

CIRRHOSIS OF THE LIVER

Cirrhosis is a chronic disease of the liver characterized by alteration in structure, degenerative changes and widespread destruction of hepatic cells, impairing cellular function and impeding blood flow through the liver. Causes include malnutrition, inflammation (bacterial or viral), and poisons (e.g., alcohol, carbon tetrachloride, acetaminophen). Cirrhosis is the fourth leading cause of death in the United States among people ages 35 to 55 and represents a serious threat to long-term health.

CARE SETTING

May be hospitalized on a medical unit during initial or recurrent acute episodes with potentially life-threatening complications. Otherwise, this condition is handled at the community level.

RELATED CONCERNS

Alcohol: acute withdrawal
Substance dependence/abuse rehabilitation
Fluid and electrolyte imbalances
Psychosocial aspects of care
Renal dialysis
Renal failure: acute
Total nutritional support: parenteral/enteral feeding
Upper gastrointestinal/esophageal bleeding

Patient Assessment Database

Data depend on underlying cause of the condition.

ACTIVITY/REST

May report: Weakness, fatigue, exhaustion
May exhibit: Lethargy
Decreased muscle mass/tone

CIRCULATION

May report: History of/recent onset of HF, pericarditis, rheumatic heart disease, or cancer (causing liver impairment leading to failure)
Easy bruising, nosebleeds, bleeding gums
May exhibit: Hypertension or hypotension (fluid shifts)
Dysrhythmias, extra heart sounds (S₃, S₄)
JVD; distended abdominal veins
Jaundiced skin, sclera

ELIMINATION

May report: Flatulence
Diarrhea or constipation; gradual abdominal enlargement
May exhibit: Abdominal distention (hepatomegaly, splenomegaly, ascites)
Decreased/absent bowel sounds
Clay-colored stools, melena
Dark, concentrated urine; oliguria (renal failure)

FOOD/FLUID

May report: Anorexia, food intolerance/ingestion
Nausea/vomiting
May exhibit: Weight loss or gain (fluid)
Tissue wasting
Edema generalized in tissues

Dry skin, poor turgor
Halitosis/fetor hepaticus, bleeding gums

NEUROSENSORY

May report: SO(s) may report personality changes, depressed mentation
May exhibit: Changes in mentation, confusion, hallucinations, coma
Slowed/slurred speech
Asterixis (involuntary jerking movements of hands/tongue/feet associated with hepatic encephalopathy)

PAIN/DISCOMFORT

May report: Abdominal tenderness/RUQ pain
Itching
Pins/needles sensation, burning pain in extremities
May exhibit: Guarding/distraction behaviors
Self-focus

RESPIRATION

May report: Dyspnea
May exhibit: Tachypnea, shallow respiration, adventitious breath sounds
Limited thoracic expansion (ascites)
Hypoxia

SAFETY

May report: Severe itching of the skin/dryness
May exhibit: Fever (more common in alcoholic cirrhosis)
Jaundice, ecchymosis, petechiae
Spider angiomas/telangiectasis, palmar erythema

SEXUALITY

May report: Menstrual disorders (women), impotence (men)
May exhibit: Testicular atrophy, gynecomastia, loss of hair (chest, underarm, pubic)

TEACHING/LEARNING

May report: History of long-term alcohol use/abuse, alcoholic liver disease
History of biliary disease, hepatitis, exposure to toxins; liver trauma; upper GI bleeding;
episodes of bleeding esophageal varices; use of drugs affecting liver function
Discharge plan considerations: **DRG projected mean length of inpatient stay: 6.4 days**
May need assistance with homemaker/management tasks
Refer to section at end of plan for postdischarge considerations.

DIAGNOSTIC STUDIES

Liver scans/biopsy: Detects fatty infiltrates, fibrosis, destruction of hepatic tissues, tumors (primary or metastatic), associated ascites.

Percutaneous transhepatic cholangiography (PTHC): May be done to rule out/differentiate causes of jaundice or to perform liver biopsy.

Esophagogastroduodenoscopy (EGD): May demonstrate presence of esophageal varices, stomach irritation or ulceration, duodenal ulceration or bleeding.

Percutaneous transhepatic portal angiography (PTPA): Visualizes portal venous system circulation.

Serum bilirubin: Elevated because of cellular disruption, inability of liver to conjugate, or biliary obstruction.

Liver enzymes:

AST/ALT, LDH, and isoenzymes (LDH₃): Increased because of cellular damage and release of enzymes.

Alkaline phosphatase (ALP) and isoenzyme (LAP1): Elevated because of reduced excretion.

Gamma glutamyl transpeptidase (GTT): Elevated.

Serum albumin: Decreased because of depressed synthesis.

Globulins (IgA and IgG): Increased synthesis.

CBC: Hb/Hct and RBCs may be decreased because of bleeding. RBC destruction and anemia is seen with hypersplenism and iron deficiency. Leukopenia may be present as a result of hypersplenism.

PT/activated partial thromboplastin time (aPTT): Prolonged (decreased synthesis of prothrombin)

Fibrinogen: Decreased.

BUN: Elevation indicates breakdown of blood/protein.

Serum ammonia: Elevated because of inability to convert ammonia to urea.

Serum glucose: Hypoglycemia suggests impaired glycogenesis.

Electrolytes: Hypokalemia may reflect increased aldosterone, although various imbalances may occur. Hypocalcemia may occur because of impaired absorption of vitamin D.

Nutrient studies: Deficiency of vitamins A, B₁₂, C, K; folic acid, and iron may be noted.

Urine urobilinogen: May/may not be present. Serves as guide for differentiating liver disease, hemolytic disease, and biliary obstruction.

Fecal urobilinogen: Decreased.

NURSING PRIORITIES

1. Maintain adequate nutrition.
2. Prevent complications.
3. Enhance self-concept, acceptance of situation.
4. Provide information about disease process/prognosis, potential complications, and treatment needs.

DISCHARGE GOALS

1. Nutritional intake adequate for individual needs.
2. Complications prevented/minimized.
3. Dealing effectively with current reality.
4. Disease process, prognosis, potential complications, and therapeutic regimen understood.
5. Plan in place to meet needs after discharge.

NURSING DIAGNOSIS: Nutrition: imbalanced, less than body requirements

May be related to

Inadequate diet; inability to process/digest nutrients
Anorexia, nausea/vomiting, indigestion, early satiety (ascites)
Abnormal bowel function

Possibly evidenced by

Weight loss
Changes in bowel sounds and function
Poor muscle tone/wasting
Imbalances in nutritional studies

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Nutritional Status (NOC)

Demonstrate progressive weight gain toward goal with patient-appropriate normalization of laboratory values.
Experience no further signs of malnutrition.

ACTIONS/INTERVENTIONS

Nutrition Therapy (NIC)

Independent

Measure dietary intake by calorie count.

RATIONALE

Provides information about intake, needs/deficiencies.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Nutrition Therapy (NIC)</p>	
<p>Independent</p>	
<p>Weigh as indicated. Compare changes in fluid status, recent weight history, skinfold measurements.</p>	<p>It may be difficult to use weight as a direct indicator of nutritional status in view of edema/ascites. Skinfold measurements are useful in assessing changes in muscle mass and subcutaneous fat reserves.</p>
<p>Assist/encourage patient to eat; explain reasons for the types of diet. Feed patient if tiring easily, or have SO assist patient. Consider preferences in food choices.</p>	<p>Improved nutrition/diet is vital to recovery. Patient may eat better if family is involved and preferred foods are included as much as possible.</p>
<p>Encourage patient to eat all meals/supplementary feedings.</p>	<p>Patient may pick at food or eat only a few bites because of loss of interest in food or because of nausea, generalized weakness, malaise.</p>
<p>Recommend/provide small, frequent meals.</p>	<p>Poor tolerance to larger meals may be due to increased intra-abdominal pressure/ascites.</p>
<p>Provide salt substitutes, if allowed; avoid those containing ammonium.</p>	<p>Salt substitutes enhance the flavor of food and aid in increasing appetite; ammonia potentiates risk of encephalopathy.</p>
<p>Restrict intake of caffeine, gas-producing or spicy and excessively hot or cold foods.</p>	<p>Aids in reducing gastric irritation/diarrhea and abdominal discomfort that may impair oral intake/digestion.</p>
<p>Suggest soft foods, avoiding roughage if indicated.</p>	<p>Hemorrhage from esophageal varices may occur in advanced cirrhosis.</p>
<p>Encourage frequent mouth care, especially before meals.</p>	<p>Patient is prone to sore and/or bleeding gums and bad taste in mouth, which contributes to anorexia.</p>
<p>Promote undisturbed rest periods, especially before meals.</p>	<p>Conserving energy reduces metabolic demands on the liver and promotes cellular regeneration.</p>
<p>Recommend cessation of smoking.</p>	<p>Reduces excessive gastric stimulation and risk of irritation/bleeding.</p>
<p>Collaborative</p>	
<p>Monitor laboratory studies, e.g., serum glucose, prealbumin/albumin, total protein, ammonia.</p>	<p>Glucose may be decreased because of impaired glycogenesis, depleted glycogen stores, or inadequate intake. Protein may be low because of impaired metabolism, decreased hepatic synthesis, or loss into peritoneal cavity (ascites). Elevation of ammonia level may require restriction of protein intake to prevent serious complications.</p>
<p>Maintain NPO status when indicated.</p>	<p>Initially, GI rest may be required in acutely ill patients to reduce demands on the liver and production of ammonia/urea in the GI tract.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Nutrition Therapy (NIC)</p> <p>Collaborative</p> <p>Consult with dietitian to provide diet that is high in calories and simple carbohydrates, low in fat, and moderate to high in protein; limit sodium and fluid as necessary. Provide liquid supplements as indicated.</p> <p>Provide tube feedings, TPN, lipids if indicated.</p> <p>Administer medications as indicated, e.g.:</p> <ul style="list-style-type: none"> Vitamin supplements, thiamin, iron, folic acid; Zinc; Digestive enzymes, e.g., pancrelipase (Viokase); Antiemetics, e.g., trimethobenzamide (Tigan). 	<p>High-calorie foods are desired inasmuch as patient intake is usually limited. Carbohydrates supply readily available energy. Fats are poorly absorbed because of liver dysfunction and may contribute to abdominal discomfort. Proteins are needed to improve serum protein levels to reduce edema and to promote liver cell regeneration. <i>Note:</i> Protein and foods high in ammonia (e.g., gelatin) are restricted if ammonia level is elevated or if patient has clinical signs of hepatic encephalopathy. In addition, these individuals may tolerate vegetable protein better than meat protein.</p> <p>May be required to supplement diet or to provide nutrients when patient is too nauseated or anorexic to eat or when esophageal varices interfere with oral intake.</p> <p>Patient may be vitamin-deficient because of previous poor diet. Also, the injured liver is unable to store vitamins A, B complex, D, and K. Anemia due to iron and folic acid deficiencies may also exist.</p> <p>Enhances sense of taste/smell, which may stimulate appetite.</p> <p>Promotes digestion of fats and may reduce steatorrhea/diarrhea.</p> <p>Used with caution to reduce nausea/vomiting and increase oral intake.</p>

<p>NURSING DIAGNOSIS: Fluid Volume excess</p> <p>May be related to</p> <p>Compromised regulatory mechanism (e.g., syndrome of inappropriate antidiuretic hormone [SIADH], decreased plasma proteins, malnutrition)</p> <p>Excess sodium/fluid intake</p> <p>Possibly evidenced by</p> <p>Edema, anasarca, weight gain</p> <p>Intake greater than output, oliguria, changes in urine specific gravity</p> <p>Dyspnea, adventitious breath sounds, pleural effusion</p> <p>BP changes, altered CVP</p> <p>JVD, positive hepatojugular reflex</p> <p>Altered electrolyte levels</p> <p>Change in mental status</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Fluid Balance (NOC)</p> <p>Demonstrate stabilized fluid volume, with balanced I&O, stable weight, vital signs within patient's normal range, and absence of edema.</p>
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ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid/Electrolyte Management (NIC)</p> <p>Independent</p> <p>Measure I&O, noting positive balance (intake in excess of output). Weigh daily, and note gain more than 0.5 kg/day.</p> <p>Monitor BP (and CVP if available). Note JVD/abdominal vein distension.</p> <p>Assess respiratory status, noting increased respiratory rate, dyspnea.</p> <p>Auscultate lungs, noting diminished/absent breath sounds and developing adventitious sounds (e.g., crackles).</p> <p>Monitor for cardiac dysrhythmias. Auscultate heart sounds, noting development of S₃/S₄ gallop rhythm.</p> <p>Assess degree of peripheral/dependent edema.</p> <p>Measure abdominal girth.</p> <p>Encourage bedrest when ascites is present.</p> <p>Provide frequent mouth care; occasional ice chips (if NPO).</p>	<p>Reflects circulating volume status, developing/resolution of fluid shifts, and response to therapy. Positive balance/weight gain often reflects continuing fluid retention. <i>Note:</i> Decreased circulating volume (fluid shifts) may directly affect renal function/urine output, resulting in hepatorenal syndrome.</p> <p>BP elevations are usually associated with fluid volume excess but may not occur because of fluid shifts out of the vascular space. Distension of external jugular and abdominal veins is associated with vascular congestion.</p> <p>Indicative of pulmonary congestion/edema.</p> <p>Increasing pulmonary congestion may result in consolidation, impaired gas exchange, and complications, e.g., pulmonary edema.</p> <p>May be caused by HF, decreased coronary arterial perfusion, and electrolyte imbalance.</p> <p>Fluids shift into tissues as a result of sodium and water retention, decreased albumin, and increased antidiuretic hormone (ADH).</p> <p>Reflects accumulation of fluid (ascites) resulting from loss of plasma proteins/fluid into peritoneal space. <i>Note:</i> Excessive fluid accumulation can reduce circulating volume, creating a deficit (signs of dehydration).</p> <p>May promote recumbency-induced diuresis.</p> <p>Decreases sensation of thirst.</p>
<p>Collaborative</p> <p>Monitor serum albumin and electrolytes (particularly potassium and sodium).</p> <p>Monitor serial chest x-rays.</p>	<p>Decreased serum albumin affects plasma colloid osmotic pressure, resulting in edema formation. Reduced renal blood flow accompanied by elevated ADH and aldosterone levels and the use of diuretics (to reduce total body water) may cause various electrolyte shifts/imbbalances.</p> <p>Vascular congestion, pulmonary edema, and pleural effusions frequently occur.</p>

ACTIONS/INTERVENTIONS	RATIONALE
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<p>Fluid/Electrolyte Management (NIC)</p> <p>Collaborative</p> <p>Restrict sodium and fluids as indicated.</p> <p>Administer salt-free albumin/plasma expanders as indicated.</p> <p>Administer medications as indicated: Diuretics, e.g., spironolactone (Aldactone), furosemide (Lasix);</p> <p>Potassium;</p> <p>Positive inotropic drugs and arterial vasodilators.</p>	<p>Sodium may be restricted to minimize fluid retention in extravascular spaces. Fluid restriction may be necessary to correct/prevent dilutional hyponatremia.</p> <p>Albumin may be used to increase the colloid osmotic pressure in the vascular compartment (pulling fluid into vascular space), thereby increasing effective circulating volume and decreasing formation of ascites.</p> <p>Used with caution to control edema and ascites, block effect of aldosterone, and increase water excretion while sparing potassium when conservative therapy with bedrest and sodium restriction does not alleviate problem.</p> <p>Serum and cellular potassium are usually depleted because of liver disease and urinary losses.</p> <p>Given to increase cardiac output/improve renal blood flow and function, thereby reducing excess fluid.</p>
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<p>NURSING DIAGNOSIS: Skin Integrity, risk for impaired</p> <p>Risk factors may include</p> <p>Altered circulation/metabolic state Accumulation of bile salts in skin Poor skin turgor, skeletal prominence, presence of edema, ascites</p> <p>Possibly evidenced by</p> <p>[Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Risk Control (NOC)</p> <p>Maintain skin integrity. Identify individual risk factors and demonstrate behaviors/techniques to prevent skin breakdown.</p>

<p>ACTIONS/INTERVENTIONS</p> <p>Skin Surveillance (NIC)</p> <p>Independent</p> <p>Inspect skin surfaces/pressure points routinely. Gently massage bony prominences or areas of continued stress. Use emollient lotions; limit use of soap for bathing.</p>	<p>RATIONALE</p> <p>Edematous tissues are more prone to breakdown and to the formation of decubitus. Ascites may stretch the skin to the point of tearing in severe cirrhosis.</p>
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<p>ACTIONS/INTERVENTIONS</p> <p>Skin Surveillance (NIC)</p>	<p>RATIONALE</p>
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<p>Independent</p> <p>Encourage/assist with repositioning on a regular schedule, while in bed/chair, and active/passive ROM exercises as appropriate.</p> <p>Recommend elevating lower extremities.</p> <p>Keep linens dry and free of wrinkles.</p> <p>Suggest clipping fingernails short; provide mittens/gloves if indicated.</p> <p>Encourage/provide perineal care following urination and bowel movement.</p> <p>Collaborative</p> <p>Use alternating pressure mattress, egg-crate mattress, waterbed, sheepskins, as indicated.</p> <p>Apply calamine lotion, provide baking soda baths. Administer medications such as cholestyramine (Questran), hydroxyzine (Atarax), diphenhydramine (Benadryl), if indicated.</p>	<p>Repositioning reduces pressure on edematous tissues to improve circulation. Exercises enhance circulation and improve/maintain joint mobility.</p> <p>Enhances venous return and reduces edema formation in extremities.</p> <p>Moisture aggravates pruritus and increases risk of skin breakdown.</p> <p>Prevents patient from inadvertently injuring the skin, especially while sleeping.</p> <p>Prevents skin excoriation breakdown from bile salts.</p> <p>Reduces dermal pressure, increases circulation, and diminishes risk of tissue ischemia/breakdown.</p> <p>May be soothing/provide relief of itching associated with jaundice, bile salts in skin.</p>
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<p>NURSING DIAGNOSIS: Breathing Pattern, risk for ineffective</p> <p>Risk factors may include</p> <ul style="list-style-type: none"> Intra-abdominal fluid collection (ascites) Decreased lung expansion, accumulated secretions Decreased energy, fatigue <p>Possibly evidenced by</p> <p>[Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Respiratory Status: Ventilation (NOC)</p> <p>Maintain effective respiratory pattern; be free of dyspnea and cyanosis, with ABGs and vital capacity within acceptable range.</p>
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ACTIONS/INTERVENTIONS	RATIONALE
<p>Ventilation Assistance (NIC)</p>	
<p>Independent</p>	
<p>Monitor respiratory rate, depth, and effort.</p>	<p>Rapid shallow respirations/dyspnea may be present because of hypoxia and/or fluid accumulation in abdomen.</p>
<p>Auscultate breath sounds, noting crackles, wheezes, rhonchi.</p>	<p>Indicates developing complications (e.g., presence of adventitious sounds reflects accumulation of fluid/secretions; absent/diminished sounds suggest atelectasis), increasing risk of infection.</p>
<p>Investigate changes in level of consciousness.</p>	<p>Changes in mentation may reflect hypoxemia and respiratory failure, which often accompany hepatic coma.</p>
<p>Keep head of bed elevated. Position on sides.</p>	<p>Facilitates breathing by reducing pressure on the diaphragm, and minimizes risk of aspiration of secretions.</p>
<p>Encourage frequent repositioning and deep-breathing exercises/coughing as appropriate.</p>	<p>Aids in lung expansion and mobilizing secretions.</p>
<p>Monitor temperature. Note presence of chills, increased coughing, changes in color/character of sputum.</p>	<p>Indicative of onset of infection, e.g., pneumonia.</p>
<p>Collaborative</p>	
<p>Monitor serial ABGs, pulse oximetry, vital capacity measurements, chest x-rays.</p>	<p>Reveals changes in respiratory status, developing pulmonary complications.</p>
<p>Provide supplemental O₂ as indicated.</p>	<p>May be necessary to treat/prevent hypoxia. If respirations/oxygenation inadequate, mechanical ventilation may be required.</p>
<p>Demonstrate/assist with respiratory adjuncts, e.g., incentive spirometer.</p>	<p>Reduces incidence of atelectasis, enhances mobilization of secretions.</p>
<p>Prepare for/assist with acute care procedures, e.g.:</p> <p style="padding-left: 20px;">Paracentesis;</p>	<p>Occasionally done to remove ascites fluid to relieve abdominal pressure when respiratory embarrassment is not corrected by other measures.</p>
<p style="padding-left: 20px;">Peritoneovenous shunt.</p>	<p>Surgical implant of a catheter to return accumulated fluid in the abdominal cavity to systemic circulation via the vena cava; provides long-term relief of ascites and improvement in respiratory function.</p>

NURSING DIAGNOSIS: Injury, risk for [hemorrhage]

Risk factors may include

Abnormal blood profile; altered clotting factors (decreased production of prothrombin, fibrinogen, and factors VIII, IX, and X; impaired vitamin K absorption; and release of thromboplastin)
Portal hypertension, development of esophageal varices

Possibly evidenced by

[Not applicable; presence of signs and symptoms establishes an *actual* diagnosis.]

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Coagulation Status (NOC)

Maintain homeostasis with absence of bleeding.

Risk Control (NOC)

Demonstrate behaviors to reduce risk of bleeding.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Bleeding Precautions (NIC)</p> <p>Independent</p> <p>Assess for signs/symptoms of GI bleeding; e.g., check all secretions for frank or occult blood. Observe color and consistency of stools, NG drainage, or vomitus.</p> <p>Observe for presence of petechiae, ecchymosis, bleeding from one or more sites.</p> <p>Monitor pulse, BP (and CVP if available).</p> <p>Note changes in mentation/level of consciousness.</p> <p>Avoid rectal temperature; be gentle with GI tube insertions.</p> <p>Encourage use of soft toothbrush, electric razor, avoiding straining for stool, forceful nose blowing, and so forth.</p> <p>Use small needles for injections. Apply pressure to small bleeding/venipuncture sites for longer than usual.</p> <p>Recommend avoidance of aspirin-containing products.</p> <p>Collaborative</p> <p>Monitor Hb/Hct and clotting factors.</p>	<p>The GI tract (esophagus and rectum) is the most usual source of bleeding because of its mucosal fragility and alterations in hemostasis associated with cirrhosis.</p> <p>Subacute disseminated intravascular coagulation (DIC) may develop secondary to altered clotting factors.</p> <p>An increased pulse with decreased BP and CVP can indicate loss of circulating blood volume, requiring further evaluation.</p> <p>Changes may indicate decreased cerebral perfusion secondary to hypovolemia, hypoxemia.</p> <p>Rectal and esophageal vessels are most vulnerable to rupture.</p> <p>In the presence of clotting factor disturbances, minimal trauma can cause mucosal bleeding.</p> <p>Minimizes damage to tissues, reducing risk of bleeding/hematoma.</p> <p>Prolongs coagulation, potentiating risk of hemorrhage.</p> <p>Indicators of anemia, active bleeding, or impending complications (e.g., DIC).</p>

ACTIONS/INTERVENTIONS	RATIONALE
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<p>Bleeding Precautions (NIC)</p> <p>Collaborative</p> <p>Administer medications as indicated: Supplemental vitamins (e.g., vitamins K, D, and C);</p> <p>Stool softeners.</p> <p>Provide gastric lavage with room temperature/cool saline solution or water as indicated.</p> <p>Assist with insertion/maintenance of GI/esophageal tube (e.g., Sengstaken-Blakemore tube).</p> <p>Prepare for surgical procedures, e.g., direct ligation (banding) or varices, esophagogastric resection, splenorenal-portacaval anastomosis.</p>	<p>Promotes prothrombin synthesis and coagulation if liver is functional. Vitamin C deficiencies increase susceptibility of GI system to irritation/bleeding.</p> <p>Prevents straining for stool with resultant increase in intra-abdominal pressure and risk of vascular rupture/hemorrhage.</p> <p>In presence of acute bleeding, evacuation of blood from GI tract reduces ammonia production and risk of hepatic encephalopathy.</p> <p>Temporarily controls bleeding of esophageal varices when control by other means (e.g., lavage) and hemodynamic stability cannot be achieved.</p> <p>May be needed to control active hemorrhage or to decrease portal and collateral blood vessel pressure to minimize risk of recurrence of bleeding.</p>
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<p>NURSING DIAGNOSIS: Confusion, risk for acute</p> <p>Risk factors may include Alcohol abuse Inability of liver to detoxify certain enzymes/drugs</p> <p>Possibly evidenced by [Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Cognitive Ability (NOC) Maintain usual level of mentation/reality orientation. Initiate behaviors/lifestyle changes to prevent or minimize recurrence of problem.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Reality Orientation (NIC)</p> <p>Independent</p> <p>Observe for changes in behavior and mentation, e.g., lethargy, confusion, drowsiness, slowing/slurring of speech, and irritability (may be intermittent). Arouse patient at intervals as indicated.</p> <p>Review current medication regimen/schedules.</p> <p>Evaluate sleep/rest schedule.</p> <p>Note development/presence of asterixis, fetor hepaticus, seizure activity.</p> <p>Consult with SO about patient's usual behavior and mentation.</p> <p>Have patient write name periodically and keep this record for comparison. Report deterioration of ability. Have patient do simple arithmetic computations.</p> <p>Reorient to time, place, person as needed.</p> <p>Maintain a pleasant, quiet environment and approach in a slow, calm manner. Encourage uninterrupted rest periods.</p> <p>Provide continuity of care. If possible, assign same nurse over a period of time.</p> <p>Reduce provocative stimuli, confrontation. Refrain from forcing activities. Assess potential for violent behavior.</p> <p>Discuss current situation, future expectation.</p> <p>Maintain bedrest, assist with self-care activities.</p> <p>Identify/provide safety needs, e.g., supervision during smoking, bed in low position, side rails up and pad if necessary. Provide close supervision.</p> <p>Investigate temperature elevations. Monitor for signs of infection.</p>	<p>Ongoing assessment of behavior and mental status is important because of fluctuating nature of impending hepatic coma.</p> <p>Adverse drug reactions or interactions (e.g., cimetidine plus antacids) may potentiate/exacerbate confusion.</p> <p>Difficulty falling/staying asleep leads to sleep deprivation, resulting in diminished cognition and lethargy.</p> <p>Suggests elevating serum ammonia levels; increased risk of progression to encephalopathy.</p> <p>Provides baseline for comparison of current status.</p> <p>Easy test of neurological status and muscle coordination.</p> <p>Assists in maintaining reality orientation, reducing confusion/anxiety.</p> <p>Reduces excessive stimulation/sensory overload, promotes relaxation, and may enhance coping.</p> <p>Familiarity provides reassurance, aids in reducing anxiety, and provides a more accurate documentation of subtle changes.</p> <p>Avoids triggering agitated, violent responses; promotes patient safety.</p> <p>Patient/SO may be reassured that intellectual (as well as emotional) function may improve as liver involvement resolves.</p> <p>Reduces metabolic demands on liver, prevents fatigue, and promotes healing, lowering risk of ammonia buildup.</p> <p>Reduces risk of injury when confusion, seizures, or violent behavior occurs.</p> <p>Infection may precipitate hepatic encephalopathy caused by tissue catabolism and release of nitrogen.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Reality Orientation (NIC)</p> <p>Independent</p> <p>Recommend avoidance of narcotics or sedatives, antianxiety agents, and limiting/restricting use of medications metabolized by the liver.</p> <p>Collaborative</p> <p>Monitor laboratory studies, e.g., ammonia, electrolytes, pH, BUN, glucose, CBC with differential.</p> <p>Eliminate or restrict protein in diet. Provide glucose supplements, adequate hydration.</p> <p>Administer medications as indicated:</p> <p style="padding-left: 20px;">Electrolytes:</p> <p style="padding-left: 40px;">Stool softeners, colonic purges (e.g., magnesium sulfate), enemas, lactulose;</p> <p style="padding-left: 40px;">Bactericidal agents, e.g., neomycin (Mycifradin), kanamycin (Kantrex).</p> <p>Administer supplemental O₂.</p> <p>Assist with procedures as indicated, e.g., dialysis, plasmapheresis, or extracorporeal liver perfusion.</p>	<p>Certain drugs are toxic to the liver, whereas other drugs may not be metabolized because of cirrhosis, causing cumulative effects that affect mentation, mask signs of developing encephalopathy, or precipitate coma.</p> <p>Elevated ammonia levels, hypokalemia, metabolic alkalosis, hypoglycemia, anemia, and infection can precipitate or potentiate development of hepatic coma.</p> <p>Ammonia (product of the breakdown of protein in the GI tract) is responsible for mental changes in hepatic encephalopathy. Dietary changes may result in constipation, which also increases bacterial action and formation of ammonia. Glucose provides a source of energy, reducing need for protein catabolism. <i>Note:</i> Vegetable protein may be better tolerated than meat protein.</p> <p>Corrects imbalances and may improve cerebral function/metabolism of ammonia.</p> <p>Removes protein and blood from intestines. Acidifying the intestine produces diarrhea and decreases production of nitrogenous substances, reducing risk/severity of encephalopathy. <i>Note:</i> Long-term use of lactulose may be required for patients with hepatic encephalopathy to reduce ammonia on a daily/regular basis.</p> <p>Destroys intestinal bacteria, reducing production of ammonia, to prevent encephalopathy.</p> <p>Mentation is affected by O₂ concentration and utilization in the brain.</p> <p>May be used to reduce serum ammonia levels if encephalopathy develops/other measures are not successful.</p>

NURSING DIAGNOSIS: Self-Esteem/Body Image disturbed

May be related to

Biophysical changes/altered physical appearance
Uncertainty of prognosis, changes in role function
Personal vulnerability
Self-destructive behavior (alcohol-induced disease)

Possibly evidenced by

Verbalization of change/restriction in lifestyle
Fear of rejection or reaction by others
Negative feelings about body/abilities
Feelings of helplessness, hopelessness, or powerlessness

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Self-Esteem (NOC)

Verbalize understanding of changes and acceptance of self in the present situation.
Identify feelings and methods for coping with negative perception of self.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Self-Esteem Enhancement (NIC)</p> <p>Independent</p> <p>Discuss situation/encourage verbalization of fears and concerns. Explain relationship between nature of disease and symptoms.</p> <p>Support and encourage patient; provide care with a positive, friendly attitude.</p> <p>Encourage family/SO to verbalize feelings, visit freely/participate in care.</p> <p>Assist patient/SO to cope with change in appearance; suggest clothing that does not emphasize altered appearance, e.g., use of red, blue, or black clothing.</p>	<p>Patient is very sensitive to body changes and may also experience feelings of guilt when cause is related to alcohol (70%) or other drug use.</p> <p>Caregivers sometimes allow judgmental feelings to affect the care of patient and need to make every effort to help patient feel valued as a person.</p> <p>Family members may feel guilty about patient's condition and may be fearful of impending death. They need nonjudgmental emotional support and free access to patient. Participation in care helps them feel useful and promotes trust between staff, patient, and SO.</p> <p>Patient may present unattractive appearance as a result of jaundice, ascites, ecchymotic areas. Providing support can enhance self-esteem and promote patient sense of control.</p>
<p>Collaborative</p> <p>Refer to support services, e.g., counselors, psychiatric resources, social service, clergy, and/or alcohol treatment program.</p>	<p>Increased vulnerability/concerns associated with this illness may require services of additional professional resources.</p>

NURSING DIAGNOSIS: Knowledge, deficient [Learning Need] regarding condition, prognosis, treatment, self-care, and discharge needs

May be related to

Lack of exposure/recall; information misinterpretation
Unfamiliarity with information resources

Possibly evidenced by

Questions; request for information, statement of misconception
Inaccurate follow-through of instructions/development of preventable complications

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Knowledge: Illness Care (NOC)

Verbalize understanding of disease process/prognosis, potential complications.
Correlate symptoms with causative factors.

Knowledge; Health Behaviors (NOC)

Identify/initiate necessary lifestyle changes and participate in care.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Teaching: Disease Process (NIC)</p> <p>Independent</p> <p>Review disease process/prognosis and future expectations.</p> <p>Stress importance of avoiding alcohol. Give information about community services available to aid in alcohol rehabilitation if indicated.</p> <p>Inform patient of altered effects of medications with cirrhosis and the importance of using only drugs prescribed or cleared by a healthcare provider who is familiar with patient's history.</p> <p>Review procedure for maintaining function of peritoneovenous shunt when present.</p> <p>Assist patient identifying support person(s).</p> <p>Emphasize the importance of good nutrition. Recommend avoidance of high-protein/salty foods, onions, and strong cheeses. Provide written dietary instructions.</p>	<p>Provides knowledge base from which patient can make informed choices.</p> <p>Alcohol is the leading cause in the development of cirrhosis.</p> <p>Some drugs are hepatotoxic (especially narcotics, sedatives, and hypnotics). In addition, the damaged liver has a decreased ability to metabolize all drugs, potentiating cumulative effect and/or aggravation of bleeding tendencies.</p> <p>Insertion of a Denver shunt requires patient to periodically pump the chamber to maintain patency of the device. Patients with a LeVeen shunt may wear an abdominal binder and/or engage in a Valsalva maneuver to maintain shunt function.</p> <p>Because of length of recovery, potential for relapses, and slow convalescence, support systems are extremely important in maintaining behavior modifications.</p> <p>Proper dietary maintenance and avoidance of foods high in sodium and protein aid in remission of symptoms and help prevent ammonia buildup and further liver damage. Written instructions are helpful for patient to refer to at home.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Teaching: Disease Process (NIC)</p> <p>Independent</p> <p>Stress necessity of follow-up care and adherence to therapeutic regimen.</p> <p>Discuss sodium and salt substitute restrictions and necessity of reading labels on food and OTC drugs.</p> <p>Encourage scheduling activities with adequate rest periods.</p> <p>Promote diversional activities that are enjoyable to patient.</p> <p>Recommend avoidance of persons with infections, especially URI.</p> <p>Identify environmental dangers, e.g., carbon tetrachloride-type cleaning agents, exposure to hepatitis.</p> <p>Instruct patient/SO of signs/symptoms that warrant notification of healthcare provider, e.g., increased abdominal girth; rapid weight loss/gain; increased peripheral edema; increased dyspnea, fever; blood in stool or urine; excess bleeding of any kind; jaundice.</p> <p>Instruct SO to notify healthcare providers of any confusion, untidiness, night wandering, tremors, or personality change.</p>	<p>Chronic nature of disease has potential for life-threatening complications. Provides opportunity for evaluation of effectiveness of regimen, including patency of shunt if used.</p> <p>Minimizes ascites and edema formation. Overuse of substitutes may result in other electrolyte imbalances. Food, OTC/personal care products (e.g., antacids, some mouthwashes) may contain sodium or alcohol.</p> <p>Adequate rest decreases metabolic demands on the body and increases energy available for tissue regeneration.</p> <p>Prevents boredom and minimizes anxiety and depression.</p> <p>Decreased resistance, altered nutritional status, and immune response (e.g., leukopenia may occur with splenomegaly) potentiate risk of infection.</p> <p>Can precipitate recurrence.</p> <p>Prompt reporting of symptoms reduces risk of further hepatic damage and provides opportunity to treat complications before they become life-threatening.</p> <p>Changes (reflecting deterioration) may be more apparent to SO, although insidious changes may be noted by others with less frequent contact with patient.</p>

POTENTIAL CONSIDERATIONS following acute hospitalization (dependent on patient's age, physical condition/presence of complications, personal resources, and life responsibilities)

Fatigue—decreased metabolic energy production, states of discomfort, altered body chemistry (e.g., changes in liver function, effect on target organs, alcohol withdrawal).

Nutrition: imbalanced, less than body requirements—inadequate diet; inability to process/digest nutrients; anorexia, nausea/vomiting, indigestion, early satiety (ascites); abnormal bowel function.

Therapeutic Regimen: risk for ineffective management—perceived benefit, social support deficit, economic difficulties.

Family Processes, dysfunctional: alcoholism—abuse of alcohol, resistance to treatment, inadequate coping/lack of problem-solving skills, addictive personality/codependency.

Caregiver Role Strain, risk for—addiction or codependency, family dysfunction before caregiving situation, presence of situational stressors, such as economic vulnerability, hospitalization, changes in employment.