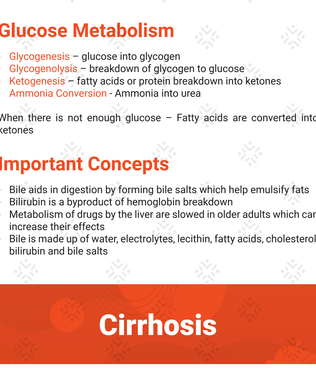
medpath

Hepatic System

The Liver

- Largest gland of the body
- Located in the RUQ of abdomen
- Stores glucose (as glycogen)
- Converts ammonia to urea for excretion by the kidneys
- Synthesizes blood proteins and clotting facrtors
- Stores vitamin A, D, K, iron and copper
- Makes bile which aids in digestion by emulsifying fats
- Metabolizes drugs and binds them to be excreted in urine

Hepatic Circulation



Glucose Metabolism

- Glycogenesis – glucose into glycogen
- Glycogenolysis – breakdown of glycogen to glucose
- Ketogenesis – fatty acids or protein breakdown into ketones
- Ammonia Conversion - Ammonia into urea

When there is not enough glucose – Fatty acids are converted into ketones

Important Concepts

- Bile aids in digestion by forming bile salts which help emulsify fats
- Billirubin is a byproduct of hemoglobin breakdown
- Metabolism of drugs by the liver are slowed in older adults which can increase their effects
- Bile is made up of water, electrolytes, lecithin, fatty acids, cholesterol, bilirubin and bile salts

Cirrhosis

Diagnostics

- Radioisotope Liver Scan – uneven uptake of isotopes
- Abdominal Ultrasound – shows ascites
- Laproscopy – can visualize tissue directly
- ERCP – endoscopy to show gallbladder, liver and pancreas
- CT Scan – shows dense fatty areas
- MRI – shows neoplasms, cysts and obstructions
- Liver Biopsy – large needle inserted into liver pt has high risk for hemorrhage

Lab Considerations

- ALP - increased high
- ALT and AST – increased high
- LDH – increased high
- DT./INR – prolonged
- Electrolytes – low k+ and low Na+
- Bilirubin – increased levels high
- Protein – low albumin/globulin
- BUN – low decreased

Liver Disease

Pathophysiology

- Usually a gradual decline in function as liver tissue is slowly destroyed
- Hepatocyte and liver lobule destruction causes decreased metabolic function
- Fibrous connective tissue forms which disrupts the flow of blood and bile – causing portal hypertension

Manifestations

- Jaundice – d/t high bilirubin levels
- Portal HTN – d/t narrowed vessels
- Ascites – d/t portal HTN
- Esophageal varices d/t portal HTN

Possible Complications

Portosystemic encephalopathy

d/t accumulation of neurotoxins

- s/sx: asterixis, in mental status and sleep
- I – normal LOC and some lethargy
- II – high lethargy, disorientation, agitation
- III – stupor, difficulty waxing, incoherent
- IV – comatose, no response to stimuli
- Tx: small freq. meals, high protein intake

Hemorrhage

d/t low clotting factors

- s/sx: tachycardia, hypotension
- Tx: transfusion, fluid replacement, emergency surgery

Etiology

- Malnutrition r/t alcoholism
- Infection
- Diabetes
- Nutritional deficiency
- Hypersensitivity

Manifestations

- Jaundice
- Portal hypertension
- Ascites
- Varices
- Hepatic encephalopathy

Early	Later
- Enlarged liver - Weight loss - Weakness - Anorexia	- Portal vein HTN - Jaundice

Nursing

- encourage patient to avoid alcohol and explain importance
- maintain fluid balacne, weight, i/o, assess urine
- assess loc and mental status
- minimize bleeding
  - bleeding precautions
  - PT/INR
- promote nutrition and protein

Alcoholic

- Metabolic changes in liver
- Women more susceptible

Biliary

- Bile flow is obstructed

Posthepatic

- Caused by hepatitis and other causes

Treatment

- Slow progression of disease
  - Stop drinking alcohol
  - End healthier diet
- Liver transplant
  - C/I with alcoholism or malignancy
- Minimize bleeding
  - Monitor coagulation p/t
  - Institute bleeding precautions
- Paracentesis – aspiration of peritoneal cavity fluid
  - Helps relieve respiratory distress
  - 500-1000mL removed daily
  - Albumin given during large volume

Nursing Interventions for Paracentesis

- Ensure informed consent obtained
- Instruct client to void to prevent puncturing bladder
- Assess weight, abd. girth and vital signs
- Place client in high fowler's/ upright