Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Milk thistle may have some therapeutic effects in liver disease, but no controlled trials have shown efficacy for ascites at this time.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient concerning good sources of key nutrients to include and which nutrients to limit. Instruct patient to follow high-energy, high-protein diet to prevent wasting.
- Ensure that the patient follows a 2-g, low-sodium diet. Explain which foods have hidden sources of sodium, and share recipes if needed.
- For chylous ascites, treatment is generally managed through a hospital stay.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Ascites
  http://www.nlm.nih.gov/medlineplus/ency/article/000286.htm
- Chylous Ascites
  http://emedicine.medscape.com/article/185777-overview
- Medicine Net
  http://www.medicinenet.com/ascites/article.htm

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leak post surgery is low (1–4%), this complication can present significant challenges (Smoke and Delegge, 2008). Any source of large fluid volume losses, lymph vessel obstruction, or leakage may cause chylous effusions in the peritoneal cavities. Most chylous effusions heal spontaneously. Early introduction of enteral feeding may encourage chyle leaks (Malik et al, 2007), whereas total parenteral nutrition along with somatostatin can relieve the symptoms rapidly (Huang et al, 2004).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: No specific genetic causes are clear in cases of ascites.

Clinical/History	Lab Work	
Height	Serum ascites-albumin gradient (>1.1 g/dL = portal hypertension)	ALT
Weight		AST
Dry weight or estimated dry weight		H & H (high in hemochromatosis)
BMI	Alb (decreased)	Serum Fe, ferritin
Diet history	Transthyretin	TIBC,% saturation
BP	CRP	Gluc
I & O	Na^+, K^+	Chol
Temperature	Ca^{++}, Mg^{++}	Trig
Ascites, mild to severe	BUN, creatinine (Creat)	
Ultrasonography		

INTERVENTION

OBJECTIVES

- Reduce fluid retention, usually by diuretics. Mild ascites may present with fluid excess of 3–5 kg; moderate ascites may present with excess of 7–9 kg; and severe ascites may present with excess of 14–15 kg above usual weight.
- Prevent electrolyte imbalances.
- Prevent further pain, fatigue, loss of lean body mass (LBM), and anorexia.
- If possible, prevent hepatorenal syndrome, which can occur in patients with severe liver disease. If severe, it may require transplantation. Prepare for surgery, especially nutritionally (Hasse, 2006).
- Individualize diet as needs change.
- For **chylous ascites**, treat the underlying cause to decrease production of the chylous fluid. Malnutrition is a common result if left untreated; essential fatty acid deficiency must be avoided. Fluid and electrolyte replacement may be needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Sodium Intake-Ascites

Assessment Data: Dietary intake records.

Nutrition Diagnosis (PES): Excessive sodium intake related to presence of ascites and portal hypertension as evidenced by paracentesis of 7–8 kg over 24 hours.

Intervention: Food and Nutrient Delivery—manage sodium intake. Educate about sodium sources and requirements. Counsel about preferred foods that are high in sodium and ways to alter intake that are acceptable; how to shop, dine out, travel.

Monitoring and Evaluation: Track food intake through food diary. Follow-up on intake of sodium and alleviation of ascites.

FOOD AND NUTRITION

- Energy needs are often as high as 1.5 times normal, and protein needs are often 1.5 g/kg of body weight (Hasse and Matarese, 2008). Smaller, more frequent meals are often better tolerated.
- If TF or central parenteral nutrition (CPN) is needed, use nutrient-dense formula but not glutamine-enriched formula; glutamine may increase ammonia production. While no high-quality data are available to prove that enteral nutrition is of benefit (Koretz, 2007), malnutrition should be addressed.
- Ensure that intake of vitamins and minerals is adequate. Water-soluble forms of vitamins may be needed; zinc and magnesium may be needed since levels are often low after diuretic therapy (Hasse and Matarese, 2008). Monitor for signs of malnutrition.
- Fluid restriction may be necessary (1–1.5 L/d), with two thirds with meals and one third for thirst/medicines.
- Restrict patient's intake of sodium to 2 g/d (Hasse and Matarese, 2008).
- Often, patients take spironolactone (Aldactone) or have renal insufficiency, which may increase potassium retention. Diet should be altered in potassium if serum levels so indicate. Other diuretics may cause potassium losses.
- For **chylous ascites**, a low-fat diet or enteral feeding is needed with MCTs as the preferred fat source; the addition of essential fatty acids (EFAs) will be needed. Adequate protein and calories are also needed since there may be significant losses. If oral diet fails, CPN may be needed (Assumpcao et al, 2008). Water-miscible forms of fat-soluble vitamins may be needed, along with extra fluid and electrolytes.

Common Drugs Used and Potential Side Effects

- Diuretics are the most important treatment (Rosner et al, 2006). Furosemide (Lasix) is not very effective. Check whether the specific drug retains or spares potassium; spironolactone spares potassium.
- Albumin replacement, while costly, may help to maintain oncotic pressure.
- Somatostatin analogs have been demonstrated to be effective (Huang et al, 2004).
- With bacterial peritonitis, antibiotic therapy is needed. Monitor for specific side effects. PPIs increase enteric bacterial colonization, overgrowth, and translocation (Campbell et al, 2008).

- Obesity, diabetes, and hyperinsulinemia play a role in the development of hepatic steatosis; weight loss remains a critical part of protecting the liver against damage.
- Identify sources of assistance for persons who need help with meal preparation or with access to meals.

Patient Education—Foodborne Illness

- If home TF is needed, teach appropriate sanitation and food-handling procedures.

For More Information

- Alcoholics Anonymous (AA) World Services
  http://www.alcoholics-anonymous.org/
- Alcoholic Hepatitis
  http://www.emedicine.com/med/topic101.htm
- International Society for Biomedical Research on Alcoholism
  http://www.isbra.com/
- National Council on Alcoholism and Drug Dependence
  http://www.ncadd.org/
- National Institute on Alcohol Abuse and Alcoholism (NIAAA)
  http://www.niaaa.nih.gov/
- International Research Society on Alcoholism
  http://www.rsoa.org/
- Substance Abuse and Mental Health Administration (DHHS)
  http://www.samhsa.gov/

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ASCITES AND CHYLOUS ASCITES

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Ascites is defined as a distended abdomen due to pathological fluid in the peritoneal cavity. The development of ascites indicates a pathological imbalance between the production and resorption of intraperitoneal fluid; appearance and composition vary based on the underlying pathophysiology (Rochling and Zetterman, 2009). Ascites develops in decompensated cirrhosis, cardiac failure, or renal insufficiency. Portal hypertensive gastropathy (PHG) causes upper gastrointestinal bleeding in advanced cases. Liver transplantation may be the only way to improve survival in refractory ascites (Sandhu and Sanyal, 2005).

Although weight is not used for nutritional assessment here, it does help determine fluid balance. The goal of diuretic therapy in ascites is to promote weight loss of 1–3 kg/d. Nutrient depletion can occur if left untreated; fat, proteins, fat-soluble vitamins, and electrolytes may be lost. An oral diet devoid of long-chain triglycerides (LCTs) but that includes medium-chain triglycerides (MCTs) may be used in mild cases.

Management of ascites from decompensated liver disease focus on low-sodium diets and diuretics, supplemented by paracentesis or transvenous intrahepatic portosystemic shunts (Rochling and Zetterman, 2009). While paracentesis improves patient comfort and reduces intra-abdominal pressure and secondary renal dysfunction, it also carries risk for spontaneous bacterial peritonitis (SBP) or renal failure (Sargent, 2006). Bacterial contamination of ascites fluid leading to SBP is caused by bacterial translocation with subsequent bacteremia; proton pump inhibitors (PPIs) suppress gastric acid secretion, and possibly should be avoided in this population (Bajaj et al, 2009).

Chylous ascites is a rare form of ascites, resulting from increased hydrostatic pressure and lymphatic blockade. Accumulation of LCT-dense chyle occurs in the peritoneum. Chyle leaks are a rare complication following abdominal surgery, trauma, cancer, or fistula. Although the incidence of chyle

leak post surgery is low (1–4%), this complication can present significant challenges (Smoke and Delegge, 2008). Any source of large fluid volume losses, lymph vessel obstruction, or leakage may cause chylous effusions in the peritoneal cavities. Most chylous effusions heal spontaneously. Early introduction of enteral feeding may encourage chyle leaks (Malik et al, 2007), whereas total parenteral nutrition along with somatostatin can relieve the symptoms rapidly (Huang et al, 2004).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: No specific genetic causes are clear in cases of ascites.

Clinical/History	Lab Work	
Height	Serum ascites-albumin gradient (>1.1 g/dL = portal hypertension)	ALT
Weight		AST
Dry weight or estimated dry weight		H & H (high in hemochromatosis)
BMI	Alb (decreased)	Serum Fe, ferritin
Diet history	Transthyretin	TIBC,% saturation
BP	CRP	Gluc
I & O	Na^+, K^+	Chol
Temperature	Ca^{++}, Mg^{++}	Trig
Ascites, mild to severe	BUN, creatinine (Creat)	
Ultrasonography		

INTERVENTION

OBJECTIVES

- Reduce fluid retention, usually by diuretics. Mild ascites may present with fluid excess of 3–5 kg; moderate ascites may present with excess of 7–9 kg; and severe ascites may present with excess of 14–15 kg above usual weight.
- Prevent electrolyte imbalances.
- Prevent further pain, fatigue, loss of lean body mass (LBM), and anorexia.
- If possible, prevent hepatorenal syndrome, which can occur in patients with severe liver disease. If severe, it may require transplantation. Prepare for surgery, especially nutritionally (Hasse, 2006).
- Individualize diet as needs change.
- For **chylous ascites**, treat the underlying cause to decrease production of the chylous fluid. Malnutrition is a common result if left untreated; essential fatty acid deficiency must be avoided. Fluid and electrolyte replacement may be needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Sodium Intake-Ascites

Assessment Data: Dietary intake records.

Nutrition Diagnosis (PES): Excessive sodium intake related to presence of ascites and portal hypertension as evidenced by paracentesis of 7–8 kg over 24 hours.

Intervention: Food and Nutrient Delivery—manage sodium intake. Educate about sodium sources and requirements. Counsel about preferred foods that are high in sodium and ways to alter intake that are acceptable; how to shop, dine out, travel.

Monitoring and Evaluation: Track food intake through food diary. Follow-up on intake of sodium and alleviation of ascites.

FOOD AND NUTRITION

- Energy needs are often as high as 1.5 times normal, and protein needs are often 1.5 g/kg of body weight (Hasse and Matarese, 2008). Smaller, more frequent meals are often better tolerated.
- If TF or central parenteral nutrition (CPN) is needed, use nutrient-dense formula but not glutamine-enriched formula; glutamine may increase ammonia production. While no high-quality data are available to prove that enteral nutrition is of benefit (Koretz, 2007), malnutrition should be addressed.
- Ensure that intake of vitamins and minerals is adequate. Water-soluble forms of vitamins may be needed; zinc and magnesium may be needed since levels are often low after diuretic therapy (Hasse and Matarese, 2008). Monitor for signs of malnutrition.
- Fluid restriction may be necessary (1–1.5 L/d), with two thirds with meals and one third for thirst/medicines.
- Restrict patient's intake of sodium to 2 g/d (Hasse and Matarese, 2008).
- Often, patients take spironolactone (Aldactone) or have renal insufficiency, which may increase potassium retention. Diet should be altered in potassium if serum levels so indicate. Other diuretics may cause potassium loss.
- For **chylous ascites**, a low-fat diet or enteral feeding is needed with MCTs as the preferred fat source; the addition of essential fatty acids (EFAs) will be needed. Adequate protein and calories are also needed since there may be significant losses. If oral diet fails, CPN may be needed (Assumpcao et al, 2008). Water-miscible forms of fat-soluble vitamins may be needed, along with extra fluid and electrolytes.

Common Drugs Used and Potential Side Effects

- Diuretics are the most important treatment (Rosner et al, 2006). Furosemide (Lasix) is not very effective. Check whether the specific drug retains or spares potassium; spironolactone spares potassium.
- Albumin replacement, while costly, may help to maintain oncotic pressure.
- Somatostatin analogs have been demonstrated to be effective (Huang et al, 2004).
- With bacterial peritonitis, antibiotic therapy is needed. Monitor for specific side effects. PPIs increase enteric bacterial colonization, overgrowth, and translocation (Campbell et al, 2008).

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Alcohol Intake

Assessment Data: Dietary intake records: low protein and energy intake for age/gender. Intake of one fifth of vodka per day to the exclusion of most meals.

Nutrition Diagnosis (PES): Excessive alcohol intake >25–30 g/d related to daily consumption above this level as evidenced by alcohol-induced liver injury, elevated LFTs and ascites.

Intervention:

Food and Nutrient Delivery: ND 1.1 General Healthful diet (avoid alcohol); ND 3.1.4 Modified food—increased calorie/protein intake.

Education: E 1.3 Survival Information. Educate about nutrient-dense foods and the role that alcohol plays in liver damage. E-1.1 Present concise and clear educational material with nutritional tips for patients with liver disease.

Counseling: C 2.5 Social support—avoid social outings with alcohol present.

Coordination of Care: RC 1.4 Referral to community agencies/programs. RC-1.3 Refer to social worker for alcohol rehabilitation. C-2.9 Relapse prevention by explaining the pros of following diet and medications as recommended, as the importance of maintain sober.

Monitoring and Evaluation: Track food intake through food diary or history. Coordinate care for rehabilitation program. Follow-up on intake of energy, protein and nutrients after omission of alcohol.

- Correct fluid and electrolyte imbalances, nutritional deficits such as iron deficiency anemia from chronic blood loss in varices, ulcers, and vomiting.
- Be honest and direct in approach. Gently confront conflicting information when stated by the patient.

FOOD AND NUTRITION

- Avoid alcohol to allow the liver to begin heal (DiCecco and Francisco-Ziller, 2006).
- Malnourished alcoholics should consume a diet rich in carbohydrate and protein, preferentially via the oral or enteral route. Provide protein as 1.5 g/kg body weight if malnourished. Plan sufficient carbohydrates and fat to spare protein, but monitor for hyperglycemia or dyslipidemia.
- In hypertensive patients, a Dietary Approaches to Stop Hypertension (DASH) diet may be planned that provides a sufficient mixture of nutrients without excessive kilocalories. All fasting or very low–calorie diets should be avoided.
- Include a mix of fat from omega-3 (fish oils), omega-6 fatty acids, and medium-chain fatty acids.
- Micronutrient deficiencies require supplementation. Supplement the diet with B-complex vitamins, but supplemental vitamins A and D may not be well tolerated. Oral diet should provide adequate amounts of vitamins C, E, and K, phosphorus, potassium, selenium, magnesium, zinc, and calcium.
- Provide small frequent meals to prevent hypoglycemia, resulting from limited glycogen storage.
- Monitor iron intake to avoid excesses from diet or supplements, especially if there is the possibility of iron storage disease.
- Make meals appealing to stimulate the appetite.
- If TF is needed, avoid glutamine-enriched formulas which may increase ammonia levels.

Common Drugs Used and Potential Side Effects

- Corticosteroids have become the standard of care in patients with severe alcoholic hepatitis (Lucey, 2009). Methylprednisolone improves the ability to produce albumin and to normalize PT and bilirubin levels. Side effects may include negative nitrogen balance, hypocalemia, or hyperglycemia.
- Pharmacotherapy for alcoholism with naltrexone, acamprosate, topiramate, and baclofen is exciting (Lucey, 2009). Naltrexone is more effective in some individuals than in others (Rubio et al, 2005).
- Disulfiram (Antabuse) is given with patient's consent. It causes the patient to vomit after ingesting alcohol and can be dangerous.
- Beta-blockers (propranolol, nadolol) or octreotide (Sandostatin) may be used to reduce portal hypertension when varices occur.
- Insulin may be necessary; do not mix with alcohol. Alcohol intake may cause severe hypoglycemia in patients taking insulin (Pedersen-Bjergaard et al, 2005). Metformin should be avoided in patients with liver disease.

Herbs, Botanicals, and Supplements

- Antioxidants are increasingly used. Agents involved in methionine metabolism such as SAM and betaine have shown efficacy in liver disease. Milk thistle (*Silybum marianum*) may have some therapeutic effect as well. *Curcuma longa* (turmeric) and *Glycyrrhiza glabra* (licorice) are being evaluated. Tea polyphenols, especially green tea, may alleviate liver damage (Zhang et al, 2005).
- Herbs and botanical supplements should not be used without discussing with the physician. Chaparral is especially toxic to the liver and should be avoided; severe hepatitis or liver failure may result. Aloe vera should be avoided orally.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient on the sources of necessary nutrients in the diet and use of the prescribed multivitamins. Help patient in the planning and preparing of appetizing, nutrient-dense meals.
- Explain that alcohol is metabolized readily by the liver but cannot be used for muscular activity or energy production. Chemical addiction is a disease; self-help programs and follow-up can reduce dependency.
- General multivitamin–mineral supplementation may improve a poor appetite.

the liver and stomach (Lieber, 2005). Alcohol dehydrogenase is made with zinc. Alcohol decreases absorption of fats, fat-soluble vitamins, thiamin, folic acid, vitamin B_{12}, and zinc. Nicotine adenine dinucleotide (NADH) is significant in alcohol metabolism by reduction of pyruvate and promotion of steatosis.

Adequate nutrition is critical and should be provided by TF if necessary (Maher, 2007). A prompt decline in serum bilirubin within 1 week indicates a favorable response to therapy; nonresponders have a 6-month mortality rate of 50% or higher (Maher, 2007).

Plasma homocysteine levels are altered in actively drinking patients, causing brain atrophy and withdrawal seizures (Bleich et al, 2005). Methionine needs to be activated to *S*-adenosylmethionine (SAM); this metabolism is impaired in liver disease. Folate deficiency accentuates abnormal methionine metabolism, lipid oxidation, and liver injury (Halsted et al, 2002; Schalinske and Nieman, 2005). SAM, betaine, and folate decrease oxidative stress by upregulation of glutathione and interleukin-10 and downregulation of tumor necrosis factor-alpha, TNF-a (Purohit et al, 2008). No benefit has been found in randomized, placebo-controlled clinical trials of colchicine, S-adenosylmethionine (SAMe), or phosphatidylcholine (Lucey, 2009). Betaine may attenuate ALD by increasing synthesis of SAM and glutathione, decreasing homocysteine (tHcy) levels (Song et al, 2008). More research is indicated.

Alcohol-induced liver injury is an immunological response of the liver; neutrophils damage liver cells through cytotoxicity (Leevy and Elbeshbeshy, 2005). Men and women metabolize alcohol differently. It takes less time and lower doses of alcohol exposure to cause liver damage in females than in males. Community-dwelling heavy drinkers who are not in alcoholism treatment have dose-related gray matter volume losses (Cardenas et al, 2005).

Treatment strategies for ALD include lifestyle changes for abstinence from alcohol consumption. Nutrition therapy and medications are also important. Serious alcoholic hepatitis has a mortality record of up to 50%. If necessary liver transplantation may be life-saving.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The dopamine (DR2) receptor promotes effects of alcohol. People with a genetic deficit of beta-endorphin peptide are susceptible (Manzardo et al, 2005; Zalewska-Kaszubska and Czarnecka, 2005). The dopaminergic mesolimbic system activates the endogenous mu and delta opioid receptors; mu receptor polymorphisms may be associated with ethanol dependence (Job et al, 2007). Polymorphisms in cytochrome P450 2E1 (CYP2E1), the major microsomal ethanol metabolizing enzyme, can alter detoxification of alcohol by glutathione-S-transferases M1 (GSTM1) and gamma-aminobutyric acid receptor gamma2 (Khan et al, 2009).

Clinical/History

- Height
- Weight
- Body mass index (BMI)
- Usual body weight (UBW)
- Diet history
- Blood pressure (BP)
- Intake and output (I & O)
- Food intolerances, taste aversions
- Anorexia, nausea, vomiting, diarrhea
- Scurvy—ecchymoses, hemorrhagic gingivitis, perifollicular hemorrhages
- Leg edema, poor wound healing
- CT scan or ultrasound of abdomen
- Liver biopsy
- Ascites (mild, moderate, or severe)
- Fatigue
- CAGE test (Cut down, Annoyed, Guilty, Eye opener)
- Alcohol Use Disorders Identification Test (AUDIT)
- Dual-energy x-ray absorptiometry (DEXA) bone scan

Lab Work

- Glucose (increased or decreased)
- Glucose tolerance test (sensitive and reliable)
- AST (increased)
- ALT (normal or only mildly elevated)
- INR
- Bilirubin (often elevated)
- Serum ammonia (may be elevated)
- Blood urea nitrogen (BUN) (low?)
- Albumin or transthyretin (low?)
- C-reactive protein (CRP)
- Triglycerides (increased?)
- Cholesterol (increased or decreased)
- WBC count
- Serum B_{12} and folate
- Plasma homocysteine (high?)
- Na^+ (hyponatremia?)
- K^+
- Hemoglobin and hematocrit (decreased)
- Serum Fe, ferritin
- Transferrin
- Uric acid (UA, increased)
- Globulin
- Alk phos (mildly elevated)
- Mg^{++} (decreased)
- Ca^{++}
- Serum phosphorus (decreased)

INTERVENTION

OBJECTIVES

- Remove alcohol to allow the disabled liver to function more effectively while protecting it from metabolic stress. Avoid alcohol in miscellaneous products, such as vinegar, sauces, and cough syrup.
- Improve health of liver so it can synthesize albumin and other serum proteins. Help liver tissue regenerate; replenish plasma proteins that are lost. Improve skeletal muscle synthesis.
- Prevent hypoglycemia from blocked gluconeogenesis. Correct metabolic syndrome, hyperglycemia, hypertension, or hypertriglyceridemia.
- Repair damage from fatty liver and diminished bile salt synthesis.
- Repair neural damage from malnutrition and malabsorption.

ALCOHOLIC LIVER DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 3

DEFINITIONS AND BACKGROUND

Alcohol is a hepatotoxin and is ulcerogenic, especially to the esophagus and other organs. Alcohol cannot be stored and is used preferentially over other energy fuels.

Alcoholic liver disease (ALD) is a major cause of illness and death. ALD affects about 2 million people in the United States. Signs and symptoms of alcoholism include restlessness, agitation, spider angiomas on the face or back or belly, insomnia, anorexia, weight loss, GI cramping, malnutrition, delirium tremens, and hand tremors. In men, altered hair distribution and gynecomastia may occur. Understanding alcohol addiction is key to treating ALD, since abstinence leads to improvement in all forms of alcoholic liver damage (Lucey, 2009). Section 4 addresses alcohol addiction. Table 8-2 lists stages and effects of alcoholism. Given the benefit of drug treatment, it is important to identify patients at risk of early mortality from alcoholic hepatitis using tools such as the Maddrey Discriminant Function, the Model of End-Stage Liver Disease score, and the Glasgow Alcoholic Hepatitis score (Maher, 2007).

Alcoholics may replace as much as one third of their daily energy requirements from alcohol. As a result, they are malnourished. Either they eat poorly or alcohol metabolism prevents them from properly absorbing, digesting, and using nutrients, particularly vitamin A (Plauth et al, 2006). Classic effects of malnutrition from alcoholism include Wernicke's encephalopathy, Korsakoff's psychosis, muscle wasting, weight loss, and liver disease.

Most tissues of the body contain enzymes capable of ethanol metabolism, but significant activity occurs only in

TABLE 8-2 Stages of Alcoholism-Related Effects

Stage	Condition	Effects
I.	Fatty liver (steatosis)	Reversible. Acetaldehyde promotes hepatic fat accumulation. Hepatomegaly, hypertriglyceridemia, hypoalbuminemia, cytochrome P-450 2E1 induction, free radical generation, lipid peroxidation, and increased transcription of proinflammatory mediators, including TNF-alpha, occur.
II.	Alcholic hepatitis	Fibrosis begins. Fever with tachycardia; liver enlargement is mild, and tenderness can occur.
III.	Cirrhosis	Not reversible. Diffuse necrosis and regeneration of fibrous tissue leading to loss of normal hepatic function.
IV.	Encephalopathy or Coma	May lead to death if not treated. Impaired mentation, altered neuromuscular function, and altered consciousness.