AACN PCCN Review

Gastrointestinal

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I. INTRODUCTION

PCCN Test Plan

Endocrine, Heme, GI, & Renal: 18%

a. Functional GI Disorders (e.g., obstruction, ileus, diabetic gastroparesis, gastroesophageal reflux, irritable bowel syndrome)
b. GI Bleed
   - Lower
   - Upper
c. GI Infections
d. Hepatic Failure
e. Ischemic Bowel
f. Malnutrition (e.g., failure to thrive, malabsorption disorders)
g. Pancreatitis

Structures/Function/Digestion

a. Mouth
b. Esophagus
c. Stomach
d. Small Intestine
e. Pancreas
f. Gallbladder
g. Liver
h. Spleen
i. Portal Circulation
j. Mesentery Circulation
k. Large Intestine
l. Digestive Hormones
m. Digestive Enzymes

Assessment

a. Inspection
b. Auscultation
c. Palpation
d. Percussion
II. THE HEPATIC SYSTEM

Liver Function

a. Metabolic Factory & Waste Disposal Plant
b. Carbohydrate, Fat & Protein Metabolism
c. Production of Bile Salts
d. Production of Clotting Factors
e. Bilirubin Metabolism
f. Detoxification: Nutrients, Drugs, Toxins, Bacteria, Everything
g. Vitamin & Mineral Storage:
h. Blood Reservoir: 10% of Total Blood Volume

Liver Function Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Proteins</td>
<td>• Total Protein: 6.0 – 8.0 g/dL</td>
</tr>
<tr>
<td></td>
<td>• Serum Albumin: 3.5 – 5.0 g/dL</td>
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<tr>
<td></td>
<td>• Serum Globulins: 2.6 – 4.1 g/dL</td>
</tr>
<tr>
<td></td>
<td>• Pre-Albumin: 17 – 40 mg/dL</td>
</tr>
<tr>
<td>Serum Ammonia</td>
<td>• 19 – 60 mcg/dL</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>• Total Bilirubin: 0.1 – 1.2 mg/dL</td>
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<tr>
<td></td>
<td>• Unconjugated Bilirubin: 0.1 – 1.0 mg/dL</td>
</tr>
<tr>
<td></td>
<td>• Conjugated Bilirubin: 0.1 – 0.2 mg/dL</td>
</tr>
<tr>
<td>Coagulation Studies</td>
<td>• Indirectly reflect liver function</td>
</tr>
<tr>
<td></td>
<td>o PT</td>
</tr>
<tr>
<td></td>
<td>o PTT</td>
</tr>
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<td></td>
<td>o INR</td>
</tr>
<tr>
<td></td>
<td>o Bleeding Time</td>
</tr>
<tr>
<td></td>
<td>o ACT</td>
</tr>
<tr>
<td>Hepatic Enzymes</td>
<td>• ALP: 42 – 136 U/L</td>
</tr>
<tr>
<td></td>
<td>• GGT</td>
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<tr>
<td></td>
<td>o Men: 0–85 U/L</td>
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<td></td>
<td>o Women: 0-70 U/L</td>
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<td>• AST</td>
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<tr>
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<td>o Men: 15-40 U/L</td>
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<tr>
<td></td>
<td>o Women: 13-35U/L</td>
</tr>
<tr>
<td></td>
<td>• ALT</td>
</tr>
<tr>
<td></td>
<td>o Men: 10-55U/L</td>
</tr>
<tr>
<td></td>
<td>o Women: 7-30U/L</td>
</tr>
</tbody>
</table>
Liver Dysfunction & Failure

Pathophysiology
a. Liver Tissue (cells) are Destroyed and Replaced with Fibrotic Tissue
b. Functions are Altered
c. Organ Changes Shape
d. Vascular Flow is Obstructed
e. Portal Hypertension

Cirrhosis
A chronic progressive liver disease where diffuse fibrotic bands of connective tissue, distort the liver’s normal architecture and functional ability. The liver loses its ability to regulate fluids, metabolize waste, regulate coagulation and nutrition.
a. Causes
   • Alcoholic, Laennec's Portal, or Fatty
   • Post Necrotic: Toxic, Nodular, or Post Hepatic
   • Biliary: Cholangitic or Obstructive

Hepatitis
Widespread Inflammation of Liver Cells
a. Causes
   • Primary Viral – Most Common
   • Hepatotoxins - Toxic or Drugs
   • Secondary Viral, Low Mortality

Clinical Presentation of Liver Dysfunction

Hepatic Encephalopathy
The liver is unable to perform its detoxification function and toxins build up. Primarily ammonia causing altered LOC, behavior and motor abilities.
a. Clinical Presentation
   • Confusion → Coma
   • Agitation → Unsafe Behavior
   • Asterixis: Flap like Tremor of Hands
   • Apraxia: Inability to Perform Purposeful Acts
   • Elevated Ammonia
b. Common Treatment Modalities
   • Limit Protein Intake
   • Limit Hepatotoxic Drugs
   • Lactulose & Neomycin
   • Safe Environment
Malnutrition
The liver is unable to perform its function of carbohydrate, protein and fat metabolism.
This leads to malnutrition
a. Clinical Presentation
b. Common Treatment Modalities
   - Need to tx the Cause of Liver Failure
   - Parenteral Nutrition
   - Limit Protein Intake
   - Restrict Fluids

Coagulopathy
The liver is unable to synthesize fibrinogen, prothrombin and factors V, VII, IX, X, XI, XIII, fibrinolytic factors and Vit. K. These are needed to maintain the ability to clot. Platelet aggregation and adhesion are also effected by liver dysfunction.
a. Clinical Presentation
   - Bleeding Tendencies
   - Nonspecific Bleeding
b. Common Treatment Modalities
   - Monitor Coagulation Studies & Platelet Ct
   - Decrease Bleeding and Bruising Risk
   - Administer Blood Products

Portal Hypertension
Increased pressure in the portal vein occurs secondary to flow obstruction from inflammation, bands, or fibrotic hepatic tissue. This retrograde pressure leads to formation of varices in the esophagus, stomach and rectal vault.
a. Clinical Presentation
   - Caput Medusae: dilated cutaneous veins radiating from the umbilical
   - Spider angiomas commonly seen in Cirrhosis
   - Upper GI Bleeding
b. Common Treatment Modalities
   - Surgical Shunting
   - TIPSS - Transjugular Intrahepatic Portosystemic Stent Shunt
   - Treat Bleeding
   - Treat Cause

Hepatorenal Syndrome
A form of pre-renal failure caused by the liver dysfunction. Mortality of liver failure is very high once renal failure develops.
a. Clinical Presentation
   - S&S of Renal Dysfunction
b. Common Treatment Modalities
   - Maintain Adequate Renal Perfusion
   - Restrict Fluids
   - Restrict Nephrotoxic Agents
   - Continuous Renal Replacement Therapies

**Ascites**
Fluid accumulation in the peritoneal space secondary to decreased production of albumin, decreased systemic oncotic pressure, increased hepatic lymph production and increased capillary permeability. The fluid accumulation impacts the respiratory (diaphragm) and cardiac (hemodynamic) systems primarily as well as comfort and body image.

a. Clinical Presentation
   - Inc. Abdominal Girth
   - Hypotension and Tachycardia
   - Dyspnea, Orthopnea, Tachypnea
   - S&S of Dehydration
   - N&V

b. Common Treatment Modalities
   - Restrict PO Fluid
   - Diuretics (if tolerated hemodynamically)
   - Restrict Na
   - Respiratory Support
   - Paracentesis
   - Peritoneovenous Shunt Surgery

**Infection**
One of the functions of the liver cells (Kupffer cells) is to clean the blood of bacteria. With liver failure this function is not provided and bacteria builds up (primarily gram negative bugs) in the systemic circulation increasing the risk of infection.

a. Clinical Presentation
   - Poor Wound Healing
   - Increased Risk of Infection

b. Common Treatment Modalities
   - Heightened Prevention Measures
   - Abx Therapy – w Caution
III. THE PANCREAS

Function

a. Endocrine Functions
   • Synthesis & Release of Hormones:
     o Glycogen
     o Insulin
     o Gastrin

b. Exocrine Functions
   • Pancreatic Enzymes Break Down Protein, Starch & Fat. > 2L/day
   • Bicarbonate Raise pH

c. PNS, Gastrin & Hormones Regulate Secretions

Pancreatic Enzymes

a. Trypsin: Aids in Protein Digestion
b. Amylase: Aids in Carbohydrate Digestion
c. Lipase: Aids in Fat Digestion

Acute Pancreatitis

Pathophysiology

a. Auto Digestion
   • Tissue Damage
   • Fat Necrosis
   • Vascular Damage & Hemorrhage
   • Increased Capillary Permeability
   • Hypotension

b. Forms/Types
   • Edematous
   • Hemorrhagic

c. Classifications
   • Acute Pancreatitis
   • Recurrent Acute
   • Recurrent Chronic
   • Chronic Pancreatitis

Cause (blocked enzyme release)

a. Alcoholism
b. Biliary Stones
c. Hyperlipidemia
d. Abd Trauma
e. Infection (bacterial or viral)
f. Shock
g. Drugs (Most Common: Cyclosporine, Acetaminophen, Cimetidine, Steroids, Salicylates, Furosemide, Thiazides, Estrogens)

**Clinical Presentation**

a. Pain  
b. Low Grade Fever  
c. N&V  
d. Distended/Tender/Rigid Abd  
e. Guarding with Rebound Tenderness  
f. Jaundice  
g. Hypoactive Bowel Sounds  
h. Steatorrhea: bulky, pale, foul-smelling stools  
i. ? Ascites  
j. Hypovolemic Shock  
k. In Necrotizing Pancreatitis  
  • Cullen’s Sign:  
    o Bluish Discoloration Umbilical  
  • Grey Turner’s Sign:  
    o Bluish Discoloration Flanks

**Labs**

Underlined labs are the MOST diagnostic  
a. Hypocalcemia (classic sign)  
b. Low Ca, Mg, K  
c. Hyperglycemia  
d. Hyperbilirubinemia  
e. Hypertriglyceridemia  
f. Increased BUN & Creatinine  
g. Elevated Amylase  
h. Elevated Lipase  
i. Elevated LFTs  
j. Elevated WBC  
k. Decreased H/H  
l. ? Increased H/H

**Treatment Options**

a. Fluid Resuscitation  
b. Rest the Pancreas: NPO, NGT  
c. Pain Management  
d. Monitor & Replace Electrolytes  
e. Tx Multisystem  
f. Nutritional Support  
g. Surgery
IV. ACUTE GI HEMORRHAGE

Lower GI Bleeding

Not Typically Life Threatening

a. Causes
   • Diverticulitis
   • Angiodysplasia
   • Cancer
   • Hemorrhoids
   • Inflammatory Bowel Disease (Ulcerative Colitis; Crohn's Disease)
   • Bowel Infarction

Upper GI Bleeding

a. Causes
   • Peptic Ulcer Disease: Duodenal, Gastric and Stomal ulcers account for 50% bleeding episodes
   • Gastritis or Esophagitis
   • Esophageal Varices
   • Mallory -Weiss Syndrome

b. Clinical Presentation
   • Hematemesis
   • Melena
   • PUD
   • Distended & Tender Abd
   • Hyperactive Bowel Sounds
   • Hypovolemia
   • Shock

c. Assessment
   • H & H
   • Coags & Platelets
   • Hemoconcentration
   • Elevated BUN
   • LFTs
   • Endoscopy
   • Angiography
   • Raionuclide Scans

d. Treatment
   • NG Decompression/Lavage – Room Temp vs Iced
   • Fluid Resuscitation
   • Blood Product Admin
   • Endoscopic Sclerotherapy
V. DISORDERS OF THE BOWEL

Bowel Infarction

a. Etiology
   - Embolic or Thrombotic Occlusion
   - Typically from the Superior Mesenteric Artery
b. Clinical Presentation
   - Severe Epigastric Pain
   - Rebound Tenderness
   - Guarding & Rigidity
   - Stimulated Sympathetic Response from Pain
c. Treatment Options
   - Angiography to Identify/Confirm Occlusion
   - Surgery to Remove Occlusion & Dead Bowel

Bowel Obstruction

a. Etiology
   - Internal Lumen Obstruction ex. Tumor
   - External Lumen Obstruction ex. Adhesions
   - Emboli: no blood flow
   - Paralytic Ileus
Clinical Presentation

- Complete vs Partial
- Distended Edematous Bowel
- Fluid and Electrolytes Leaking from Bowel
- Elevated WBC
- Fever

Small Intestine
- Acute Pain w Sudden Onset
- N & V (movement on both ends)
- Wave-Like Hyperactive High Pitched Bowel Sounds
- May Have Some Gas or Feces
- Distention (mild)

Large Intestine
- Slow Onset Pain Progression Mild ➔ Severe, Lower Abd
- No N & V (nothing moving)
- No Stool
- Low Pitched Bowel Sounds
- Distention (large amount)

Treatment Options
- Diagnosis Obstruction by Hx, X-Ray, CT, Upper or Lower Barium Radiology Tests
- Pain Management
- IV Fluids
- Decompress w NG, Rectal or Intestinal Tube
- Abx
- NPO and Time (rest the bowel)
- Surgery

Perforation/Peritonitis

a. Etiology
- Gastric/Intestinal Contents Leak into Peritoneal Cavity
- Ulcer Perforation
- Diverticular Rupture
- Trauma
- Bowel Infarction

b. Clinical Presentation
- Infection/Sepsis (all the S&S)
- Sudden Onset of Severe Pain
- Rigid Abdomen w Rebound Tenderness
- Hypoactive Bowel Sounds ➔ No Bowel Sounds
c. Treatment Options
   • Surgery to Repair Cause & Clean Up
   • ABX
   • Fluids
   • Tx of Sepsis
   • Tx of MODS

**Functional GI Disorders**

a. Ileus
b. Diabetic Gastroparesis
c. Gastroesophageal Reflux
d. Irritable Bowel Syndrome