I. DEFINITION
   A. End stage of chronic liver disease. Progressive, irreversible disorder, eventually leading to liver failure

B. Pathophysiology
   Functional liver tissue is destroyed and replaced by fibrous scar tissue
   Metabolic functions of the liver are lost
   Bile stasis occurs due to constrictive bands
   Blood does not flow freely through the liver to inferior vena cava
   Increased pressure in portal venous system - congested veins result

II. CAUSES
A. Alcoholic cirrhosis – most common cause
   1. Alcohol causes metabolic changes in liver
   2. Fatty infiltration of hepatocytes
   3. Inflammatory cells infiltrate the liver causing necrosis, fibrosis and destruction of functional liver tissue
   4. Liver shrinks and develops a nodular appearance
   5. Malnutrition commonly accompanies alcoholic cirrhosis

B. Billary Cirrhosis
   1. Obstructed bile damages and destroys liver cells
   2. Leads to inflammation, fibrosis and formation of regenerative nodules

C. Posthepatic Cirrhosis
   1. Results from chronic hepatitis B or C or unknown cause
   2. Liver is shrunken and nodular with cell loss and fibrosis

III. SIGNS AND SYMPTOMS
A. Early – few symptoms
   1. Liver is usually enlarged and may be tender
   2. Dull ach in RUQ
   3. Weight loss, weakness and anorexia
   4. Bowel changes with diarrhea or constipation

B. Late – related to liver cell failure and portal hypertension
   1. Malnutrition, muscle wasting
      Impaired nutrient metabolism
      Impaired fat absorption
   2. Bleeding problems/bruising
      Decreased clotting factor synthesis
      Increased platelet destruction by enlarged spleen
      Impaired vitamin K absorption and storage
   3. Ascites/edema
      Impaired plasma protein synthesis
      Impaired pressure in portal venous system
   4. Jaundice
      Impaired bilirubin metabolism and excretion
5. Neurologic changes/encephalopathy
   Accumulated metabolic toxins
   Impaired ammonia metabolism and excretion

MULTISYSTEM EFFECTS OF CIRRHOSIS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
</tr>
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<tbody>
<tr>
<td>Neurologic System</td>
<td>Agitation leading to lethargy, stupor, coma</td>
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<tr>
<td></td>
<td>Asterixis (liver flap) flapping tremor of hands when arms are extended</td>
</tr>
<tr>
<td>Endocrine system</td>
<td>Gynecomastia in males, possible diabetes</td>
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<tr>
<td>Respiratory system</td>
<td>Dyspnea</td>
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<tr>
<td>Cardiovascular system</td>
<td>Bounding pulses, pulmonary hypertension, dysrhythmias</td>
</tr>
<tr>
<td>Hepatic system</td>
<td>Splenomegaly, possible liver cancer</td>
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<tr>
<td>Gastrointestinal System</td>
<td>Abdominal pain, anorexia, Nauseas, Clay-colored stools, peptic ulcers, GI bleeding, hemorrhoids</td>
</tr>
<tr>
<td>Hematologic System</td>
<td>Thrombocytopenia, anemia</td>
</tr>
<tr>
<td>Reproductive System</td>
<td>Oligomenorrhea (female)</td>
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<tr>
<td></td>
<td>Testicular atrophy (male)</td>
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<tr>
<td>Integumentary System</td>
<td>Jaundice, erythema of palms, spider angioma, decreased body hair, pruritis, ecchymoses, caput medusae (dilated veins around the umbilicus)</td>
</tr>
<tr>
<td>Immune System</td>
<td>Leukocytopenia, increased susceptibility to infection</td>
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IV. COMPLICATIONS

A. Portal hypertension
   1. Normal blood flow returning to the heart from the abdominal organs collects in the portal veins and travels through the liver
   2. Pressure increases in the portal vein due to restricted flow
   3. Collateral channels develop between the portal and systemic veins that supply the lower rectum and esophagus and the umbilical veins
   4. Results in hemorrhoids, esophageal varices and caput medusae (dilated veins around the umbilicus)

B. Splenomegaly
   1. Spleen enlarges due to portal hypertension and shunting of blood into splenic vein
   2. Increased destruction of red and white blood cells and platelets
   3. Leads to anemia, leukopenia and thrombocytopenia

C. Ascites
   1. Accumulation of fluid in abdominal cavity
   2. Hypoalbuminemia – low serum albumin levels

D. Esophageal Varices
   1. Enlarged, thin-walled veins in the esophagus due to portal hypertension
   2. May bleed, rupture causing massive hemorrhage

E. Hepatic Encephalopathy
   1. Accumulation of neurotoxins in the blood
   2. Ammonia accumulation – destroys brain cells
F. Hepatorenal Syndrome
1. Renal failure with azotemia
2. Sodium retention, oliguria, hypotension

V. TREATMENT
A. Medications
   1. Avoid known hepatotoxic drugs and alcohol (barbiturates, sedatives, hypnotics, and acetaminophen)
   2. Diuretics – reduce fluid retention and ascites
      Spironolactone – Lasix
   3. Reduce nitrogenous load and lower serum ammonia levels
      Lactulose and neomycin. Reduce the number of ammonia forming organisms in the bowel and ammonium is excreted in the feces
   4. Lower hepatic venous pressure - prevent rebleeding of esophageal varices
      Corgard, Imdur, Monoket
   5. Ferrous sulfate and folic acid – treat anemia
   6. Vitamin K – reduce risk of bleeding
   7. Antacids are prescribed as indicated
   8. Serax a benzodiazepine antianxiety/sedative drug, not metabolized by liver to treat acute agitation

B. Dietary and fluid management
   1. Sodium intake is restricted to under 2 g/day
   2. Fluids are limited to 1500mL/day
   3. Adequate calories 75-100g of protein per day
   4. With encephalopathy is present, 60-80 g/day
   5. Vitamins and mineral supplements. Particularly B-complex
   6. Magnesium deficiency common in alcohol-induced cirrhosis
   7. TPN (total parenteral nutrition) may be initiated through a Central line
      Contains carbohydrates high concentration of dextrose), protein, e-lytes vitamins, minerals and fat emulsion.
      New containers every 24 hours – procedure for checking similar to blood checks.
      Solutions are mixed specifically for patient based on lab value
      Always infused with pump
      Blood glucose levels carefully monitored and insulin may be administered as needed. e-lytes also closely monitored and formula adjusted as needed
      High risk for infection due to disruption of skin barrier and high glucose solution. Monitor closely for S&S of infection

C. Paracentesis
   1. Aspiration of fluid from peritoneal cavity to relieve respiratory distress
   2. Moderate withdrawal 500ml to 1L to reduce risk of fluid and electrolyte imbalances
   3. 4-6L of fluid may be done Albumin intravenously during large volume paracentesis
   4. Nursing care:
      Informed consent
      Weight prior to paracentesis
      Vital signs for baseline
CIRRHOSIS AND PORTAL HYPERTENSION

Have client void immediately prior to test to avoid bladder puncture
Position seated, on side of bed or in chair
Assess site for fluid leakage, change dressing prn

D. Gastric Endoscopic
1. Gastric lavage – saline to improve visualization; decrease bleeding
   Nursing care during lavage
   Baseline assessment – VS, abdominal inspection, girth BS
   Pt teaching – gain cooperation during procedure
   Fowler’s or semi-fowlers position
   Verify placement - test

2. Varices may be sclerosed to reduce risk of recurrent bleeding
3. Banding – small rubber bands are placed on varices to occlude blood flow
4. Balloon tamponade – Sengstaken-blakemore tube and balloons are
   inflated to apply direct pressure on the bleeding varices
   ET tube inserted prior to support airway and reduce aspiration risk
   Short term measure only

E. Transjugular intrahepatic portosystemic shunt (TIPS)
1. Channel created through the liver tissue – shunt inserted to allow blood flow to
   bypass the cirrhotic liver
2. Relieves pressure in esophageal varices
3. Stenosis and occlusion of shunt are frequent complications
4. Increases the risk of developing hepatic encephalopathy
5. Short term measure
F. Surgery - liver transplantation
   Indicated for some clients with irreversible, progressive cirrhosis

G. Central Venous Catheter
   Placement confirmed by X-ray before use.
   Triple lumen most common – administer meds, parenteral solutions, draw labs.
   Review nursing care of Central Venous Catheter care

VI. NURSING DIAGNOSIS

   Excess fluid volume
   Disturbed thought process
   Impaired skin integrity
   Imbalanced nutrition: less than body requirements
   Ineffective health maintenance
   Fatigue

VII HOME CARE
   A. Teaching
   1. Avoid alcohol and other hepatotoxic drugs
   2. Diet and fluid intake restriction and recommendations
   3. Medications – timing, adverse effects
   4. Bleeding precautions
   5. Manifestations of potential complications to be reported
   6. Skin care techniques to reduce pruritus and damage
   7. Ways to manage fatigue and conserve energy
   B. Referals
1. Home health services, etc.
2. Local support groups
3. Hospice if indicated