Care of a Client with Liver Cirrhosis

Understanding cirrhosis, nursing management, and prevention of complications.

Kechi Iheduru-Anderson DNP, RN, CNE, CWCN

Introduction

Nurses are seeing increasing number of client with liver disease. Most of these clients are under 65 years of age. Most common causes of liver disease is avoidable and complications may take up to 30 years to develop. The key to preventing liver disease is to identify those at risk and institute preventive measure. This article describes liver cirrhosis, its complications, and management.

Liver cirrhosis

Liver cirrhosis occurs as a result of any chronic liver disease. Repeated damage to the hepatocytes results in the development of fibrosis and nodular tissue. This alters the liver’s cellular structure, impedes function and affects the blood flow in and around the liver, leading to portal hypertension. Once cirrhosis has developed, it is usually irreversible and can lead to liver failure. Viral hepatitis C has emerged as the leading cause of liver cirrhosis surpassing alcoholic liver disease in the last decade.

Incidence

Liver cirrhosis is an important public health concern in the United States. According to the Centers for Disease Control and Prevention (CDC) chronic liver disease and cirrhosis result in about 35,000 deaths each year in the United States. Cirrhosis is the ninth leading cause of death in the United States and is responsible for 1.2% of all US deaths, with many client dying in the fifth and sixth decade of life. In women, the risk of cirrhosis increases with the ingestion of 20 g of alcohol per day over a span of 10 years or more. In men, a high cirrhosis risk is associated with ingestion of 60 to 80 g/day over the same period. There is increased risks of mortality from cirrhosis among men and women drinking 12 to 24 g of alcohol per day.

Learning objectives

1. Describe the functions of the liver.
2. Describe the pathology of cirrhosis of the liver.
3. Discuss the physiologic changes of liver cirrhosis
4. Discuss management of liver cirrhosis, including importance of monitoring for complications.
5. Describe the appropriate interventions for hepatic encephalopathy and esophageal varices.
Structure and Functions of the Liver

The liver, the body’s largest glandular organ, lies just below the diaphragm in the upper right quadrant of the abdominal cavity. A human liver normally weighs 1.44–1.66 kg (3.2–3.7 lb), and is a soft, pinkish-brown, triangular organ. The Falciform ligament divides the liver into two main lobes, right and left, with the right lobe being larger than the left. The lobes are further divided into lobules, the functional units of the liver. Each lobule consists of a central vein surrounded by 6 hepatic portal veins and 6 hepatic arteries. These blood vessels are connected by many capillary-like tubes called sinusoids, which extend from the portal veins and arteries to meet the central vein. The central veins empty into the right and left hepatic veins which then drain into the vena cava.

Each sinusoid passes through the liver tissue containing 2 main cell types: Kupffer cells and hepatocytes.

Kupffer cells are a type of macrophage that capture and break down old, worn out red blood cells passing through the sinusoids. Hepatocytes are cuboidal epithelial cells that line the...
sinusoids and make up the majority of cells in the liver. Hepatocytes perform most of the liver’s functions – metabolism, storage, digestion, and bile production.

The liver receives its blood supply from the hepatic artery and portal vein. The hepatic artery carries blood from the aorta to the liver, whereas the portal vein carries blood containing digested nutrients from the entire gastrointestinal tract and also from the spleen and pancreas to the liver to process the nutrients and byproducts of food digestion (About 60% of the blood perfusing the liver is from the hepatic portal vein).

The main digestive function of the liver is the production of bile. Bile aids in fat digestion and absorption of fat and fat-soluble vitamins from the small intestine. The salts in bile emulsify fat (break fat into small droplets) so that digestive enzymes can act on fat more effectively (See table 1 for some other functions of the liver.

Liver Cirrhosis.

Liver Cirrhosis also known as hepatic cirrhosis, refers to the diffuse destruction of and progressive replacement of normal liver tissue with fibrous scar tissue. As necrotic tissues yields to fibrosis, the diseases alters the liver structure and normal vasculature, impairs blood and lymph flow, and ultimately causing hepatic insufficiency.

Causes of Liver Cirrhosis

Cirrhosis is caused by chronic (long-term) liver diseases that damage liver tissue. It can take several years for cirrhosis to develop. Any progressive liver disease may lead to cirrhosis. Chronic alcoholism is by far the most common cause of cirrhosis, followed by chronic hepatitis C, nonalcoholic fatty liver disease, and chronic hepatitis B. Other causes of liver cirrhosis include bile duct diseases, chronic biliary obstruction and infection, (such as biliary atresia, primary sclerosing cholangitis, and primary biliary cirrhosis), cystic fibrosis, long-standing severe, right-sided heart failure, autoimmune hepatitis, and inherited metabolic liver diseases (such as hemochromatosis, Wilson’s disease, and alpha-1 antitrypsin deficiency). Also, drug toxicity (Methotrexate) and heart failure may cause cirrhosis.

Pathophysiology of Liver Cirrhosis

The term cirrhosis denotes chronic tissue degeneration in which liver cells are destroyed leading to the formation of fibrous scar tissue. As the cellular destruction continues, blood, lymph and bile channels within the liver become distorted and compressed, leading to intrahepatic congestion, portal hypertension and impaired liver function.
<table>
<thead>
<tr>
<th>Secretions</th>
<th>Functions</th>
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<tbody>
<tr>
<td>Bile (from the breakdown products of hemoglobin).</td>
<td>Fat Emulsification</td>
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Synthesis of these plasma proteins (albumins and the globulins).

<table>
<thead>
<tr>
<th>Albumin</th>
<th>Functions</th>
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<tr>
<td>Transport of several key substances (iron, copper, lipids); Serves as a precursor to fibrin (fibrino- gen); Serves as antibodies or immunoglobins (Gamma globulin, IgG, IgE, IgA, IgD, IgM).</td>
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<thead>
<tr>
<th>Globulins</th>
<th>Functions</th>
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<tr>
<td>Provides osmotic pressure for blood pressure</td>
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<th>Albumin</th>
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<td>Influences osmotic pressure, plasma volume and tissue fluid balance.</td>
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<tr>
<th>Fibrinogen</th>
<th>Functions</th>
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<tr>
<td>Necessary for blood clotting</td>
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<tr>
<th>Prothrombin</th>
<th>Functions</th>
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<tr>
<td>The liver metabolizes the by-products of cellular metabolism and exogenous materials such as drugs. The removal of ammonia which is toxic to the human organism. Ammonia is removed from amino acids via deamination and converted to a normally non-toxic material called urea, which is excreted in urine via the kidney.</td>
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<th>Factor VII</th>
<th>Functions</th>
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<th>Detoxification</th>
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<th>Other Functions of the Liver</th>
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<tr>
<td>Stores glucose (as glycogen). Breaks down glycogen into glucose (i.e. glycogenolysis)</td>
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<td>Produces body heat</td>
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<tr>
<td>Synthesizes vitamin A, Storage of iron, copper, B12, vitamins A, D, E and K</td>
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<tr>
<td>Synthesizes triglycerides and cholesterol</td>
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<td>Metabolism of steroidal hormones</td>
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Table 1: Functions of the liver.

The fibrous changes within the organ cause it to become firmer and smaller. The surface, however, becomes rough and lumpy because of the development of nodules (regenerative nodules) on the surf ace of the organ in an attempt to repair itself. Eventually, cirrhosis progresses throughout the liver resulting in irreversible liver damage and impaired liver function.
Portal hypertension: Normally, blood from the GI tract, spleen, and pancreas flows to the liver via the portal vein, then moves into the vena cava for return to the heart. Increased resistance to portal blood flow and a sustained increase in portal venous pressure characterize portal hypertension. Varices (veins behind the obstruction that dilate) and collateral blood flow channels develop. Complications of portal hypertension include ascites, congestive splenomegaly, portosystemic shunts, and bleeding from varices.

Ascites and peripheral edema develop as a result of several mechanisms. Portal hypertension causes leakage of proteins from the blood vessels into the lymph spaces in the liver tissue. When the lymphatic system is unable to carry off the excess proteins and water, they leak through the liver capsule into the peritoneal cavity. The osmotic pressure of the proteins pulls additional fluid into the peritoneal cavity, creating ascites. Hypoalbuminemia resulting from impaired liver synthesis of albumin also contributes to ascites and peripheral edema by decreasing colloidal osmotic pressure.

Jaundice of the sclera and skin results from functional derangement of liver cells and compression of bile ducts by connective tissue growth that impairs the ability of the liver to conjugate and excrete bilirubin. Hepatomegaly occurs from the fatty infiltration, inflammatory reactions, and scarring of the liver that occurs with cirrhosis, whereas splenomegaly occurs as a result of portal hypertension and congestion of the spleen.

The Four types of cirrhosis
Alcoholic (Laennec’s): Long term ETOH abuse leading to metabolic changes in liver, particularly fat.

- Preceded by a theoretically reversible fatty infiltration of the liver cells,
- widespread scar tissue formation surrounds portal area

Biliary cirrhosis: Associated with chronic biliary obstruction and infection;

- Bile stasis; Inflammation
- scarring around bile ducts and lobes of liver

Post necrotic - Massive hepatic cell necrosis: Complication of toxic or viral hepatitis.

- Post viral hepatitis;
- Toxic exposure;
- Autoimmune process.
- Broad bands of scar tissue form within the liver

Cardiac: Results from longstanding severe right-sided heart failure (Severe RHF).

**Clinical manifestations**

The liver is a vital organ with many functions including: metabolizing carbohydrates, fats and bilirubin; storing glycogen; and cleansing blood. The cirrhotic liver may be able to function adequately – termed a “compensated” liver cirrhosis – but once the functions start to deteriorate and complications of portal hypertension arise, it is “decompensated” and the client has ELD. In the early stages of the disease client is usually asymptomatic. The clinical features of liver failure develop because blood flow in the liver becomes obstructed and the liver loses its normal ability to support digestion, metabolize toxins, and produce proteins for normal clotting function. The symptoms include;

- Loss of appetite
- Tiredness
- Difficulty breathing due to ascites and fluids in the lungs.
- Nausea
- Weight loss
- Abdominal pain
- Dilated abdominal wall veins;
- Spider angiomata (Spider-like blood vessels)
- palmar erythema; peripheral edema
- Severe itching
- Jaundice: Occurs because of insufficient conjugation of bilirubin by the liver cells, and local obstruction of biliary ducts by scarring and regenerating tissue

<table>
<thead>
<tr>
<th>Table 2: Compensated cirrhosis vs. Decompensated cirrhosis</th>
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<tr>
<td><strong>Compensated cirrhosis</strong></td>
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<tr>
<td>Compensated cirrhosis means that the liver is heavily scarred but can still perform many important bodily functions. Many people with compensated cirrhosis experience few or no symptoms and can live for many years without serious complications.</td>
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<tr>
<td>➢ Intermittent mild fever</td>
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<tr>
<td>➢ Vascular spiders</td>
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<tr>
<td>➢ Unexplained epistaxis</td>
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<tr>
<td>➢ Morning indigestion</td>
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<tr>
<td>➢ Flatulent dyspepsia</td>
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<tr>
<td>➢ spider angiomas</td>
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<tr>
<td>➢ Ankle edema</td>
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<tr>
<td>➢ Hepatomegaly</td>
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<tr>
<td>➢ Splenomegaly</td>
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<tr>
<td>➢ Abdominal pain</td>
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Care of a client with liver cirrhosis.

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February 29, 2016

pg. 8
Assessment and Diagnosis

Clients often present with signs and symptoms of cirrhosis or its complications. Liver biopsy remains the diagnostic standard (However sampling error may affect the results). The degree of fibrosis can be estimated by measurement of biomarkers, such as type I and type III collagen, laminin, and hyaluronic acid. Key lab values include complete blood cell count, liver enzymes, renal function tests, electrolytes, and coagulation studies such as PT and international normalized ratio (INR). A liver panel, including serum ALT, AST, GGT, INR, and albumin levels, can be used to evaluate changes in liver function in clients with liver cirrhosis.

Elevation of serum prothrombin time or International Normalized Ratio (INR) may indicate a decreased ability of the liver to synthesize clotting factors. Due to altered synthesis of coagulation proteins, a prolonged PT has been shown to correlate with the degree of liver fibrosis. Thrombocytopenia may indicate splenic sequestration.

Elevated ammonia level. A major source of ammonia is the bacterial and enzymatic deamination of amino acids in the intestines. The ammonia that results from this deamination process normally goes to the liver via the portal circulation and is converted to urea, which is then excreted by the kidneys. When the blood is shunted past the liver via the collateral anastomoses or the liver is unable to convert ammonia to urea, large quantities of ammonia remain in the systemic circulation.

Elevated enzyme levels, including alkaline phosphatase, AST, ALT, and GGT because of the release of these enzymes from damaged liver cells. Elevated total bilirubin. Total (conjugated and unconjugated) bilirubin increases as a result of the liver’s altered ability to take up bilirubin from the blood or to conjugate or excrete it.

CT scan: Determines the size and extent of liver damage; Hepatomegaly

Medical management

It is important to determine the cause of cirrhosis because management of the underlying disease (e.g., hepatitis B virus infection) may slow the progression of the disease and prevent additional liver injury. Cirrhosis due to autoimmune hepatitis treated with steroids, and HBV treated with antiviral therapy. For clients with decompensated cirrhosis, liver transplantation may be the only long-term treatment option. Outcomes after liver transplant have improved significantly, with a five year survival rate of up to 77 percent. Treatment of liver cirrhosis is discussed under the following headings. The treatment of hepatic encephalopathy is discussed separately under complications of liver cirrhosis.
Portal hypertension and esophageal varices

Esophageal varices are a common complication, occurring in 66% to 75% of clients with cirrhosis. These collateral vessels contain little elastic tissue and are quite fragile. They have poor tolerance for the high pressure, and the result is distended, tortuous veins that bleed easily. Large varices are more likely to bleed. In addition, because of compromised liver function, there are alterations in normal blood clotting mechanisms. Current guidelines recommend a screening endoscopy once a client is diagnosed with cirrhosis even without decompensation or previous GI bleeding. Medical prophylaxis with nonselective beta-blockers such as propranolol or nadolol is indicated for small esophageal varices (<5 mm). In cases of large varices with the high-risk criteria, nonselective beta-blockers or endoscopic variceal ligation should be used for prophylaxis. Endoscopic variceal ligation is used in small varices when nonselective beta-blockers are contraindicated. Nonselective beta-blockers such as propranolol and nadolol reduce portal hypertension and are often prescribed for large varices or those at high risk for bleeding. Propranolol may be contraindicated in clients with refractory ascites.

Acute variceal bleeding is an emergency and clients should be admitted with close monitoring in intensive care unit or at least intermediate care level. Immediate treatment of variceal hemorrhage includes protecting the airways to prevent aspiration, providing hemodynamic support, treating coagulopathy, and reducing portal pressure. Intravascular volume support and blood transfusions should be started with a goal to maintain hemoglobin around 8 g/dL. (Alternatively, the goal can be to maintain hematocrit at 24% to 30%.) as more aggressive transfusion can lead to elevation in portal pressure that can worsen variceal bleeding. Significant coagulopathy should be corrected using fresh frozen plasma and/or platelets transfusion. Octreotide (Sandostatin) is used in the management of acute variceal bleeding to reduce portal pressure. Vasopressin (Pitressin) is also effective, but it’s used less often because it’s “extremely potent” and can have serious adverse effects. Once hemostasis has been achieved, definitive treatment by endoscopy can be performed. Monitor client closely from rebleeding.

Octreotide is the drug of choice in the management of acute variceal bleeding. An analogue of the peptide somatostatin, it works by inhibiting the release of vasodilatory hormones such as glucagon, which indirectly causes vasoconstriction of the viscera and decreased portal vein flow. Vasopressin constricts mesenteric arterioles and decreases portal flow, thereby lowering portal pressure.
Pain control

Cirrhotic clients are subjected to higher risk with pain medications compared to general population. Nonsteroidal anti-inflammatory drugs (NSAIDs) should generally be avoided because of the associated GI toxicity (in a client that may be coagulopathic with varices) and also reduction of renal function and diuretic response by inhibiting vasodilating prostaglandins release. Acetaminophen is not contraindicated in clients with liver disease but should be used with caution. The suggested safe daily limit is 2-4 grams in the client with cirrhosis and in case of active alcohol drinking, the daily limit should be 2 grams or even less. Whenever possible, opioids should be avoided in decompensated cirrhotic clients because of the altered elimination (hepatic and renal) and prolonged half-life precipitating encephalopathy through accumulating CNS suppression effect and/or associated constipation. Hypotension is another side effect of narcotics that can exacerbate systemic hypotension seen in cirrhosis.

Opiates should be avoided or used sparingly at low and infrequent doses because of the risk of precipitating hepatic encephalopathy.

Tramadol appears relatively safe as it works through other mechanisms beside opioid receptors and it should be the first line narcotic used. Other narcotics such as oxycodone, morphine and hydromorphone can be cautiously used if pain is not controlled but dose reduction and less frequent administration are recommended. Other ways to control pain include paracentesis for tense ascites and switching diuretics for symptomatic gynecomastia.

As a general rule unnecessary use of medication should be avoided in cirrhosis because of potential hepatotoxic or nephrotoxic effects and because of possible drug interaction.

Spontaneous bacterial peritonitis (SBP).

Spontaneous bacterial peritonitis (SBP) is a common complication of uncontrolled ascites and is diagnosed by ascitic fluid polymorphonuclear cell count greater than 250 cells per mm3 or positive Gram stain/culture. SBP may develop when bacteria from the intestines are translocated to mesenteric lymph nodes. SBP should be suspected if a client with cirrhosis presents with fever, abdominal pain or tenderness, altered mental status, or hypotension. Broad spectrum antibiotic therapy should be started immediately after culture specimens are obtained. Clients who have spontaneous bacterial peritonitis should receive antibiotics within six hours if hospitalized and within 24 hours if ambulatory. Clients who survive an episode of spontaneous bacterial peritonitis should be given prophylactic antibiotics.
**Ascites**

Ascites is a sign of advanced liver failure, or cirrhosis of the liver. Ascites may cause back pain, changes in bowel function, and fatigue. As clients condition worsens lower extremity edema and shortness of breath develops. The basis of treatment includes:

- Avoiding further liver damage
- Low salt (sodium) diet (2 grams of sodium daily).
- Diuretics: Diuretic regimens typically include a combination of spironolactone (Aldactone) and a loop diuretic (Monitor client closely for weight loss while on diuretics). Usually a daily dose of 40 mg of furosemide and 100 mg of spironolactone 100 mg is started, then the dose is titrated to response every 3-5 days to a maximum of 160 mg of furosemide and 400 mg of spironolactone. Worsening of kidney function and electrolyte disturbances is common with diuretics.
  - Diuretics are often temporarily held for: uncontrolled or recurrent encephalopathy, serum sodium less than 120 mmol/L with no response to fluid restriction, or serum creatinine greater than 2.0 mg/dL.
- Paracentesis: The goals of therapy in clients with ascites are to minimize ascitic fluid volume and decrease peripheral edema, without causing intravascular volume depletion. Clients with new-onset ascites should have diagnostic paracentesis performed, consisting of cell count, total protein test, albumin level, and bacterial culture and sensitivity. Serum-ascites albumin concentration is used to calculate the serum-ascites albumin gradient, which aids in the differential diagnosis of portal hypertension (cirrhotic) ascites, heart failure–associated ascites, peritoneal carcinomatosis, or nephrogenic ascites. Clients with tense ascites should have enough fluid removed to relieve the intra-abdominal pressure in order to make the client comfortable and to minimize the chance of a leak of ascitic fluid. Clients requiring diagnostic or therapeutic paracentesis do not need to receive fresh frozen plasma if their INR is less than 2.5 or platelets if their platelet count is greater than 100 × 103 per mm3. Therapeutic paracentesis or transjugular intrahepatic portosystemic shunt procedure should be considered in clients with recurrent ascites that does not respond to diuretic therapy.
- Monitoring: daily weight, clinical signs of encephalopathy or hypovolemia, kidney function and serum electrolytes. Fluid restriction in the absence of severe hyponatremia and frequent albumin infusions are not indicated in treatment of ascites due to liver cirrhosis.

Paracentesis provides a very quick relief of ascites symptoms, but it does not correct the underlying cause so the fluid eventually returns. Clients must follow strict sodium restriction and diuretic therapy in order to slow down the re-accumulation of fluid.
Paracentesis has several complications.

- A large-volume paracentesis (removal of >5 liters of ascitic fluid) can cause severe hypotension (shock) and kidney damage. To decrease the frequency of this complication clients can be pretreated with a colloid solution, such as albumin.
- Persistent Ascitic fluid leak from puncture site.
- Bleeding
- Wound infection
- Bowel perforation and infection; Frequent taps can increase the risk of infection, and cause an electrolyte imbalance (potassium and sodium).

After paracentesis instruct client to report the following:

- A fever higher than 100°F (38°C).
- Severe abdominal pain.
- Increased redness or tenderness in the abdomen.
- Bloody urine.
- Bleeding or a lot of drainage from the tap site.

**Nutrition**

Muscle wasting is a common problem in cirrhosis due to appetite suppression and ascites. Recommended calorie intake for cirrhotic clients is 40 kcal/kg/day (25-30 kcal/kg ideal body weight impaired glucose tolerance) in energy and 1.2-1.5 kcal/kg/day in proteins. 4 to 6 small meals a day with late evening snack rich in protein. Complex instead of simple carbohydrates are recommended. Unnecessary diet restrictions should be avoided. Including sodium restriction in compensated clients without evidence of fluid retention as this can worsen malnutrition by making food less palatable. Similarly free water restriction is not recommended unless serum sodium is markedly low. Unnecessary protein restriction should be avoided as current evidence shows no added benefit for strict protein restriction compared to moderate protein intake.

Life style modifications are shown to improve cirrhosis progression. Alcohol cessation, smoking cessation, and avoiding cannabis use are associated with less fibrosis progression in clients with chronic viral hepatitis.

**Jaundice:**

**Complications list for Liver Cirrhosis:**

- Hepatic encephalopathy
- Ascites
- Esophageal varices
- Gastrointestinal bleeding
- Peritonitis.
- hepatorenal syndrome
- hepatocellular carcinoma
Hepatic encephalopathy (HE)

Hepatic encephalopathy is a complex and potentially reversible neuropsychiatric syndrome seen in clients with liver cirrhosis. Clinical manifestations of encephalopathy are changes in neurologic and mental responsiveness, ranging from lethargy to deep coma. It may be episodic and is a result of the cirrhotic liver’s inability to break down nitrogen-based substances that arise from the bacteria in the gut and cross the blood-brain barrier. Changes may occur suddenly because of an increase in ammonia in response to bleeding varices or gradually as blood ammonia levels slowly increase. In the early stages, manifestations include euphoria, depression, apathy, irritability, memory loss, confusion, yawning, drowsiness, insomnia, agitation, slow and slurred speech, emotional lability, impaired judgment, hiccups, slow and deep respiration, hyperactive reflexes, and a positive Babinski reflex.

Clinical manifestations of impending coma include disorientation as to time, place, or person. A characteristic symptom is asterixis, or flapping tremors (liver flap). This may take several forms, the most common involving the arms and hands. Other signs include hyperventilation, hypothermia, grimacing, and grasping reflexes. HE negatively affects quality of life and can be distressing for clients and their families. It also affects decision-making processes, so mental capacity needs to be assessed regularly. Severity of hepatic encephalopathy should be graded (Table 3) and documented on the clients medical record. Reversible factors such as constipation, noncompliance with medical therapy, infection (i.e., spontaneous bacterial peritonitis), electrolyte imbalances, gastrointestinal bleeding, worsening renal failure, and use of benzodiazepines should be sought and managed. In clients with ascites paracentesis should be performed to rule out peritonitis as a cause of the encephalopathy.

The goal in managing hepatic encephalopathy is to reduce ammonia formation. Several measures to reduce ammonia formation in the gastrointestinal tract are used. Treatment using mild laxative (aperient) such as lactulose, which assist with evacuating bowel contents. In the colon, Lactulose is split into lactic acid and acetic acid, which decreases the pH from 7.0 to 5.0.

The acidic environment discourages bacterial growth. The lactulose traps the ammonia in the gut, and the laxative effect of the drug expels the ammonia from the colon. It is usually given orally but may be given as a retention enema or via NG tube. Non-adherence by clients result from development of diarrhea, the nurse should advise clients to titrate aperient dose so they aim to have at least two to three soft, bulky stools a day without developing diarrhea. Another method is the sterilization of the intestines with antibiotics such as neomycin sulfate and Rifaximin, which are poorly absorbed from the GI tract. Neomycin can be given orally or rectally. This reduces the bacterial flora of the colon.
Bacterial action on protein in the feces results in ammonia production. Rifaximin can decrease colonic levels of ammoniagenic bacteria, with resulting improvement in the symptoms of hepatic encephalopathy. Constipation should be prevented. Control of hepatic encephalopathy also involves treatment of precipitating causes. This involves controlling GI hemorrhage and removing the blood from the GI tract to decrease the protein in the intestine. Electrolyte and acid-base imbalances and infections should also be treated.

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<tr>
<th>Grade/stages</th>
<th>Description of clinical symptoms</th>
<th>Selected nursing intervention and rationale</th>
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| 0            | Subclinical; normal mental status but minimal changes in memory, concentration, intellectual function, coordination | ▪ Maintain a pleasant, quiet environment and approach in a slow, calm manner. Encourage uninterrupted rest periods. (Reduces excessive stimulation and sensory overload, promotes relaxation, and may enhance coping).
▪ Reorient to time, place, and person as needed. (Assists in maintaining reality orientation, reducing confusion and anxiety).
▪ Observe for signs and symptoms of behavioral change and mentation: lethargy, confusion, drowsiness, slurring of speech, and irritability. Arouse client at intervals as indicated. (Ongoing assessment of behavior and mental status is important because of fluctuating nature of impending hepatic coma). |
| 1            | Mild confusion, euphoria or depression, decreased attention, slowing of ability to perform mental tasks, irritability, disorder of sleep pattern (i.e. inverted sleep cycle) | ▪ Evaluate sleep and rest schedule. (Difficulty falling or staying asleep leads to sleep deprivation, resulting in diminished cognition and lethargy).
▪ Consult with significant other about client’s usual behavior and mentation; (this provides baseline for comparison of current status). |
| 2            | Drowsiness, lethargy, gross deficits in ability to perform mental tasks, obvious personality changes, inappropriate behavior, intermittent disorientation (usually with regard to time) | |
| 3            | Somnolent, but arousable state; inability to perform mental tasks; disorientation with regard to time and place; marked confusion; amnesia; occasional fits of rage; speech is present but incomprehensible | |
| 4            | Coma, with or without response to painful stimuli | |

**Hepatorenal syndrome (HRS)**
Hepatorenal syndrome is the development of renal failure in clients with advanced chronic liver disease. Clients with advanced liver cirrhosis are more susceptible to acute kidney injury (AKI) due to the reduced effective circulating blood volume and mean arterial pressure secondary to splanchic vasodilation leading to kidney hypoperfusion. The development of HRS in people with cirrhosis is a sign of poor prognosis. The hallmark of HRS is renal vasoconstriction. Confirm diagnosis, exclude other reversible causes such as overdiuresis or acute GI bleeding;

Rule out infections; Treatment using combination albumin and vasoconstrictor (terlipressin or noradrenaline or both midodrine and octreotide). Definitive treatment for HRS is a liver transplant. In HRS, the histological appearance of the kidneys is normal, and the kidneys often resume normal function following liver transplantation.

Nursing management

1. Imbalanced nutrition: less than body requirements related to nausea manifested by weight loss and low serum protein levels.
2. Disturbed sensory perception or Altered thought processes related to disturbances in central nervous system functioning associated with accumulation of toxic substances (e.g. ammonia) in the brain, toxic effects of long-term alcohol use, deficiencies of certain vitamins (e.g. thiamine), and hypoxia if anemia is moderate to severe.
3. Ineffective breathing pattern related to diminished lung/chest wall expansion associated with: pressure on the diaphragm as a result of ascites;

   - Desired Outcome: The client will have an improved breathing pattern as evidenced by: normal rate and depth of respirations; decreased dyspnea.
   - Interventions:
     - Assess for signs and symptoms of an ineffective breathing pattern (e.g. shallow respirations, dyspnea, tachypnea, use of accessory muscles when breathing, limited chest excursion).
     - Implement measures to improve breathing pattern: place client in a semi-Fowler's position (a high Fowler's position is uncomfortable if ascites is severe)
     - Instruct client to deep breathe or use incentive spirometer every 1-2 hours
4. Impaired gas exchange, related to pressure of ascites fluid on the diaphragm as manifested by tachypnea and decreased oxygen saturation
5. Impaired skin integrity
6. Knowledge deficit

Note: For detailed nursing care plan for a client diagnosed with liver cirrhosis visit http://www1.us.elsevierhealth.com/SIMON/ULrich/Constructor/diagnoses.cfm?disid=17
pected Outcomes
The expected outcomes established in the plan of care are as follows:
- Respiratory rate and O2 saturations will be within normal limits.
- Abdominal girth will decrease by 1 to 2 cm per day; peripheral edema will decrease.
- Will gain 1 lb (0.45 kg) per week without evidence of increased fluid retention. Serum albumin levels will return to normal range.
- Will be alert and oriented; serum ammonia levels are within normal range.
- Will demonstrate no further evidence of active bleeding.
- Will verbalize willingness to join a community support group.

Nursing care
- Nutrition in liver cirrhosis is very essential aspect of nursing care. A thin and malnourished state in liver failure results from the anorexia, dyspepsia, and nausea and vomiting caused by altered metabolism of carbohydrates, fats, and protein by the liver.
- Provide high-calorie, low-salt, and low-protein diet with between meal snacks.
- Daily weight monitoring is an excellent way to objectively monitor response and avoid over diuresis.
- Evaluate mental status. Promptly report changes in status or laboratory values.
- Observe closely for signs of behavioral or personality changes. Report increasing stupor, lethargy, hallucinations or neuromuscular dysfunction. Arouse the client periodically to determine level of consciousness. Watch for asterixis, a sign of developing encephalopathy.
- Watch for signs of anxiety, epigastric fullness, restlessness and weakness.
- Measure abdominal girth every 8 hours, marking level of measurement.
- Institute bleeding precautions.
- Avoid intramuscular injections.
- Observe and document for bleeding gums, ecchymosis, epistaxis, petechiae and degree of sclera, skin jaundice. Remain with the client during the hemorrhagic episodes.
- Inspect stools for amount, color and consistency. Test stools and vomitus for occult blood as ordered.
- Elevate head of bed; assist to chair with legs elevated t.i.d. as tolerated.
- Include significant others in care and teaching; refer to community agencies for discharge follow-up.
- Provide both written and verbal information about the medication and cirrhosis, including measures to prevent complications.
Health promotion and Prevention

Health promotion for cirrhosis depends on the cause of the cirrhosis. Liver disease is a leading cause of death but the risk of developing it can be minimized. Individuals can decrease their risk for acquiring a liver disease by limiting alcohol consumption and avoiding behaviors that increase the risk for hepatitis, such as unprotected sex and intravenous drug use. The most beneficial intervention is early treatment of any underlying liver disease before progression to cirrhosis.

- Adequate nutrition, especially for the alcoholic and other individuals at risk for cirrhosis, is essential to promote liver regeneration.
- Hepatitis must be identified and treated early so that it does not progress to chronic hepatitis.
- Drugs that are potentially toxic to liver cells should also be avoided.
- Biliary disease must be treated so that the stones do not cause obstruction and infection.

In the United States, vaccination against hepatitis B virus is recommended for all children and adolescents younger than 19 years, as well as for adults who are health care workers, who are infected with human immunodeficiency virus or hepatitis C virus, or who participate in high-risk sexual activity or use intravenous drugs.

- The underlying cause (e.g., chronic lung disease) of right-sided heart failure must be treated so that the heart failure does not lead to cirrhosis.
- Screen clients for heavy drinking using the NIH alcohol screening guide [http://pubs.niaaa.nih.gov/publications/Practitioner/pocketguide/pocket_guide.htm](http://pubs.niaaa.nih.gov/publications/Practitioner/pocketguide/pocket_guide.htm) and make referrals and recommendations as appropriate.
- Hepatitis A and B vaccinations.

Client education

- Instruct client about signs of bleeding varices include vomiting of large amounts of fresh blood or clots. Clients who have signs of bleeding varices should go to an emergency room immediately.
- Clients with varices have no symptoms and do not know they have varices. Instruct client to take Beta blockers as prescribed to help reduce blood flow and pressure in varices.
- Clients must be educated about potential side effects of beta blockers and when to call their healthcare provider such as if dizziness and lightheadedness occur after taking these medicines.
- If a client with ascites develop fever or new abdominal pain, the client must go to the emergency room immediately. These could be signs of a life threatening spontaneous bacterial peritonitis.
- Stress the importance of diet compliance
- Eat small frequent balanced meals instead of three large meals.
- Advise the client that rest and good nutrition conserve energy and decrease metabolic demands on the liver.
• Teach client to alternate periods of rest and activity to reduce oxygen demand and prevent fatigue.
• To minimize the risk for bleeding, warn the client against taking nonsteroidal anti-inflammatory drugs, straining to defecate and blowing his nose or sneezing too vigorously. Suggest using an electric razor and a soft toothbrush.
• Clients with any grade of hepatic encephalopathy from 1 to 4 should not drive or operate heavy machinery.

Evaluation

Effective nursing management should result to;

- Maintenance of normal body weight
- Maintenance of skin integrity
- Effective breathing pattern
- No injury
- No signs of infection

End-of-life care in liver disease

For clients with liver disease there are triggers that should prompt consideration of end-of life discussions and planning these include clients with advanced cirrhosis for whom liver transplant is contraindicated, and who have experienced one or more of the following complications in the last year:

- Diuretic-resistant ascites;
- Hepatic encephalopathy;
- Hepatorenal syndrome;
- Spontaneous bacterial peritonitis;
- Recurrent variceal bleeds.

A comprehensive assessment is needed for client to ensure they have the right support when discharged from hospital.

Learn more about it/ References


NCLEX style questions for student nurses.

1. Which of the following prescribed treatments for ascites in patients with liver cirrhosis will the nurse question?
   a. Fluid restriction
   b. Spironolactone
   c. Sodium restriction
   d. Furosemide

2. A client with end stage liver disease due to cirrhosis stated to the nurse “I don’t understand why my belly is so big, I have not been eating much.” The nurse will include the following as the causes of ascites formation in patients with end-stage liver disease except:
   a. Hypoalbuminemia
   b. Portal hypertension
   c. Increased hepatic clearance of aldosterone
   d. Splanchnic vasodilation

3. Which of the following is not an appropriate treatment for hepatic encephalopathy?
   a. Valium
   b. Lactulose
   c. Neomycin
   d. Rifaximin

4. A client is brought to the emergency department for experiencing acute esophageal variceal hemorrhage. Which of these is a priority nursing action for this client?
   a. Close observation and maintenance of their away
   b. Saline resuscitation to maintain systolic blood pressure >140 mmHg
   c. Transfusion of packed red blood cells to maintain Hgb >12
   d. Sedation with morphine to decrease portal pressure

5. A client presenting with ascites due to liver cirrhosis is being evaluated for fluid balance. Which of the following provides the best indicator of fluid status?
   a. Accurate intake and output measurement
   b. Daily liver function tests
   c. Caloric intake and serum protein levels
   d. Measurement of daily weight
6. While providing discharge teaching to the client with cirrhosis, his wife asks the nurse why there is so much emphasis on bleeding precautions. Which of the following is the most appropriate response by the nurse?

   a. “The low protein diet will result in reduced clotting factors.”
   b. “The increased production of bile decreases clotting factors.”
   c. “The required medications reduce clotting factors.”
   d. “The liver affected by cirrhosis is unable to produce clotting factors.”

7. When explaining the rationale for the use of lactulose to a client with chronic cirrhosis, the nurse would choose which of the following statements?

   a. “Lactulose reduces constipation, which is a frequent complaint with cirrhosis.”
   b. “Lactulose suppresses the metabolism of ammonia and aids in its elimination through feces.”
   c. “Lactulose helps to reverse cirrhosis of the liver by regenerating new liver cells.”
   d. “Lactulose can be taken intermittently to reduce side effects.”

8. The patient has just had a liver biopsy. Which of the following nursing actions would be the priority after the biopsy?

   a. Monitor pulse and BP every 30 minutes until stable and then hourly for up to 24 hours.
   b. Ambulate every 4 hours for the first day, as long as the patient can tolerate it.
   c. Measure urine specific gravity every 8 hours for the next 48 hours.
   d. Maintain NPO status for 24 hours post-biopsy.

9. A male client is being treated for ruptured esophageal varices with a Sengstaken-Blakemore tube. His vital signs have been stable, and the suction port is draining scant amounts of drainage. He suddenly becomes acutely dyspneic, and oximetry reveals an O2 sat of 74%. The nurse’s immediate action is to

   a. release the esophageal balloon
   b. increase the suction
   c. Lavage the tube with ice water.
   d. release the gastric balloon
   e. irrigate the gastric balloon
10. A newly admitted client with cirrhosis of the liver has a distended abdomen and the umbilicus is protruding. The nurse knows the pathological basis for this is:

a. Increased fluid intake resulting from excessive use of alcohol causing overhydration.
b. Increased size of the liver resulting in abdominal distention.
c. Hypoalbuminemia causing fluid to leave the vascular system and enter the peritoneal cavity.
d. Shunting of the blood to the collateral circulation in the esophagus resulting in decreased blood volume and accumulation of fluid.

11. Which of the following food choices by a client with decompensated liver cirrhosis indicates accurate understanding of nutritional education?

a. Fried fish and potato fries for dinner.
b. Corned beef sandwich with feta cheese for lunch.
c. Frozen yogurt and steamed rice with chicken for dinner.
d. Deli meat sandwich and chicken broth for lunch.

12. Which of these measures should the nurse include in the plan of care for a client admitted with hepatic encephalopathy? Select all that apply.

a. Monitor the patient’s blood glucose as ordered
b. Monitor the patient’s protime (PT) as ordered
c. Institute droplet precautions
d. Assess deep tendon reflexes
e. Provide high-protein feedings

13. A patient diagnosed with chronic hepatitis is prescribed lactulose (Enulose). Which of these laboratory data would indicate that the medication is achieving the desired therapeutic effect?

a. Increased serum albumin
b. Decreased serum ammonia
c. Decreased alanine aminotransferase (ALT)
d. Decreased serum bilirubin
14. Which of these medications ordered for a client with liver cirrhosis should the nurse question? (Select all that apply).

   a. Ibuprofen 600mg po twice daily for pain.
   b. Acetaminophen 650mg po 3x daily for pain.
   c. Acetamenophen with codeine).
   d. Diazepam (Valium) 5mg po QHS for sleep.
   e. Tramadol 25mg po every 8 hours for severe pain

15. Which of these findings in a client admitted with hepatic encephalopathy requires immediate follow-up by the nurse?

   a. Abdominal pain, hypotension, and fever.
   b. Disorientation, indigestion, and euphoria.
   c. Hypoalbuminemia, pruritus, and Splenomegaly.
   d. Spider angiomata and palmar erythema.

16. A client with liver cirrhosis and ascites reported persistent back pain of 7/10 not relieved by paracentesis and repositioning. Which of these medications will the nurse expect to be prescribed by the physician?

   a. Morphine sulfate 2mg IV
   b. Tramadol 25mg po every 8 hours
   c. Neurontin 300mg po daily
   d. OxyContin 15mg po twice daily.
   e. Hydromorphone 1 mg orally every 4 hours.

17. After administering diuretics to client admitted with ascites which nursing action would be most effective in ensuring safe care?

   a. Measuring serum potassium for hyperkalemia
   b. Assessing the client for hypervolemia
   c. Measuring the client’s weight weekly
   d. Documenting precise intake and output.

18. A client diagnosed with chronic cirrhosis who has ascites and pitting peripheral edema also has hepatic encephalopathy. Which of the following nursing interventions are appropriate to prevent skin breakdown? (Select all that apply.)
a. Range of motion every 4 hours  
b. Turn and reposition every 2 hours  
c. Abdominal and foot massages every 2 hours  
d. Alternating air pressure mattress  
e. Sit in chair for 30 minutes each shift

19. The student nurse who is providing care for a client who has jaundice whose plan of care include keeping the client’s fingernails short and smooth. Which statement indicates that the student nurse understands the rationale for this plan of care for the client?

a. “Jaundice is associated with pressure ulcer formation.”  
b. “Jaundice impairs urea production, which produces pruritus.”  
c. “Jaundice produces pruritus due to impaired bile acid excretion.”  
d. “Jaundice leads to decreased tissue perfusion and subsequent breakdown.”

20. Which interventions should the nurse implement when addressing hepatic encephalopathy in a client who has liver cirrhosis? (Select all that apply.)

a. Assessing the client’s neurologic status every 2 hours  
b. Monitoring the client’s hemoglobin and hematocrit levels  
c. Evaluating the client’s serum ammonia level  
d. Monitoring the client’s handwriting daily  
e. Preparing to insert an esophageal tamponade tube  
f. Making sure the client’s fingernails are short

21. A client diagnosed with chronic cirrhosis who has ascites and pitting peripheral edema also has hepatic encephalopathy. Which of the following nursing interventions are appropriate to prevent skin breakdown? (Select all that apply.)

a. Range of motion every 4 hours  
b. Turn and reposition every 2 hours  
c. Abdominal and foot massages every 2 hours  
d. Alternating air pressure mattress  
e. Sit in chair for 30 minutes each shift

22. *Spironolactone (Aldactone)* is prescribed for a client with chronic cirrhosis and ascites. The nurse should monitor the client for which of the following medication-related side effects?
a. Hyperkalemia
b. Tachycardia
c. Hypocalcemia
d. Jaundice

23. A client with liver cirrhosis is placed 2-gram sodium restricted diet. The nurse interprets which client behavior as being compliant with the dietary restrictions?

a. Using only two packets of salt found on the meal tray.
b. Limiting milk to 1 cup per day.
c. Using salt free butter with meals.
d. Avoiding the use of salt in cooking.

24. The nurse is caring for a client who hemorrhaging from esophageal varices due to liver cirrhosis. Which collaborative intervention should the nurse implement? (Select all that apply).

a. Turned to one side and the head of the bed raised to a high Fowler position.
b. Obtain a type and crossmatch for 2 units of packed red blood cells.
c. Assess the client’s vital signs.
d. Administer Octreotide or Vasopressin (Pitressin) intravenously.
e. Prepare to insert a Sengstaken–Blakemore tube.

25. A client admitted with liver cirrhosis is being discharge home. The nurse is educating the client about minimizing his risk of further injury. What will the nurse include to decrease his risk of hemorrhage from esophageal varices?

a. Avoid irritating fluids such as colas and alcohol
b. Avoid lifting heavy objects to prevent muscular exertion.
c. Avoid vigorous physical exercise
d. Chew food carefully and into small pieces.
e. Do not strain to have bowel movements.
f. Monitor your blood pressure and pulse daily.

26. A client admitted with severe liver cirrhosis with grade 1 encephalopathy has a dietary order for low-protein diet. What is the rationale for this order? A low protein diet is recommended for the client with encephalopathy to
a. Minimize the breakdown of food protein and portal hypertension which causes encephalopathy.

b. Minimize the breakdown of dietary protein and subsequent conversion to ammonia.

c. Low protein will help decrease hypertension from ammonia deposits.

d. Minimize the breakdown of dietary protein sodium retention and fluid accumulation.
Answers to NCLEX style questions.

1. A
2. C
3. (A). Ascites is caused by increased production of aldosterone which causes sodium and water retention which worsens ascites. Extracellular fluid leakage into the abdominal cavity.
4. A
5. Rationale D: Daily weights are the single most important indicator of fluid status. Intake and output measurements are an important nursing intervention for monitoring fluid status; however, daily weights are the best indicator. Caloric intake and serum protein levels measure nutritional intake.
6. D
7. B
8. (A). Care after liver biopsy include; apply direct pressure to the site immediately after the needle is removed; position client on the right side; Frequent assessment of vital signs, observe and report any signs of hemorrhage immediately; NPO for 2 hours, Avoid coughing, straining or heavy lifting for 1 to 2 weeks
9. (A). The balloons can migrate superiorly and obstruct the airway. When the client has a Sengstaken-Blakemore tube, a pair of scissors must be kept at the client's bedside at all times. The client needs to be observed for sudden respiratory distress, which occurs if the gastric balloon ruptures and the entire tube moves upward. If this occurs, the nurse immediately cuts all balloon lumens and removes the tube.
10. C
11. C
12. (A, B, D). The liver normally converts the byproducts of protein metabolism (e.g. ammonia) into a non-toxic, water soluble form (urea) to be excreted by the kidneys. Interventions for this patient include blood glucose monitoring (because of decreased glycogen synthesis and storage), monitoring PT and INR (because of decreased clotting factors), checking reflexes (because of the neurological effects of increased ammonia), providing diet/feedings that are low in protein (to decrease ammonia levels), and following standard precautions.
13. (B). Clients with decompensated liver cirrhosis have ascites and should avoid food that are high in sodium.
14. (A, C, D). Nonsteroidal anti-inflammatory agents are contraindicated as they can induce GI bleed and renal failure. Acetaminophen is not contraindicated in liver patients but should be used with caution. The suggested safe daily limit is 2-4 grams in the patient with cirrhosis and in case of active alcohol drinking, the daily limit should be 2 grams or
Care of a client with liver cirrhosis

even less, because of glutathione depletion. Sedatives and opioids are common precipitants of hepatic encephalopathy and hospitalization in patient with liver cirrhosis.

15. (A). A is correct. The signs listed are indicative of spontaneous bacterial peritonitis and must be reported immediately to the physician. The other signs are expected in a client with liver cirrhosis admitted with hepatic encephalopathy.

16. (E). Nonsteroidal anti-inflammatory agents are contraindicated as they can induce GI bleed and renal failure. Opioid analgesic should be used with caution as it can precipitate encephalopathy. Acetaminophen at a dose less than 2 gm/day is a reasonably safe option. In case of inadequate pain relief with acetamnenophen, tramadol 25 mg every 8 hours can be used. For intractable pain hydromorphone orally or fentanyl topical patch can be used. Combination of these drugs with tramadol should not be done.

17. (D). For the client with ascites receiving diuretic therapy, careful intake and output measurement is essential for safe diuretic therapy. Diuretics lead to fluid losses, which if not monitored closely and documented, could place the client at risk for serious fluid and electrolyte imbalances. Hypokalemia, not hyperkalemia, commonly occurs with diuretic therapy. Because urine output increases, a client should be assessed for hypovolemia, not hypervolemia. Weights are also an accurate indicator of fluid balance. However, for this client, weights should be obtained daily, not weekly.

18. (B, D).

19. (C). The client with pruritus experiences itching, which may lead to skin breakdown and possibly infection from scratching. Keeping his fingernails short and smooth helps prevent skin breakdown and infection from scratching. Jaundice is a symptom characterized by increased bilirubin concentration in the blood. Bile acid excretion is impaired, increasing the bile acids in the skin and causing pruritus. Jaundice is not associated with pressure ulcer formation. However, edema and hypoalbuminemia are. Jaundice itself does not impair urea production or lead to decreased tissue perfusion.

20. (A, C, D.) Hepatic encephalopathy results from an increased ammonia level due to the liver’s inability to covert ammonia to urea, which leads to neurologic dysfunction and possible brain damage. The nurse should monitor the client’s neurologic status, serum ammonia level, and handwriting. Monitoring the client’s hemoglobin and hematocrit levels and insertion of an esophageal tamponade tube address esophageal bleeding. Keeping fingernails short address jaundice.

21. (B, D.) Edematous tissue must receive meticulous care to prevent tissue breakdown. Range of motion exercises preserve joint function but do not prevent skin breakdown. Abdominal or foot massage will not prevent skin breakdown but must be cleansed carefully to prevent breaks in skin integrity. The feet should be kept at the level of heart or higher so Fowler’s position should be employed. An air pressure mattress, careful repositioning can prevent skin breakdown.

22. (A.) Spironolactone is a potassium-sparing diuretic so clients should be monitored closely for hyperkalemia.
23. (D.) 2-gram sodium diet requires use of no salt in cooking, no added salt at the table, avoiding high sodium foods, and limiting milk to 2 cups per day. I cup of milk per day and use of salt-free butter are requirements of a 1-gram sodium restricted diet.

24. B, D, And E requires orders from the physician (HCP), and B also requires obtaining laboratory data and are therefore collaborative intervention. A and C are independent nursing interventions and do not require HCP’s orders.

25. Rationale: All responses should be included in the discharge instruction. Others include. Consult your HCP before taking salicylates, which can erode the esophagus. Teach client and care givers how to assess V/S and monitor for S/S of bleeding such as blood pressure below 80/60, bloody or black, tarry stools (melena), cool clammy skin, decreased level of consciousness, hematemesis, tell client to avoid sneezing, coughing or vomiting which increases the risk of rupturing esophageal varices.

26. Rationale: (B). A low protein diet is recommended for the client with encephalopathy to minimize the breakdown of dietary protein and subsequent conversion to ammonia which is then absorbed by the GI tract into the blood stream.

**Tags:** ammonia encephalopathy, liver Cirrhosis, hepatic cirrhosis, hepatic Encephalopathy, Encephalopathy care plan, encephalopathy, hepatic encephalopathy nursing care plan, hepatic encephalopathy pathophysiology, esophageal varices, NCLEX review, NCLEX review questions, GI NCLEX review questions, liver cirrhosis NCLEX review.