LIVER DYSFUNCTION

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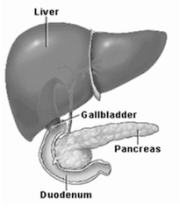
OBJECTIVES

- Discuss the etiology, risk factors and incidence of liver failure and hepatitis
- Discuss the clinical manifestations of liver failure, hepatitis, alcohol withdrawal
- Discuss diagnostic studies for liver dysfunction
- Discuss the therapeutic management for liver failure, hepatitis, alcohol withdrawal



REVIEW OF FUNCTIONS

VASCULAR

- Blood Storage
- Blood Filtration

METABOLIC

- Carbohydrate
- Protein
- Fat
- Detoxification
- Steroid metabolism

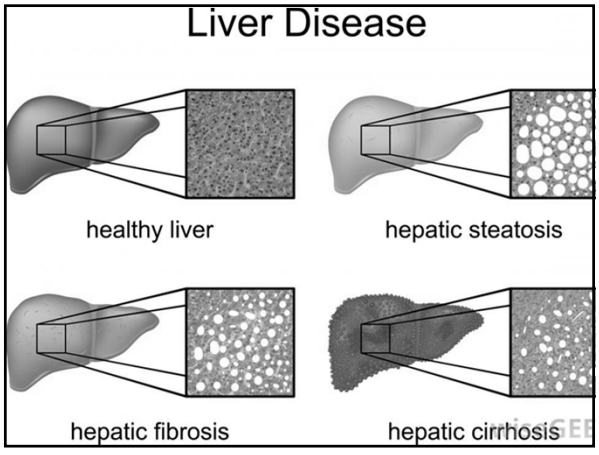
REVIEW OF FUNCTIONS

STORAGE

- Blood
- Glucose
- Vitamins
- Fat
- Minerals
- Proteins

MONONUCLER PHAGOCYTE SYSTEM

- Breakdown of RBC's. WBC's and bacteria
- Breakdown of hemoglobin from old RBC's to bilirubin



CIRRHOSIS

A chronic progressive disease of the liver characterized by excessive degeneration and destruction of the liver cells.

TYPES OF CIRRHOSIS

ALCOHOLIC

(Portal or Nutritional)

- Associated with ETOH abuse
- Accumulation of fat in the liver cells
- Fatty changes are potentially reversible

POSTNECROTIC

- Massive necrosis of the liver following viral infection, metabolic disorders, and exposure to industrial chemicals and hepatoxins, NAFLD or idiopathic.

TYPES OF CIRRHOSIS

BILIARY

- Associated with chronic biliary obstruction and infection.
- There is diffused fibrosis of the liver with jaundice as the main feature.

CARDIAC

- Chronic liver disease results from long term severe right sided heart failure associated with cor pulmonale, constrictive pericarditis, and tricuspid insufficiency.

TYPES OF CIRRHOSIS

Primary Biliary Cirrhosis

- Chronic inflammatory condition
- Has genetic and environmental factors
- T-cell mediated attack of the small bile duct epithelial cells

Nonalcoholic Fatty Liver Disease (NAFLD)

- Group of disorders characterized by hepatic steatosis
- Fatty changes in the hepatocytes
- Inflammation and scaring that is called nonalcoholic steatohepatitis (NASH)

DIAGNOSTIC TEST

- Liver function test (AST, ALT, GGT)
- APTT, PT, INR
- Total protein, albumin, MP, Y-globulins
- Bilirubin (T. Bili and D. Bili)
- Cholesterol (↓ cholesterol levels)
- Ammonia levels
- CT, liver scan, liver biopsy
- EGD, Abdominal ultra sound

CLASSIFICATIONS CHILD CIRRHOSIS

- Child-Pugh is a classification system.
- This scoring system is used to determine the prognosis with cirrhosis.
- Scoring is based upon : albumin, ascites, total bilirubin, PT, and encephalopathy.

Classification

- Class **A** is a score 5-6
- Class **B** is a score 7-9
- Class **C** is a score of > 9
- Clients with a score of 10 or more have a prognosis with a 1-year survival being about 50%.
- Class A or B have a prognosis of 5-years.

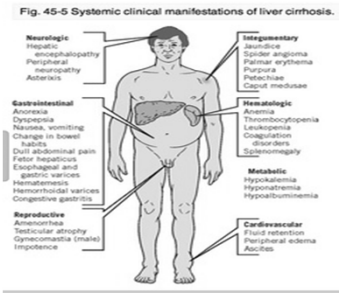
CLASSIFICATION
M.E.L.D

A scoring system for assessing the severity of chronic liver disease. It was initially developed to predict death within three months of surgery in patients who had undergone a transjugular intrahepatic portosystem shunts (TIPS) procedure.

It was subsequently found to be useful in determining and prioritizing for receipt of a liver transplant.

MELD

40 or more---71.3% mortality
30-39----52.6% mortality
20-29----19.6% mortality
10-19----6.0% mortality
0-9 ------1.9% mortality



CLINICAL MANIFESTATIONS (EARLY)

- Onset usually insidious
- Anorexia
- Dyspepsia
- Flatulence
- Nausea, vomiting
- Change in bowel habits (diarrhea, constipation)
- Abdominal pain (dull, heavy feeling)
- (S/S usually due to the livers inability to metabolize carbs, fat, and protein)
- Palpable liver

CLINICAL MANIFESTATIONS (LATE)

NEROLOGIC

- Hyperactive reflexes
- (+) Babinski
- Asterixis (Liver flaps)
- Altered LOC
- Peripheral neuropathy d/t dietary deficiency

JAUNDICE

- Occurs as a result of liver's decreased ability to conjugate and excrete bilirubin.
- Pruritus: Bile salt accumulation under the skin

LATE MANIFESTATIONS

SKIN LESIONS

- *Spider Angiomas* are small dilated blood vessels with a bright red center.
- *Palmar Erythema* a red area that blanches with pressure located on the palms of the hands.

HEMATOLOGIC DISORDERS

- Thrombocytopenia
- Leukopenia
- Anemia
- Coagulation disorders
- Splenomegaly

LATE MANIFESTATIONS

ENDOCRINE PROBLEMS

- The liver is unable to metabolize or inactivate various adrenocortical hormones, estrogen, and testosterone.

OTHERS

- Fector hepaticus "liver breath"
- Hyperventilation
- Cardiac arrhythmias
- Peripheral edema
- Peripheral neuropathy

THERAPY

- Propranolol
- Lactulose
- Vitamin K
- Proton Pump inhibitors
- Diuretics
- Magnesium sulfate
- H2 blocker
- High calorie diet
- High carbs
- Protein 1 to 1.5 gm/kg/day
- Mod/low fat
- Sodium restriction 2gm
- Enteral formulas

COMPLICATIONS

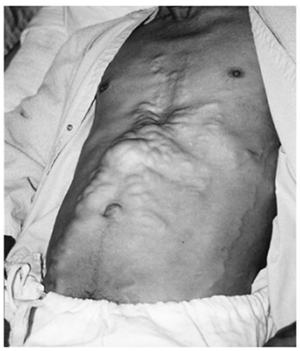
PORTAL HYPERTENSION
ESOPHAGEAL VARICES
ASCITES
HEPATIC ENCEPHALOPATHY
HEPATORENAL SYNDROME

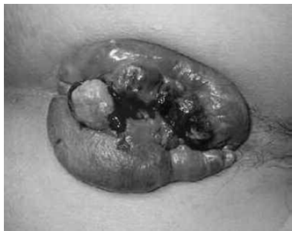
PORTAL HYPERTENSION

- Structural changes in the liver causes compression and destruction of the portal and hepatic sinusoids. This results in obstruction in the normal blood flow through the portal system.
- Collateral circulation develops to reduce the portal pressure and plasma volume.

PORTAL HYPERTENSION

- Common areas of collateral channels are the lower esophagus, anterior abdominal wall, parietal peritoneum, and the rectum.
- Varicosities develop in areas were collateral and system circulation communicate resulting in esophageal and gastric varices, *caput medusae* and *hemorrhoids*.



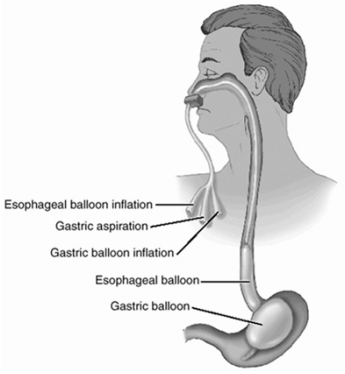


ESOPHAGEAL VARICES

- Collateral vessels contain little elastic tissue and are quite fragile.
- Vessels tolerate high pressure poorly, which result in distended, tortuous veins that bleed easily.
- The varices rupture and bleed in response to ulceration and irritation. Varices are life threatening complication of cirrhosis.

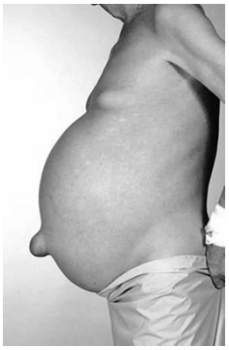
TREATMENT MODALITIES
Esophageal Varices

- Avoidance of bleeding
- Avoid ASA, ETOH, irritating foods
- Treat respiratory infections promptly
- Drug therapy
- Beta blockers
- H2 blockers, PPI
- Vitamin K
- Blood products
- Balloon tamponade
- Room temperature/ice water lavage
- Sclerotherapy or banding
- Ligation of varices
- Shunt therapy
- Lactulose
- Neomycin



ASCITES

- Accumulation of serous fluid in the peritoneal or abdominal cavity.
- Lymphatic system is unable to carry excess proteins and water, therefore they leak through the liver capsule into peritoneal.
- Osmotic pressure from the proteins pulls additional fluid into the peritoneal.

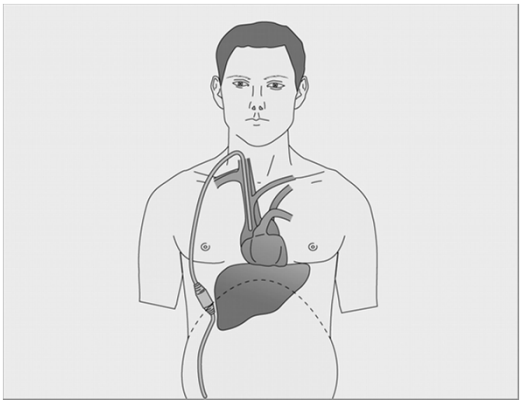


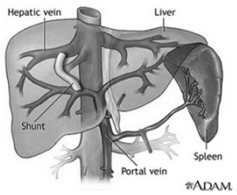
ASCITES

- ***Hypoalbuminemia*** resulting from the liver's inability to synthesize albumin. This results in ↓ colloidal osmotic pressure.
- ***Hyperaldosteronism*** results when aldosterone is not metabolized by the damage liver. The ↑ level of aldosterone causes ↑ sodium and water and ADH.

TREATMENT MODALITIES
ASCITES

- Diet (sodium restriction 250-500 mg/day)
- Diuretics (Spironolactone. Lasix, Amiloride)
- Bedrest (produces diuresis)
- Paracentesis
- Albumin
- Peritoneovenous Shunts
  La Veen shunt
  Denver Shunt
  Transjugular intrahepatic portosystemic shunts (TIPS)





HEPATIC ENCEPHALOPATHY

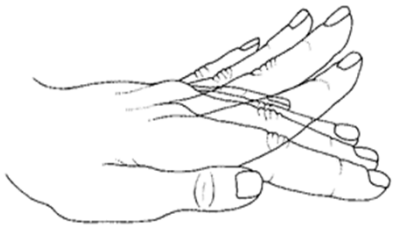
- Terminal complication of liver disease
- A disorder of protein metabolism
- Ammonia enters the systemic circulation without liver detoxification (neurotoxin)
- Major source of ammonia is the bacterial and enzymatic deamination of amino acids in the intestines. Ammonia is normally converted by the liver into urea and is excreted via kidneys.

HEPATIC ENCEPHALOPATHY

- When blood is shunted past the liver or the liver is unable to convert ammonia to urea, large quantities of ammonia remain in the systemic circulation.
- The ammonia crosses the *blood-brain barrier* and produces neurologic toxic manifestations.

MANIFESTATIONS (ENCEPHALOPATHY)

- Euphoria
- Depression
- Apathy
- Memory loss
- Confusion
- Yawning
- Drowsiness
- Slow slurred speech
- Hyperactive reflexes
- Asterixis
- Fector Hepaticus
- Hyperventilation
- Deep and slow respirations
- Coma



TREATMENT MODALITIES (ENCEPHALOPATHY)

- Ammonia Reduction
- Protein may be restricted
- 3000 kcal/day
- ↑ carbohydrate intake 1500-2000 cal/day
- Low/mod fat
- Sterilization of intestine
- Neomycin 1-3 grams tid (po, rectal)
- rifaximin, vancomycin
- metronidazole
- Sodium restriction
- Lactulose
- Peritovenous shunts
- Liver transplant

Nursing Care Plan

- Health Promotion
- Adequate Nutrition (Small frequent meals)
- Rest (ROM exercise)
- Skin care (turning, specialty beds, air mattress)
- I&O, monitor labs
- Daily weights
- Measurement of ascites
- Positioning

HEPATORENAL SYNDROME

- There is no structural abnormalities of the kidney
- Functional renal failure with advancing azotemia, oliguria, and intractable ascities
- Due to redistribution of blood flow from the kidney to the systemic and splanchnic vasodilation, there is ↓arterial blood volume. Renal vasoconstriction occurs and renal failure follows.

HEPATITIS

- Inflammation of the liver secondary to a virus. Viral hepatitis is the most common cause of hepatitis. The types of hepatitis are A, B, C, D, E, and G.
- Hepatitis may also be caused by ETOH, drugs, chemicals, and autoimmune liver diseases.
- Rarely is hepatitis caused by a bacteria

Diagnostic Test

Ag= antigen	Ab= antibody Anti= antibody
IgM= antigen	IgG= antibody
This is recognition of the viral load	Antibodies show lifetime immunity or previous infection

HEPATITIS

Hepatitis A

Route:
- Fecal oral

Prevention:
- HAVRIX vaccine

Prophylaxis:
- Immune globulin

Hepatitis B

Route:
- Bloodborne

Prevention:
- RECOBBIVAX vaccine

Prophylaxis:
- HBIG (Immune globulin)

HEPATITIS

Hepatitis C

Route:
- Bloodborne

Prevention:
- No vaccine

Prophylaxis
- No post-exposure prophylaxis

Hepatitis D

Route:
- Bloodborne

Prevention
- Same as HBV (Recombivax)

Prophylaxis
- Same as HBV (HBIG)

HEPATITIS

Hepatitis E

Route:
- Fecal-oral

Prevention:
- No vaccine
- Enteric precautions

Prophylaxis
- None

ACUTE PHASE OF HEPATITIS

- *Preicteric or Prodromal Phase*
  Last from 1-21 days
  Maximal infectivity for HAV

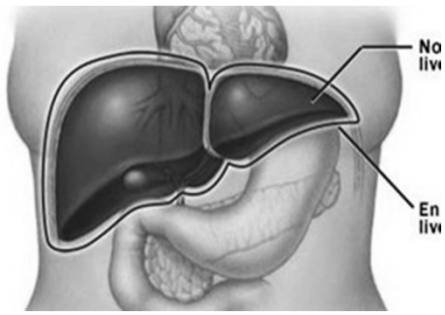
- *Icteric Phase*
  Last from 1 to 4 months
  Characterized by ***jaundice***, pruritus, dark urine
  May be icteric or anicteric

ACUTE PHASE OF HEPATITIS

- *Posticteric or Convalesent Phase*
  Begins when jaundice disappears and last months. The average length being 2-4 months.

SYMPTOMS

- Anorexia, N/V, weight loss, ↓*sense of smell*
- Abdominal discomfort (RUQ)
- Malaise, Arthralgias
- Skin rash, urticaria, *pruritus, jaundice*
- Low grade fever
- Hepatomegaly, splenomegaly
- ↑AST, ALT, GTT, ALK Phos, Bilirubin, APTT
- *Dark urine*, *light stools*



COMPLICATIONS

CHRONIC HEPATITIS

- Chronic HBV is identified by HBsAG for longer than 6 months.
- Evaluate by LFT's , HBV DNA, HBeAG and Anti-HBeAg.
- Liver biopsy may be required to assess inflammation and degree of fibrosis.
- Risk factor for development of hepatic carcinoma

CHRONIC HEPATITIS

- There is a greater risk for HCV infection to become chronic and develop into cirrhosis and hepatocellular carcinoma.

Symptoms

Easy fatigability, malaise, hepatomegaly remains but splenomegaly subsides

DRUG THERAPY (HBV)

- Focus is on decreasing the viral load and AST, ALT levels
- **INTERFERON**

  Blocks viral entry into cells, synthesis of viral proteins, and viral assembly and release.
- **NUCLEOSIDE ANALOGS (antivirals)**

  Suppress HBV replication (adefovir,lamivudine,tenofovir)

DRUG THERAPY (HCV)

- Drug therapy is directed at eradicating the virus, reducing viral load, and decreasing the progression of the disease.
- **INTERFERON**
- **RIBAVIRIN** (Has a synergistic effect when combined with interferon. Used to reduce the rate of relapse following interferon therapy for HCV.

NEW DRUG THERAPY FOR HCV

- Sofosbuvir (Sovaldi)
- Cost is a 1000 dollars per tablet
- Need 84 tablets (84,000 dollars) for a full course of therapy
- This cost is before mark up.

COLLABORATIVE CARE

- No specific treatment for acute viral hepatitis.
- Emphasis is placed on measure to ensure rest and assist the liver in regenerating.
- Rest reduces the metabolic demands on the liver and promotes cell regeneration

NUTRITIONAL THERAPY

- No special diet is needed
- Ensure there is enough calories to prevent weight loss.
- May require a decrease in fat content if poorly tolerated.
- Vitamin supplements (Vitamin B and K)
- Small frequent meals ↑carbs,protein,↓fats
- Adequate fluid intake: 2500-3000ml/day

INTERVENTIONS

JAUNDICE

- Comfort measures to relieve pruritus, HA, and arthralgias
- Psychologic and emotional rest is essential

FULMINANT HEPATIC FAILURE

- Active liver failure
- A clinical syndrome characterized by severe impairment of liver function associated with hepatic encephalopathy

Common Causes

Drugs

- Acetaminophen with ETOH
- INH
- Halothane
- Sulfa-containing drugs
- NSAIDS
- Mushroom posioning

Viral hepatitis

- HBV second most common cause of fulminant hepatitis
- HAV
- HCV (infrequent)

FULMINANT HEPATIC FAILURE

- Clinical syndrome that results in severe impairment of liver cells.
- Occurs more frequently with HBV and co-infection HDV. HAV can cause fulminant hepatic failure.
- Toxic reactions to drugs and congenital metabolic disorders cause fulminant hepatitis and liver failure

TOXIC AND DRUG INDUCED HEPATITIS

- Many substances, including ETOH, certain drugs, and other toxins, can directly damage the liver cells.
- The degree of the damage often depends on age and extent of exposure (dose) to the hepatotoxin.
- Acetaminophen overdose is the leading cause of acute liver failure

INTERVENTIONS

- Bed rest
- Measures to prevent problems of immobility
- Modify activities
- Oral hygiene
- Relieve itching Monitor urine
- I&O
- Daily weights
- Measure abdominal girth
- Assist with paracentesis
- Monitor fluid and electrolytes
- Positioning
- Skin care

HEALTH PROMOTION

- Outbreaks of viral hepatitis is usually due to HAV
- Preventive measures should be implemented.
- Hand washing is essential and the most important precaution.
- Vaccination is the best protection against HAV. All children > age 1 and adults at risk should receive the vaccination.
- Change at risk behavior (HBV, HCV)

LIVER DISEASE

Autoimmune

- A chronic disorder in which cell-mediated immune response directed against liver cells causes persistent inflammation and necrosis with fibrosis scarring.
- Many affected individuals have personal or family history of other autoimmune disorders.
- Circulating autoantibodies (ANA) are present

Wilson's Disease

- Familial, terminal neurologic disease accompanied by CLD
- Autosomal recessive
- Accumulation of cooper and hepatic cell injury
- S/S: corneal Kayser-Fleischer rings, movement disorders, neurological dysfunction, drooling, Sz

MANIFESTATIONS

- Changes in mentation are the first clinical signs. Cerebral edema, encephalopathy
- Jaundice
- Coagulation abnormalities
- Metabolic acidosis
- Renal failure
- Multiorgan failure

ALCOHOL WITHDRAWAL SYNDROME

In the USA an estimated 10% of the population have chronic alcoholism. Not all persons with chronic alcoholism experience clinically apparent alcohol withdrawal on cessation of alcohol consumption.

Approximately 1.2 million hospital admission are for problems related to alcohol abuse.

ALCOHOL WITHDRAWAL SYNDROME

Many drugs and medications produce withdrawal symptoms when their use is discontinued. Tolerance occurs when long-term use of a substance produces adaptive changes, so that larger amounts of the substance is needed to produce similar effects.

ASSESSMENT

- Type of alcohol ingested
- Duration of addiction
- Time of last ingestion
- Reason for stopping the drug
- Previous withdrawal symptoms
- Alternative treatments
- Medications
- Other medical problems

Withdrawal symptoms appear within 6-12 hours and usually relieved by consuming additional alcohol.

The "***classic hangover***" is likely an early and mild alcohol withdrawal as symptoms are promptly relieved by ingesting additional alcohol.

STAGES OF WITHDRAWAL

Stage I: Early withdrawal consists of mild mild anxiety and alcohol craving. ***6-12 hours***

Stage II: Intermediate severity, at *24-36* hours is characterized by excessive adrenergic effects.

STAGES OF WITHDRAWAL

Stage III: This stage consists of tonic-clonic seizures and occurs at *12-48* hrs.

Stage IV: Occurs *48-72* hours after alcohol intakes stops. This stage consists of DT's, often occurring following seizure.

ALCOHOLIC HALLUCINOSIS

Up to 25% of patients with a prolonged history of alcohol abuse experience hallucinations. They consist of persecutory, auditory, or more common visual hallucinations with an an otherwise clear sensorium.

ALCOHOL WITHDRAWAL SEIZURES

Seizures are usually brief generalized tonic-clonic type *without* an aura.

They are usually in a cluster of 1-3 seizures with a short postictal period.

30-50% of patients with seizures progress to DT's.

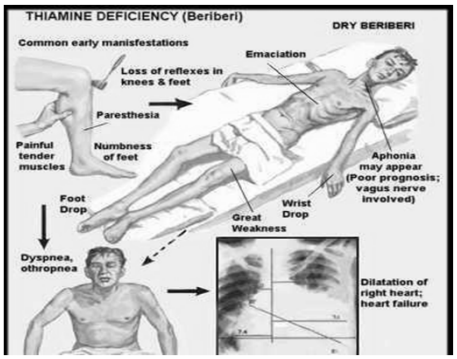
Incidence peaks 24 hours following the most recent ingestion/withdrawal of alcohol.

TREATMENT

- Benzodiazepines
- Cardiovascular agents
  Propranolol
  Clonidine
- Vitamins
  Thiamine
  Vitamin B-12
  MVI, Folic Acid
- Pharmacologic Antidotes
  Ethanol 10% solution
  50-75 mL/hr
- Magnesium Sulfate
  2 GM/liter
- Anesthetics
  Propofol

COMPLICATIONS BERIBERI

DRY	WET
Nervous system involvement .	Cardiovascular involvement
• Occurs with poor caloric intake	• Peripheral vasodilation
• Peripheral neuropathy\	• High cardiac output
• Loss of sensory, motor and reflex function	• Salt and water retention



Wernicke Encephalopathy
Wernicke-Korsakoff Syndrome

- Thiamine plays a large role in cerebral energy utilization. Thiamine dependent cellular system fails, resulting in cellular death.
- Symptoms include: confusion, disinterest, memory disturbance, nystamus, gaze palsies, gait ataxia, hypotension, hypothermia, and coma.

QUESTION

Which finding would strongly indicate the possibility of cirrhosis?

a. Dry skin
b. Hepatomegaly
c. Peripheral edema
d. Pruitus

QUESTION

Which assessment finding is consistent with cirrhosis?

a. ↑ Co2 levels
b. ↑ pH level
c. ↑ PTT
d. ↑ WBC

QUESTION

Which instruction would reduce the risk of hepatitis A to children in daycare?

a. Hand washing after diaper change
b. Isolation of sick children
c. Use of mask during contact with children
d. Sterilization of all eating utensils

QUESTION

Which symptom is common in viral hepatitis?

a. Arthralgia
b. Excitability
c. Headache
d. Polyphagia