylori
  – Decreased perfusion, less mucous for barrier
• Esophageal varices
• Mallory-Weiss tear
  – Mucosal tear of esophagogastric junction
• Neoplasms
• Arterio-venous malformations
• Zollinger-Ellison Syndrome (gastrinoma)
  • Hypersecretion of gastric juices from pancreatic tumors

Patterns of GI Bleeding
• Hematemesis
  – Vomiting of fresh, unaltered blood
• Melena
  – Black, tarry stool from UGI bleed

Diagnosis of GI Bleed
• Gastric aspiration
  – Presence of gross blood
• Endoscopy
  – Preferred method
  • Angiographic exam
• Tagged red blood cell study
– Diagnostic accuracy
– Therapeutic interventions
• Barium study to identify:
  – Site of bleeding
  – Peptic ulcers
  – Tumors
  – Inflammatory processes

**Laboratory Diagnosis of GI Bleed**

• Hemoglobin and hematocrit
  – Not reliable indicator of degree of bleeding
  – Helpful for trending
• WBCs may be elevated
• Nasogastric drainage and stool guaiac positive for blood
• BUN and creatinine elevated – RBC breakdown
• Calcium decreased d/t blood transfusion
• Cultures for H. pylori

**Collaborative Management**

• Nasogastric drainage and lavage as performed in diagnostic phase
  – Use room temperature fluids
• Assess hemodynamic consequences of blood loss
• Instability managed by aggressive resuscitation with blood, blood products and crystalloid

**Assess for Perforation**

• Sudden, severe, generalized abdominal pain
• Rebound tenderness
• Abdominal rigidity
• Fever, leukocytosis, tachycardia

**Other Causes of Perforation**

• Invading tumor
• Ruptured diverticuli
• Eroding ulcer
• Bowel obstruction

**Surgical Emergency**

• Placement of patch
• Oversew of perforation
• Colostomy/diverting ostomy
Resuscitative Efforts for GIB

- Large-bore intravenous access
- Rapid fluid administration of balanced salt solution
- Blood loss of over 1500 ml requires PRBC’s
- Replace other blood products as needed
- Vasopactive drugs

Interventional and Surgical Therapies

- Endoscopic or Angiographic
  - Bipolar coagulation of ulcers, AVM’s
  - Thermal coagulation
  - Laser coagulation
  - Phototherapeutic coagulation
  - Vessel embolization with coils, fat, etc
- Surgical
  - Oversew of bleeding site
  - Vagotomy to interrupt acid production with pyloroplasty to promote gastric emptying

Pharmacological Therapy

- Histamine blockers
  - Block gastric acid secretion
    - Ranitidine (Zantac)
    - Famotidine (Pepcid )
- Proton pump inhibitors
  - Block gastric acid secretion
    - Omeprazole (Prilosec)
    - Pantoprazole (Protonix)
- Antacids
  - Neutralize acids
    - Mylanta
    - Amphojel
- Antibiotics for H. Pylori
  - Amoxicillin
  - Ampicillin
  - Metronidazole
- Mucosal barrier enhancers
  - Sulcralfate
- Vasopressin infusion
  - Constricts arterioles

Lower GI Bleeding

- Etiology
  - Diverticulosis
  - Angiodysplasia (AVM)
  - Neoplasm
  - Polyps/benign growths
  - Ischemic colitis/bowel infarction
  - UGI sources
- Presentation
  - Hematochezia
• Maroon stool
  • May be due to UGI or LGI
    – BRBPR
    – Melena
    – Hemodynamic instability as with UGI

**Diagnosis**

- Nasogastric aspiration
- X-Ray
- Colonoscopy-gold standard
- Angiography
  - Labs
    - As with UGI
  - Interventions
    - As with UGI but using colonoscopy
  - Surgery
    - May require colostomy

**Surgical Interventions for GIB**

- Simple excision
- Oversew of bleeding site
- Vagotomy with pyloroplasty
  - decrease acid production
  - Promote gastric emptying
- Billroth I – vagotomy, antrectomy and anastomosis of stomach to duodenum
- Billroth II – Vagotomy, resection of antrum, anastomosis of stomach to jejunum

**Acute Pancreatitis**

- An autodigestive process whereby the pancreas is attacked by its own enzymes resulting in acute inflammation and edema.

**Pathophysiology of Acute Pancreatitis**

- Premature activation of exocrine pancreatic enzymes (trypsin, lipase, amylase) prior to transport into the duodenum
- Events begin within the acinar cells and ducts
- Protective mechanisms are overwhelmed by massive autodigestion
- Reflux of duodenal juices, leakage of enzymes
- A systemic inflammatory response ensues.
- Enzymes, oxygen-free radicals, inflammatory mediators (cytokine) released

**Etiology of Acute Pancreatitis**

- Cholelithiasis
- Lipid abnormalities
— Most common cause
● Medications
• Alcoholism
  - Thiazides, oral contraceptives, NSAIDS
• Infection
  ● Idiopathic
• Abdominal surgery
• Hypercalcemic states

Presentation of Acute Pancreatitis
• Severe epigastric, abdominal & back pain
• Diffuse abdominal tenderness and rebound
• Low grade fever
• Nausea and vomiting
• Glucose elevation
• Jaundice
• Ileus
• Circulatory instability
• Cullen’s sign
  - Periumbilical ecchymosis
• Grey Turner’s Sign
  - Ecchymosis of groin, flank, thigh
• Bibasilar crackles
• Weight loss

Prognosis - Ranson’s criteria

• At admission
  – Age >55 years
  – Hypotension
  – Abnl pulmonary findings
  – Hemorrhagic or discolored peritoneal fluid
  – LDH > 350 U/L
  – AST > 250 U/L
  – Leukocytosis >16,000
  – Glucose >200 mg/dl w/o hx of DM
# Factors present associated with mortality risk

Diagnostic Evaluation for Acute Pancreatitis

• Laboratory
  – Elevated serum amylase, lipase
  – Elevated urine amylase
  – Liver function tests may be elevated
  – Elevated WBC
  – Hgb & Hct may be elevated or decreased
• Radiologic
  – CXR and abdominal films
  – Abdominal CT scan
  – MRI
  – Ultrasound
  – ERCP (endoscopic retrograde cholangio-pancreatography)

Collaborative Management of Acute Pancreatitis
• Volume resuscitation
  – Blood products if bleeding
  – May require hemodynamic monitoring
• Pain management
• Parenteral nutrition or jejunostomy feedings
• Electrolytes replacement
  – Hypocalcemia, hypokalemia, hypomagnesemia
  – Monitor ECG, Chvostek’s and Trousseau’s signs
• NG tube if persistent vomiting or abdominal distention
• Somatostatin (Octreotide)
  – Inhibit pancreatic secretion
• Histamine blockers or PPI
• Pancreatic necrosectomy, peritoneal lavage, planned re-exploration
• Detect, minimize and treat complications
  – Hypovolemic shock
  – Renal failure
  – Hemorrhage
  – Respiratory failure
• Pulmonary support
  – Atelectasis, ARDS
• Insulin if hyperglycemia
  – Usually due to critical illness
  – Due to pancreatitis only if severe
• Monitor for sepsis

Systemic Complications
• Fluid, electrolyte imbalance
• Vascular losses
• Losses from massive fluid sequestration
• Electrolytes lost via blood vessel leakage especially calcium
• Chvostek’s Sign-spasm of facial muscles by tap
• Trousseau’s Sign-hand spasms with pressure
• Glucose monitoring and insulin infusion

**Local Complications**

• Pancreatic pseudocyst
• Local damage to pancreatic tissue
• Leakage of pancreatic fluid and enzymes
• May obstruct bowel or rupture  
  – Monitor via CT scan and drain

**Surgical Interventions**

• Cholecystectomy
• Pancreatic resection  
  – Acute necrotizing pancreatitis
• Drainage of pseudocyst  
  – Walled off collection of pancreatic enzymes, fluid, debris, and blood
• Drainage of abscess  
  – Infected pseudocyst  
  – Requires immediate treatment

**Exam Questions**

Patients with gastrointestinal bleeding are at greatest risk for developing:
A. adult respiratory distress syndrome  
B. disseminated intravascular coagulation  
C. hypovolemic shock  
D. septic shock.

**Exam Question**

The physician orders gastric lavage to aid in cleansing your patient’s stomach before an esophagastroduodenoscopy (EGD). The best fluid choice for your patient who is experiencing a GI bleed is:
A. Iced sterile water  
B. Iced sterile saline  
C. Room-temperature normal saline  
D. Body temperature tap water

**Exam Question**

A 40 year old patient who presents with nausea, vomiting, jaundice, and severe abdominal pain is diagnosed with acute pancreatitis. In this disorder, autodigestion of the pancreas causes release of cytokines and kinins, which alter capillary wall permeability and the vascular vasoactive properties. As the nurse anticipates potential complications that may result from these changes, which of the following interventions is most important to institute at this point?
A. Monitoring of intra-abdominal pressure  
B. Monitoring for Cullen’s sign and measuring abdominal girth  
C. Fluid resuscitation and pain control
D. Auscultation of bowel sounds and peripheral pulses

**Intestinal Obstruction/Infarction**

- **Obstruction**
  - Interference with normal passage of intestinal contents due to partial or complete occlusion of the lumen or inadequate propulsive motility
- **Etiology of Intestinal Obstruction**
  - Adhesions
  - Hernias
  - Malignancies
  - Volvulus-twisting of the bowel upon itself
  - Abscess
  - Inflammatory diseases

- **Infarction:**
  - Intestinal ischemia due to decrease in blood flow
- **Etiology of Intestinal Infarction**
  - Alpha-stimulating sympathomimetic amines (vasopressor medications)
  - Hypovolemia
  - Decreased cardiac output

**Diagnosis**

- **Clinical signs and symptoms**
  - Abdominal pain sometimes out of proportion to physical findings
  - Distension
  - Fever
  - Decreased or loss of bowel sounds
- **Obstruction series**
- **Elevated WBC**
- **Elevated BUN and signs of hemoconcentration**

**Management**

- Correct fluid and electrolyte losses
- Detect strangulation (loss of blood supply to a hernia) for emergency surgery
- NG suction
- Rectal tube for decompression of cecum
- Analgesia
- Miller-Abbott/Cantor tube-stimulate peristalsis
- Surgical lysis of adhesions, resection
• Gastric Bypass Surgery
  – Surgical treatment of morbid obesity (more than 100 pounds overweight) with associated complications (CAD, HTN, DM)
  – Modification of GI tract to reduce net intake of food

**Types of Gastric Bypass Surgery**

• Vertical banded gastroplasty (lap band surgery)
• Common complications
  • Infection
  • Obstruction
  • Band slippage
  • Esophageal dilation

• Roux-en-Y-proximal gastric pouch whose outlet is a limb of small bowel
• Complications
  – Atelectasis, pulmonary infection and pulmonary embolus are the most common
  – Wound disruption
  – Rhabdomyolysis
  – Anastomotic leak
  – Deep vein thrombosis
  – Dumping syndrome

**Whipple Procedure**

• Surgical treatment for benign and malignant tumors of the pancreas, bile ducts
• Involves removal of distal portion of stomach, duodenum, head of pancreas, distal common bile duct with anastomosis of all structures to jejunum
• Potentially curative vs double bypass procedure which is palliative

**Postoperative Complications**

• Bleeding
• Anastomotic breakdown
• Incisional and other pain
• Psychosocial issues

**Esophagogastrectomy**

• Surgical procedure for cancer of the esophagus
• Procedure involves removal of involved digestive tract and 10 cm on either side of tumor and insertion of a conduit to replace the esophagus, usually the stomach
• Pathophysiology
  – Commonly adenocarcinoma
  – Risk factors of smoking, alcohol, GERD
Classic symptom of progressive dysphagia
• Performed in conjunction with chemotherapy and radiation therapy

Postoperative Management
• Pain control with epidural/PCA
• Preventing respiratory complications (most common)-early mobilization
• Monitoring for dysrhythmias (atrial)
• Monitoring for anastomotic leak

Exam Question
Mr. G., a 74 year old man who lives alone comes to the emergency department complaining of abdominal pain lasting several days now accompanied by vomiting. He reports not having had a bowel movement in 7 days. His abdomen is firm and distended without bowel sounds. You suspect:

A. An obstructing colon tumor
B. Peptic ulcer disease
C. Variceal hemorrhage
D. Acute pancreatitis

Exam Question
A 96 year old man with a history of long-standing esophageal reflux is admitted to the surgical ICU post esophagogastrectomy. His vital signs are:
T 99.00F
BP 146/76
P 122 bpm
Incisional pain 8/10
Which of the following interventions would be key to reducing this patient’s risk of mortality and morbidity?
A. Pulmonary care
B. Pain management
C. Administration of large amounts of IV fluids
D. Immediate institution of tube feedings

Exam Question
A patient is in the ICU after undergoing a gastric resection for stomach cancer and has been started on a regular diet prior to transfer out of the unit. Within minutes of eating, the patient develops diaphoresis, weakness, cramping, and palpitations. Which of the following conditions best explains these clinical findings?
A. Myocardial infarction
B. Pulmonary embolism
C. Severe rebound hyperglycemia
D. Dumping syndrome

Gastrointestinal Motility Disorders: Ileus
• Causes
  – Postoperative
• Delay in GI motility beyond 3 days
• Common after GI surgery
  – Narcotic-induced due to altered propulsion, increased absorption of water, decreased secretions
  – Intra-abdominal processes (abscesses)

**Clinical Manifestations**

• Nausea, vomiting
• Bloating
• Sense of abdominal fullness
• Abdominal distension
• Tympany
• Scattered, occasional bowel sounds
• Absence of flatus, bowel movement

**Prevention and Management**

• Non-narcotic interventions for pain
• Early postoperative feeding with liquids
• Progressive ambulation- needs more study
• Fluid and electrolyte replacement
• Nasogastric tube to suction
• Treat underlying causes

**Gastrointestinal Motility Disorders: Gastroparesis**

• Causes
  – Delayed gastric emptying
  – Consequence of systemic conditions such as diabetes mellitus, scleroderma
  – Also occurs with GERD
  – Unknown prevalence

**Clinical Manifestations**

• Nausea, vomiting
• Early satiety
• Abdominal discomfort
• Distention
• Bloating
• Anorexia

**Management**

• Suspect and exclude mechanical obstruction
• Assess gastric and small bowel motility
• Hydration
• Electrolyte repletion
• Nutritional supplementation
• Metaclopramide- prokinetic
• Erythromycin- accelerates gastric emptying
• Octreotide, antiemetics, antibiotics

**GI Infections: Clostridium Difficile**

• Organism responsible for antibiotic associated colitis
  — C. diff colonizes and over-grows GI tract after the normal flora has been altered by the antibiotic
  — Two protein exotoxins released by the C. diff bacteria (toxin A and toxin B)
    • Mucosal injury and inflammation

**Clinical Manifestations**

• Mild to moderate disease
  — Lower abdominal cramping pain
  — May have absence of systemic symptoms/ physical findings

• Moderate to severe colitis
  — Profuse diarrhea- 10 + liquid foul smelling stools/day
  — Abdominal distension with pain
  — Occult colonic bleeding
  — Fecal leukocytes

**Pathophysiology**

• C. diff toxins cause shedding of cells of basement membrane into lumen of colon
  — Shallow ulcer left on the mucosal surface-yellow raised plaques which slough
  — Serum proteins, mucus and inflammatory cells flow out from the ulcer

**Diagnostic Testing**

• Stool sample to detect C. diff in the stool
• Stool culture
  — Most specific, sensitive test
• Most labs use commercially available enzyme immunoassay (EIA kits) to detect toxin A and B
• Abdominal radiograph and CT
• Colonoscopy

**Management**

• Supportive care
Correction of fluid and electrolyte losses
Avoidance of antiperistaltic agents
Strict adherence to infection control practices

• Withdrawal of antibiotic therapy
• Oral metronidazole
  — Recommended first line agent
• Alternative first line drug is oral Vanco
• Duration of therapy 10 to 14 days

Malnutrition in Hospitalized Patients

• Optimum nutrition to maintain health
  — Malnutrition present in > 50% of inpatients
• Deficiency of any nutrient
  — Abnormal intake
  — Impaired digestion
  — Malabsorption
  — Abnormal metabolism or excretion of a nutrient

Acute Malnutrition: Inadequate Intake

• Causes
  — Injury or stress
  — Trauma
  — Infection
  — Surgery
  — Medications

Result is rapid depletion of nutrient stores
• Also seen in chronic form
  — Alcohol abuse
  — Chronic diseases such as cancer, liver disease, cardiovascular or renal disease
  — Eating disorders
• Most common type is protein-calorie malnutrition (marasmus)
  — Depletion of tissue energy stores
  — Depletion of body protein
• Also may see kwashiorkor
  — Protein depletion without weight loss

Nutrition-Associated Complications
• Result of organ wasting and impaired functional capability
• Weakness
• Decreased wound healing
• Altered hepatic metabolism of drugs
• Respiratory failure
• Decreased cardiac contractility
• Infections such as pneumonia

**Nutrition Assessment**
• Nutritional and weight history
• Physical exam
• Anthropometrics
• Height, weight, BMI
• Biochemical Assessment
  – Albumin
  – CRP
  – Prealbumin
  – Transferrin
  – Cholesterol
• Estimation of protein and calorie requirements

**Management of Malnutrition**
• Oral or enteral- best route is oral
• Peripheral parenteral nutrition
• Total parenteral nutrition
  – Central venous access needed
• Multivitamins

**Exam Question**
• Your 69 year old patient admitted status post a fall has a history of extremely reduced appetite and now her diet is orally restricted due to an injury to her spleen. You are concerned about her nutritional status and expect the following laboratory testing to be done to evaluate for malnutrition:
  A. Bilirubin
  B. Complete blood count
  C. Albumin
  D. Electrolytes

**Hepatic Failure**
• Clinical Indicators
  – Bilirubin concentrations > 10 mg/dL
  – Serum albumin < 2.5 mg/dL
– Prothrombin time > 5 seconds beyond control
– Incapacitating hepatic encephalopathy
– Recurrent variceal bleeding
– Intractable ascites
– Recurrent spontaneous bacterial peritonitis

**Etiologies of Hepatic Failure**

– Hepatitis A
  • Oral-fecal route
  • Mild, self-limiting

– Hepatitis B
  • Contact with blood, body fluids, needles
  • More serious; may develop cirrhosis, cancer

- Hepatitis C
  • Contact with blood, body fluids, needles
  • Usually mild
  • Progresses to chronic state
  • Leading indicator for liver transplant

**Manifestations of Hepatitis**

• Flulike symptoms
• Low grade fever
• Anorexia
• Jaundice
• Urticaria

• Dark urine
• Presence of antibodies
• May be asymptomatic

**Etiologies of Hepatic Failure**

• Cirrhosis
  – Chronic disease characterized by diffuse damage and regeneration of hepatocytes resulting in fibrosis and nodular parenchymal tissue replacement in the liver

**Classification of Cirrhosis**

– Laennec’s (alcoholic)
– Biliary
  • Scarring due to biliary obstruction/infection
– Cardiac
  • Right sided heart failure
– Postnecrotic
  • Late result of previous, acute viral hepatitis

**Clinical Presentation of Hepatic Failure**
- Jaundice
- Urticaria
- Fever
- Anorexia
- Nausea

- Easy bruising
- Abdominal discomfort
- Petechiae
- Anemia

**Diagnosis of Hepatic Failure**
*(End Stage Liver Disease)*

- Liver function tests
  - Transaminases (AST & ALT)
  - Direct and total bilirubin
  - Protein and albumin levels
  - Coagulation studies
- Ultrasound & CT scan
- Liver biopsy is gold standard

**Complications of Hepatic Failure**

- Hepatic Encephalopathy
- Portal hypertension
  - Esophageal Varices
  - Ascites
- Hepatorenal Syndrome
- Hepatopulmonary Syndrome

**Hepatic Encephalopathy**

- Pathophysiology
  - Liver unable to convert ammonia to urea
  - Ammonia crosses blood-brain barrier
  - Increased ammonia levels cause hepatic coma
- Clinical Presentation
  - Constructional apraxia
  - Worsening ability to draw, deterioration in handwriting
  - Asterixis
  - Flapping tremor of hands
  - Fetor hepaticus
  - Sweet, fetid character to the breath

- Diagnosis
  - Serial neurological exams
  - Serum ammonia levels
• Management
  – Lactulose and Neomycin
  – Moderate protein intake
  – Eliminate hepatotoxins
  – Liver transplantation

**Esophageal Varices**
• Portal hypertension causes increased pressure in esophageal veins
• Become fragile and distended
• Bleed easily and can rupture

**Diagnosis and Treatment of Esophageal Varices**
• Diagnostic esophagogastroduodenoscopy (EGD)
• Prevention of rupture
  – Beta blockers and nitrates to reduce portal pressure
  – Endoscopic variceal ligation (EVL banding)
• Treatment of Acute Rupture
  – Balloon tamponade
  – EVL banding
  – TIPS procedure (transjugular intrahepatic portosystemic shunt)
    - EVL Banding
    - Balloon Tamponade
    - Transjugular Intrahepatic Portosystemic Shunt (TIPS)

  • Decreases portal pressure, decreasing risk for acute hemorrhage
  • High rate of occlusion (50%) within 1 year
  • Encephalopathy is worsened in 20% of patients

**Ascites**
• Free fluid within the peritoneal cavity
• Complex pathophysiology: inappropriate renal sodium and water retention, increased lymph production and increased capillary membrane permeability

**Management of Acites**
• Sodium and fluid restriction
• K-sparing diuretics (Aldactone)
• Albumin
• Paracentesis
• Peritoneal-venous shunt

**Hepatorenal Syndrome**
• Renal failure unresponsive to fluid administration or diuretics
• May require hemodialysis or CRRT

**Hepatopulmonary Syndrome**
• Deterioration of the pulmonary system associated with ESLD
• Hallmark of worsening hypoxemia requiring mechanical ventilation

**Fulminant Hepatic Failure (Acute Liver Failure)**
• Sudden and severe impairment of hepatic function
• Most frequently due to an acute insult in a previously healthy person
  – Occurs over 1-3 weeks
• Require urgent liver transplant
  – Spontaneous recovery is rare, about 10-20%
  – Liver function normalizes with recovery

**Causes of Acute Liver Failure (ALF)**
• Infection
  – Hepatitis
  – Herpes
• Metabolic disorders
  – Heat stroke
• Toxins
  – Industrial substances such as hydrocarbons
  – Drugs
  – Acetaminophen, phenytoin, cocaine
• Other
  – Reye’s syndrome
  – Malignant infiltration

**Clinical Manifestations of ALF**
• Hepatic Encephalopathy
  – May develop within 2 weeks of insult
• Jaundice
• Coagulopathy
• Cerebral edema
• Hypoglycemia
• Circulatory instability
• Respiratory dysfunction/failure
• Renal dysfunction/failure
• Increased risk of infection

**Management of ALF**
• Treat encephalopathy as previously discussed
• Stress ulcer prophylaxis; FFP, vitamin K for active bleeding and invasive procedures
• Management of cerebral edema with ICP monitoring, mannitol, and pentobarbital coma with continuous EEG monitoring

**Abdominal Trauma**
• Blunt trauma
• Penetrating trauma
Blunt and Penetrating Abdominal Injury

Assessment

- Complicated by drug/alcohol, decreased LOC
- Entry/exit wounds in penetrating trauma
- Cullen’s sign: purplish discoloration of umbilicus
  - Blood in abdominal wall
- Grey-Turner’s sign: ecchymosis of flank, groin
  - Retroperitoneal bleeding or injury to pancreas
- Kehr’s sign: referred pain to left shoulder
  - Ruptured spleen
- Hematoma in flank
  - Renal injury, major vessel or pancreatic injury
- Distended Abdomen

Diagnosis

- NGT and Foley
- Diagnostic Peritoneal Lavage (DPL)
- Ultrasound: Focused Assessment with Sonography for Trauma (FAST)
  - Pericardial sac, LUQ (splenorenal), pelvis, RUQ
  - Free fluid in abdomen; hemoperitoneum

Abdominal Compartment Syndrome (ACS)

- Tissue edema or free fluid collects in abdominal cavity
- Causes increased pressure in abdomen

- Trauma causes
  - Fluid resuscitation
  - Multiple trauma with or without abdominal trauma
  - Retroperitoneal / abdominal wall bleeding

Definitions

- Intra-abdominal Pressure (IAP):
  Intrinsic pressure within the abdominal cavity

- Intra-abdominal Hypertension (IAH):
  A sustained IAP > 12 mm Hg without obvious organ failure
• Abdominal Compartment Syndrome (ACS): IAH > 20 mm Hg with at least one organ dysfunction or failure

**Evaluation of Abdominal Pressure Measurement**

• 0-5 mmHg—normal
• 5-10 mmHg—common in many ICU patients
• > 12 mmHg (Grade I)—intra-abdominal HTN
• 16-20 mmHg (Grade II)—dangerous IAH. Need to begin non-invasive interventions
• >21-25 mmHg (Grade III)—impending abdominal compartment syndrome.

**Measuring IAP**

• Pressure bag/ transducer tubing
• Cable to monitor
• Stopcock with syringe
• Connector to Foley
• Transducer at iliac crest, mid axillary line
• Clamp foley below sampling port
  – If using special device, will automatically close
• Draw up 25 ml of fluid
• Inject into bladder
• Obtain measurement
• Advise physician according to findings

**Collaborative Management**

• Identify patient at risk
• Intra-abdominal pressure measurement Q 1-2 Hrs
• Medical interventions
  – Intra-abdominal pressure 12 – 15 mmHg
    • Intra-abdominal hypertension
    • Cardiac, pulmonary, GI, renal and neurological dysfunction can occur if pressure not reduced
    • Optimize fluid management
  – IAP 15 – 20 mmHg and no evidence organ dysfunction
    • Sedation/NMB
    • Paracentesis if due to free fluid
    • Decompression of GI tract
      – NGT, colonic tube
      – Rectal enema
• Abdominal compartment syndrome IAP > 20 mmHg or > 15 mmHg with evidence of organ dysfunction
  – Surgical intervention
  – Decompressive laparotomy with open abdomen

**Exam Questions**

A patient with hepatic failure becomes increasingly confused. Which of the following
laboratory values will assess this patient for encephalopathy?
A. Hemoglobin and hematocrit
B. Liver enzymes
C. Serum albumin
D. Serum ammonia

Exam Question
A patient is admitted to the medical ICU with decompensated cirrhosis. He is encephalopathic with splenomegaly, ascites, and portal hypertension on CT scan. Portal hypertension places this patient at risk for gastrointestinal bleeding owing to the:
A. Development of right sided heart failure
B. Excessive circulating blood volume associated with cirrhosis
C. Fibrotic nature of the hepatic tissue
D. Elevated pressure in esophageal veins

Exam Questions
To determine the presence of jaundice in a patient of color, the nurse would assess:
A. Sclera
B. Oral mucosa
C. Soles of feet
D. Nail beds

Exam Question
A patient is in the ICU following an emergency hemicolecotomy following a gunshot wound to the abdomen. Clinical findings include pulse 145 bpm, temperature 102°F, abdominal distention and hypoactive bowel sounds. The BP had been 140-86 but now is 108/80. His extremities are warm to the touch with bounding pulses.
The physician orders rapid fluid administration, cultures and antibiotics. What clinical finding would the nurse look for in this patient as evidence of an optimal clinical response to rapid IV fluid administration?
A. CVP of 4 mmHg
B. Lactate level less than 4 mmol/L
C. Urine output of 25 ml/hr
D. Cool, dry skin

Exam Question
A patient admitted after a MVC, in which he was the driver, now complains of severe left shoulder pain when lying supine. There are bruises across the abdomen and chest from the seat belts. VS: BP 120/80; HR 112 bpm; RR 18 breaths/min. The nurse is reassessing the patient. Which of the following findings should the nurse interpret as an emergency situation?
A. Epigastric pain with belching
B. Abdominal distention with absent bowel sounds
C. Decreased breath sounds bilaterally
D. Pain and burning with hematuria

References


