Management of Patients with Hepatic/Biliary Dysfunction

- Hepatitis
- Cirrhosis
- Gall Bladder Disease

Key Questions

- What is the role of Glisson’s capsule around the liver?
- What clinical manifestations occur when the patient has distention of the liver?
Functions of the Liver

- Metabolizes CHO, proteins, fat
- Synthesizes plasma proteins
- Stores vitamins, minerals & glucose
- Forms blood clotting factors
- Detoxifies drugs & toxins
- Produces & excretes bile
- Regulates hormone function
- Phagocytic: breakdown RBC’s, bacteria, etc
- Acts as reservoir for blood volume

Circulation of the liver

“Dual Blood Supply”

- Portal system
  - Hepatic veins drain liver & empty into IVC - 1000-1200 ml/min
    (rich in nutrients)
  - Hepatic artery
    - 400-500 ml/min blood flow
    - Oxygenated blood

- Portal Vein
  - Receives 1050 mL/min from
    - Spleen
    - Intestines
    - Pancreas
    - Stomach
    - Incompletely saturated
  - Supplies 60-70% O₂ needs
  - Empty into IVC
  - Stores 450 mL blood that can be shifted in times of stress
Overview of liver pathophysiology

Inflammation
- Edema
- ↑ pressure

Obstruction
- ↑ internal pressure
- ↑ external pressure

Hepatocellular damage
- ↓ breakdown of urea → ↑ NH₃ → encephalopathy
- ↓ absorption of fat soluble vitamins (Vit. K) → ↓ synthesis of clotting factors → bleeding
- ↓ synthesis of plasma proteins (albumin) → malnutrition & edema

Diagnostic Tests - non-invasive

Non invasive: LFT’s to rule out Pathology-
- Enzymes
- Proteins
- Prothrombin time
- CBC
- ↑ ALP, LDH, GGT, AST, ALT
- ↑ serum & urinary bilirubin
- ↓ serum albumin & proteins
- ↑ Prothrombin time
- ↓ platelet count

Diagnostic Tests - Invasive

- Liver Bx
- Nursing role
- Angiograms

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Hepatitis

- Hepatitis A (HAV)
  - Contaminated food or water
  - 15-50 days
  - Communicable: 1-2 weeks post-symptoms
- Hepatitis B (HBV)
  - Blood transfusion
  - IV drug abuse
  - Sexual contact
  - Hemodialysis, HCW
  - 14-180 days
- Hepatitis C (HCV)
  - Non-A, Non-B

You are the home care nurse evaluating a 45-year-old RN after her discharge from the hospital for acute liver failure.

Previous history includes:
- Working in a munitions factory
- OSHA compliance officer monitoring chemical regs
- Auto accident (age 16) with multiple transfusions
- Frequent “socializing” with alcohol intake
- Frequent intake of shell fish

What are her risk factors for Hep. A, B, C?

Case Situation (continued)

Clinical manifestations include:
- Mild confusion
- Jaundice
- Pruritis
- Chronic fatigue

What is the pathophysiological cause of each of her clinical manifestations?
Pathophysiology of Hepatitis

Liver damage
- Inflammation
- Cellular degeneration
- Cellular necrosis
- Interruption of bile flow
- Stone development

Impaired function

Clinical manifestations - similar

Preicteric
- Non-specific
- RUQ pain
- Anemia
- Bruising/bleeding

Icteric
- Altered bilirubin excretion

Posticteric
- Fatigue
- Relapses
- Jaundice

Diagnosis of Hepatitis

Specific viral serological markers
- (Surface antigens)
  - Current infection
  - Carrier state

Antibodies
- Current or recent infection
- Carrier state

IgM = acute infection
IgG = past exposure
probable immunity
Diagnosis of Hepatitis - lab findings

Laboratory tests

- ↑ ALP, LDH, GGT, AST, ALT
- ↑ serum & urinary bilirubin
- ↓ serum albumin & proteins
- ↑ Prothrombin time
- ↓ platelet count

Prevention

Eliminate exposure

- Fecal/oral routes
- Contact with infected blood or body fluids
- Safer sexual contact
- Mother/newborn exposure
- Needle Exchange Prgms

Vaccines

- A
- B
- C (unavailable)
- D (protected by Hep B vaccine)

Nursing Diagnoses

- Activity Intolerance
- Fatigue
- Altered Nutrition
- Risk for infection r/t ↓ immune function
- Risk for transmission
- Ineffective health maintenance

- Physical & emotional rest
- ↓ fat w/ vit. Supp.
- Protein may be restricted
- ↓ exposure
- ↓ invasive procedures
- Patient & family education
Drug Therapy
- Hepatotoxic drugs
  - Alpha interferon
  - Ribavirin
  - Immune globulin
- Vaccines

Flu-like symptoms for HCV patients on interferon
Take medication at night

Complications of Hepatitis
HAV & HBV
- Most acute cases resolve without complications
- Chronic active (mild/mod./severe) hepatitis may progress to cirrhosis
- Chronic persistent (minimal/mild) hepatitis has a delayed convalescent period
- Fulminant hepatitis is a complication of HBV that leads to liver failure

Cirrhosis
Diffuse fibrotic bands of connective tissue in response to inflammation
Distorts normal architecture and function
Cirrhosis of the Liver

Pathophysiology

- Degeneration
- Destruction
- Necrosis

- Regeneration attempts
- Nodule (scar) formation

Compression of vascular system & lymphatic bile duct channels

Fibrous tissue proliferation in a disorganized pattern

Altered flow
Poor cellular nutrition
Hepatocellular hypoxia

Cirrhosis - 4 Types

- Alcoholic (Laennec’s)
  - Long term ETOH abuse

- Biliary
  - Chronic biliary obstruction
  - Bile stasis
  - Inflammation

- Post necrotic - Massive hepatic cell necrosis
  - Post viral hepatitis
  - Toxic exposure
  - Autoimmune process

- Cardiac
  - Severe RHF
  - Corpulmonale
  - Constrictive pericarditis
  - Tricuspid insufficiency

Cirrhosis
Early Clinical Manifestations:

- GI disturbances
  - Anorexia
  - Dyspepsia
  - Flatulence
  - Nausea & vomiting
  - Abnormal bowel habits

- Abdominal pain
  - Dull, heavy
  - RUQ or epigastrum

- Altered metabolism of fats, CHO, proteins
- Swelling/stretching of liver capsule
- Spasm of biliary ducts
- Intermittent vascular spasm
Additional Early CM’s:

- Fever
- Lassitude
- Slight weight loss
- Hepatosplenomegaly
- Palpable liver
- Jaundice
- Pruritus
- Amber-Colored Urine

Cirrhosis Later Clinical Manifestations:

- Jaundice
- Skin Lesions
- Hematologic Problems
- Endocrine Disturbances
- Peripheral Neuropathy

Diagnosis

- Liver function studies
  - enzymes
  - proteins
  - cholesterol
  - prothrombin time
  - Liver may be contracted or enlarged
- Invasive studies
  - liver biopsy
  - angiograms

Liver Biopsy Site
Normal Bilirubin Excretion (absence of Jaundice)

Breakdown of Hb bilirubin (non water-soluble).
Carried by albumin to the liver for conjugation where it is made water-soluble.

Lab tests:
- Indirect = unconjugated, BU or pre-hepatic
- Direct = conjugated, BC or post-hepatic

Urobilinogen is the breakdown of conjugated bilirubin that is excreted in the urine (small amount) and feces (most).

Bilirubin alterations:

Unconjugated
- "indirect" bilirubin
  - Overproduction d/t Hemolysis
  - Impaired hepatic intake d/t certain drugs
  - Impaired conjugation by glucuronide

Conjugated
- "direct" bilirubin
  - Impaired excretion of bilirubin from liver d/t hepatocellular disease
    - Drugs
    - Sepsis
    - Hereditary disorders
    - Extra-hepatic biliary obstruction

RBC - bilirubin in bloodstream
- In blood stream to liver
- Liver - bilirubin releases from albumin, combines with glucuronic acid (conjugation)
- Small amount
- Conjugated enters circulation
- Excreted via kidneys
- To intestine in bile
- Intestine - bilirubin converted to urobilinogen & stercobilinogen
- Excreted in stool
- Bilirubin joins with albumin
- Unconjugated

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**Lab Test Abnormalities**

**Cirrhosis**
- ↑ ALP, LDH, GGT, AST, ALT indicate liver damage or altered function
- ↑ serum bilirubin
- urinary bilirubin
- ↑ PT
- ↓ platelet count
- ↓ serum albumin & proteins

**Jaundice**
- Inability of liver to conjugate bilirubin
- Bilirubin- bile pigment from breakdown of Hb from RBC's by macrophages
- Skin & sclera - jaundice
- Excreted in urine - tea colored urine
- Blocked from flow into intestines - clay colored stools

**What changes do you see and why?**
- Skin & Sclera - Jaundice
- Urine - Amber or Tea colored
- Stool - Clay colored

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Skin Lesions

- Spider angiomas
  - Small, dilated blood vessels with red center and spider-leg like branches
  - ↑ in circulating estrogen
  - d/t ↓ ability of liver to metabolize steroids

- Palmar erythema
  - Reddened palms that blanch with pressure

Hematologic Problems

- Thrombocytopenia
- Leukopenia
- Anemia

  - d/t splenomegaly
  - back up of blood from portal vein into spleen
  - Overactivity of enlarged spleen - ↑ removal of blood cells from circulation

  - Coagulation defects
  - d/t liver's inability to produce Prothrombin and other clotting factors
  - d/t ↓ synthesis of bile fats → ↓ absorption of fat soluble vits
  - Without Vit. K, clotting factor production ↓

Endocrine Problems

- Gynecomastia
- Loss of axillary/pubic hair
- Testicular atrophy
- ↓ libido
- Impotence
- Amenorrhea/vaginal bleeding

- Hyperaldosteronism
  - ↑ Na+
  - ↑ H₂O
  - ↓ K⁺
Peripheral Neuropathy—more common with Alcoholic Cirrhosis

- Mixed form
- Sensory predominant

- Dietary ↓ of
  - Thiamine
  - Folic acid
  - Cobalamin—Vit. B12

Complications of Cirrhosis

- Portal Hypertension
- Esophageal Varices
- Ascites
- Peripheral Edema
- Hepatic Encephalopathy
- Hepatorenal Syndrome

Portal Hypertension & Esophageal Varices

- Compression & destruction
  - Portal veins
  - Hepatic veins
  - Sinusoids

- Obstruction of normal flow through portal system → portal hypertension

- Collateral circulation develops primarily in
  - Lower esophagus
  - Anterior abdominal wall
  - Rectum
  - Parietal peritoneum

- Collateral circulation develops to avoid varices
  - Portal pressure ↓
  - Plasma volume ↓
  - Lymphatic flow ↓
Esophageal Varices

- **Collateral Circulation** due to portal hypertension

**Lower Esophagus**
- Abdominal Wall
- Rectum
- Esophageal Varices
- Caput Medusae
- Hemorrhoids

**Risk for bleeding**
- Fragile, inelastic, thin-walled esophageal veins become distended or irritated leading to rupture

**Chemical irritants**
- Alcohol
- Medications
- Refluxed gastric acid

**Mechanical trauma**
- Poorly chewed, coarse food
- Vomiting
- N/G insertion

**Increased esophageal pressure**
- Vigorous exercise, heavy lifting
- Coughing, sneezing
- Retching/vomiting
- Straining at stool

**Medical Management**

- **Prevent initial hemorrhage**
- **Manage acute hemorrhage**
- **Prevent recurrent hemorrhage**
Prevent initial hemorrhage

Pharmacological Mgt.
- β-blockers
- ↓ portal pressure
- ↓ flow in collateral channels
- Stool softeners
- H-2 blockers, PPI's

Dietary Modifications
- ☒ alcohol
- ☐ caffeine
- ☐ spicy foods
- ☒ coarse foods

Manage acute hemorrhage

65-75% of cirrhotic patients develop esophageal varices. Ruptured varices have a 30-60% mortality rate

Pharmacological Mgt.
- Vasopressin/NTP
- Octreotide

Endoscopic injection sclerotherapy
- Band Ligation

Supportive Rx
- FFP, RBC's
- Vit. K
- H2 blockers
- Neomycin

Nursing Management

Impaired Gas Exchange r/t ↓ O2 exchange 2°

- Aspiration Pneumonia
  - Assure suction port
  - Suction frequently
- Nares Erosion
  - Clean, lubricate external nares
  - Pad if necessary
- Airway Obstruction

occurring after balloon tamponade with Sengstaken-Blakemore tube
Prevent recurrent hemorrhage

Shunts
- ↓ portal pressure
- divert flow away from collateral channels
- send portal venous blood directly to IVC bypassing liver

Complications
- Hepatic encephalopathy
- Heart Failure
- Bacteremia
- Shunt Clotting

Shunts: Post Operative Priority

Potential complication:
- Shunt clotting

Assessment:
- pain
- distention
- nausea
Shunts: Post - Operative Priority

Fluid Volume ↑↑ r/t retention of fluids 2°
- Portal hypertension
- Liver failure
- Hemodilution r/t new shunt

Outcomes: normovolemia:
- Stable or ↓ abd. Girth
- Regular resp. rate/rhythm
- Unlabored breathing
- Output ≥ intake

Interventions: Assess
- Abd girth, Weight, I/O
- Edema
- Pulm. Ed.: dyspnea, orthopnea

Shunts - post-op complications

Hepatic encephalopathy
- NHs
- Bilirubin
- Liver enzymes

Post-op hemorrhage
- Hb, Hct, Pro-times

Other labs
- Renal - BUN
- Electrolytes
- Serum Proteins

Heart failure
- Shunt ↑ pre-load

Anticipate:
- Transfusions
- Vit. K
- PN
- Albumin IV

Ascites - Pathophysiology/Interventions

Protein (PRO) leak through liver capsule to peritoneal cavity → oncotic pressure of PRO pulls more fluid

↓ albuminemia d/t liver’s inability to synthesize PRO → ↓ colloidal osmotic pressure

↑ aldosteronism d/t liver’s inability to metabolize aldosterone → ↑ Na reabsorption → ↑ serum osmolality → ↑ ADH secretion → ↑ water retention

- Semi ↑ Fowler’s Position
- ↓ Pro, ↑ Na diet
- oral care r/t dehydration
- K-sparing diuretics
- Salt Poor Albumin
- Paracentesis
- Peritoneovenous shunt
Ascites and Peripheral Edema

- Portal hypertension
  - protein & plasma “leak” into the peritoneum
  - osmotic pressure pulls more fluid in
- Hypoalbuminemia
- Hyperaldosteronism

Therapeutic Goals & Outcomes

- ↓ metabolic demand on the liver
- Treat complications
  - Balanced fluid volume
  - Absence of breathing problems
  - Corrected coagulation defects
  - Absence of infection
  - Adequate nutritional intake
  - Normal LOC

Portal Systemic Encephalopathy

Build up of NH$_3$ in serum and CSF → neurotoxicity
- Altered LOC
- Impaired thinking
- Neuromuscular disturbance
- Early Sign: Change in handwriting

Manage with:
- Neomycin & Lactulose=reduce bacterial action on feces which ↓ NH$_3$ production

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Hepatorenal Syndrome
CM’s & Pathophysiology

- Azotemia (↑ BUN, creatinine)
- Sudden oliguria
- Intractable ascites
- Redistribution of blood flow from kidneys to peripheral & splanchic
- Hypovolemia d/t ascites
- Intrarenal imbalance of vasoconstriction & vasodilating mechanisms d/t Liver disease

Hepatorenal Syndrome
Risks & management

Precipitants:
- Overly vigorous Diuretics
- GI/Verteal hemorrhage
- Paracentesis
- Hepatic encephalopathy
- NSAID’s
- Sepsis

Treatments:
- Salt Poor Albumin
- Na & H2O restriction
- Diuretic therapy

Alcohol Withdrawal Syndrome
(48-72 Hours after last Drink)

Facts
- Hidden disease
- Potent CNS depressant
- Withdrawal awakens SNS
- Untreated or undertreated ETOH withdrawal → ↑ mortality and morbidity
- Delirium-Tremens (DT’s) can be a life-threatening medical condition

Clinical Manifestations
- Tremor/shakiness
- ↑ VS
- Diaphoresis
- Agitation, Anxiety
- GI
- Confusion
- Sleep disturbance
- Hallucinations
- Seizures
**Alcohol Withdrawal - Goals**

- ↓ patient discomfort
- ↓ dangerous cm's
- Prevent complications
- Prepare patient for rehabilitation

**Consider primary diagnosis**

- Admission assessment
- Frequent monitoring
- Prompt & adequate treatment
- Benzodiazepines

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**Hepatocellular Carcinoma**

- Surgical resection
  - Lobectomy
  - Hepatectomy
- Chemotherapy
  - Portal vein or Hepatic Artery perfusion
  - 5-FU, Adriamycin
- Palliative Care
  - Same as for cirrhosis

**Liver CA - treatment & survival**

- Transplantation
  - Cirrhosis d/t hepatitis viruses
  - Hepatic malignancy confined to liver
  - Congenital diseases

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The Biliary Tract

Function of the Gallbladder
- Concentration and storage of bile produced by the liver
- Bile release stimulated by presence of food in GI tract

Disorders of the Gallbladder
- Cholelithiasis
  - cholesterol, bile and calcium stone formation
- Cholecystitis
  - inflammation and/or obstruction
  - stones
  - bacterial
Clinical Manifestations

- Indigestion & fat intolerance
- Steatorrhea (fatty stools)
- Moderate to severe pain
  - "biliary colic" RUQ abdominal tenderness
  - Referred pain to right shoulder and scapula
- Nausea and vomiting
  - "silent cholelithiasis"
- Temperature, ↑ WBCs
- Jaundice
- Dark urine
- Clay-colored stools
- Pruritis
- Bleeding tendencies

Diagnosis

- History
- Ultrasound
- Oral cholecystograms
- Percutaneous transhepatic cholangiography
- Endoscopic retrograde cholangiopancreatography (ERCP)
- Lab studies
  - Elevated direct and indirect bilirubin
  - Elevated AST (aspartate aminotransferase) (SGOT)

Treatment

- Cholecystitis (conservative)
  - Pain control
  - Anti-nausea meds
  - Antibiotics
  - NG tube
  - Diet restrictions/ NPO
  - Anticholinergics
  - Fat soluble vitamins (A, D, E, K)

- Cholelithiasis
  - Dissolve stones
  - Endoscopic intervention
  - Extracorporeal shockwave lithotripsy (ESWL)
Surgical Intervention

- Laparoscopic Surgery
  - preferred treatment
- Open (incisional) Cholecystectomy
  - for more complicated cases
Post-Operative Care

- Laparoscopic
  - pain management
  - meds
  - Sim's position
  - mobility
  - C&D
  - DC teaching
  - activity & diet

- Open or incisional
  - pain management
  - mobility
  - C&D
  - wound care
  - T-tube monitoring
  - DC teaching
  - activity & diet

Care & Teaching: T-tube

- Keep bag level w/abd
- Prevent tension
- Monitor output
- Skin site care
- Clamp 1-2 hr ac and unclamp 1-2 hr pc
- Unclamp if distress
- Time: Approx. 10 days
References

- Medical-Surgical Nursing, Clinical Management for Positive Outcomes, Black, J., Hawks, J., 8th Ed., 2009 Saunders
- Pathophysiology, Copstead, L., Banasik, J., 3rd Ed., Elsevier
- Mosby’s Medical & Nursing Dictionary 1983 Mosby Co., St. Louis