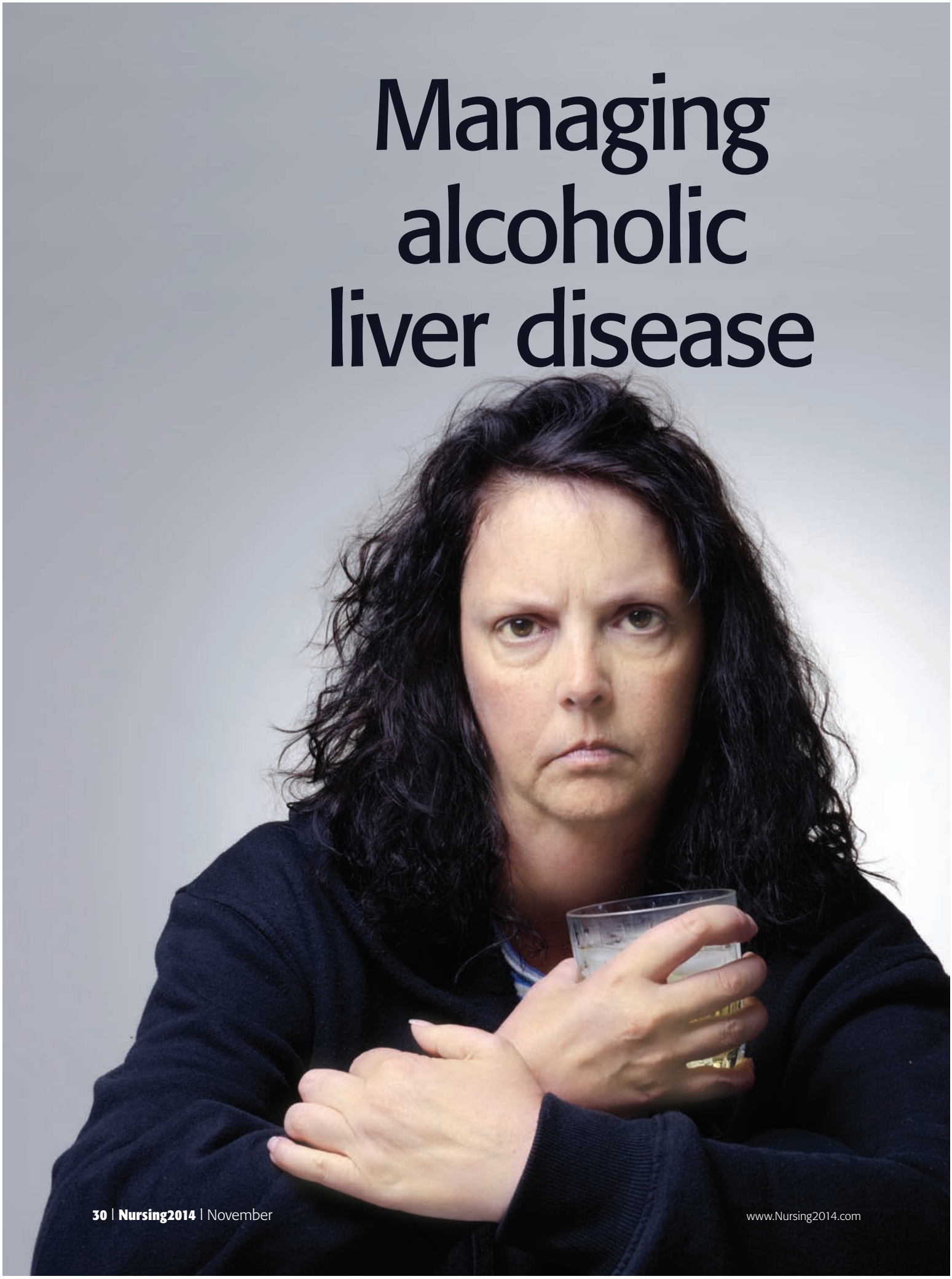
Managing alcoholic liver disease



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MS. J, 54, HAS A MEDICAL HISTORY that includes alcohol use disorder and steatohepatitis. This morning, she was admitted to the hospital with a diagnosis of alcoholic cirrhosis.

Ms. J is alert and oriented to person, place, and time but appears mildly agitated. She complains of fatigue, weakness, anorexia, nausea, abdominal discomfort, and pruritus. On physical assessment, the nurse notes jaundice of her skin and sclerae, petechiae and ecchymoses on her upper and lower extremities, peripheral edema, a distended abdomen with shifting dullness and a positive fluid wave, and dilated paraumbilical veins.

Blood test results include increased levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) with an AST-to-ALT ratio of 2:1, as well as increased levels of gamma-glutamyl transpeptidase (GGT), ammonia, and bilirubin. The nurse also notes thrombocytopenia, hypoalbuminemia, and a prolonged prothrombin time (PT).

Ms. J's case is an example of alcoholic cirrhosis caused by alcoholic liver disease (ALD). This article discusses the pathophysiology, incidence, and etiology of ALD, potential complications, medical and pharmacologic management, and nursing priorities. For a review of normal hepatic anatomy and physiology, including the liver's many functions in the body, see *Basic liver functions: A quick review*.

Major cause of liver disease

A serious consequence of chronic alcohol consumption, ALD poses complex medical and psychosocial challenges for the patient, family, and the healthcare team. ALD is a major cause of preventable liver disease in the United States and worldwide. In 2003, 44% of deaths from liver disease in the United States were attributed to alcohol.[1] And in 2004, alcohol consumption was responsible for 3.8% of global mortality.[2,3]

Research indicates that women are twice as susceptible to hepatic damage from excess alcohol consumption. Compared with men,

women are more prone to severe ALD even when consuming lower doses of alcohol.[4] Explanations for gender disparities are linked to differences in alcohol absorption and metabolism. Women achieve higher blood alcohol concentrations and have a higher proportion of body fat, aiding in increased absorption and distribution.[5] The synergistic impact of estrogen on oxidative stress and inflammation may also play a role.[3]

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.[1] The amount of alcohol in a standard 14-g drink depends on the type of alcohol (see *Standard alcohol measures by alcohol type*).

A recent meta-analysis found increased risks of mortality from cirrhosis among men and women drinking 12 to 24 g of alcohol per day.[4] Furthermore, drinking apart from meals increases the risk of ALD compared with drinking alcohol only with meals.[3]

Three stages of disease

ALD is a spectrum of hepatocellular injury that includes three stages of damage: fatty liver disease (steatosis), alcoholic hepatitis, and chronic hepatitis with hepatic fibrosis or cirrhosis. These stages of disease may be present simultaneously and aren't mutually exclusive.[1,3]

Simple ***steatosis,*** the hallmark of early ALD, may present with no clinical signs or symptoms. It's potentially reversible by abstaining from alcohol.[1,6] Steatosis is common in those who regularly consume 60 g of alcohol per day (for example, a half bottle of wine or more than 1 L of beer) or more but may also develop in people who drink less. In some people recovering from alcohol abuse, liver disease progresses to fibrosis and cirrhosis despite abstinence.[1]

Alcoholic hepatitis is characterized by liver inflammation and necrosis, resulting in edema and congestion. These changes compromise hepatocyte function and bile excretion.[3] Rapid onset of jaundice is a hallmark of alcoholic hepatitis. Other signs and symptoms include anorexia, nausea, vomiting, abdominal pain or tenderness, fever, ascites, and liver failure.[6] Alcoholic hepatitis is also potentially reversible if the patient abstains from alcohol, but failure to stop drinking may lead to disease progression from hepatitis to cirrhosis.[3]

In ***cirrhosis***, fibrotic scar tissue replaces normal liver tissue, disrupting blood flow. This stage of ALD brings a barrage of clinical manifestations. These patients are critically ill and are usually hospitalized with multisystem organ failure.[7] Signs and symptoms of cirrhosis are variable and may include anorexia, weight loss, jaundice, epigastric or upper right quadrant pain, pruritus, diarrhea, palmar erythema, spider nevi, peripheral edema, and caput medusae. Patients with alcoholic cirrhosis are prone to develop clinical decompensation due to portal hypertension and liver failure and are at risk for developing ascites, variceal bleeding, hepatic encephalopathy (HE), and hepatocellular carcinoma.[1,3,6,8]

Complications of ALD

The complications of all forms of liver disease, whether alcohol or nonalcohol related, arise from derangements in cellular structure and basic metabolic functions.

• **Portal hypertension**. Advanced cirrhosis results in portal hypertension, which affects blood flow from the intestines and abdominal organs to the liver. (See *Understanding portal hypertension*.) Development of portal hypertension depends on many factors, including increased resistance within the portal circulatory system with increased portal flow and endothelial dysfunction. To compensate for increased pressure in the portal circulation, collateral circulation develops to circumvent the affected vessels.

Although compensatory, bypassing the liver with collateral flow has several negative consequences. For example, as discussed below, decreased clearance of gut bacteria can lead to life-threatening infections such as spontaneous bacterial peritonitis (SBP).

Portal hypertension causes varices or dilated veins in the gastrointestinal (GI) tract that are at risk for rupture and hemorrhage. Because of collateral blood flow, paraumbilical veins may dilate and become visible on the abdomen (caput medusae). Right-sided pleural effusions often

Standard alcohol measures by alcohol type

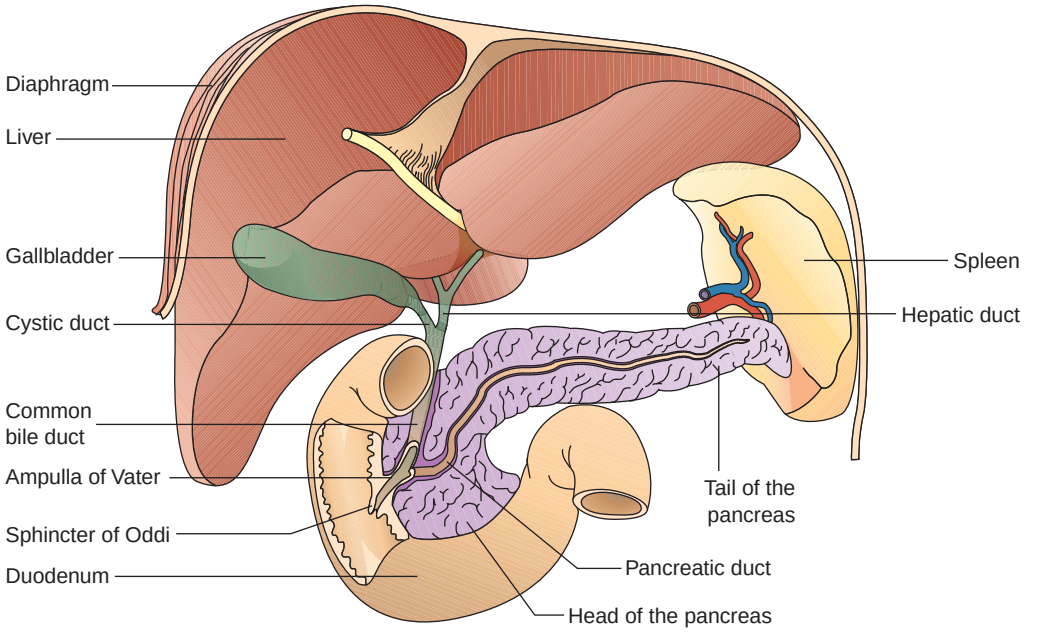
A standard drink contains 0.6 oz (14.0 g or 1.2 tablespoons) of pure alcohol. The number of ounces of alcohol comprising a standard drink depends on the type of alcohol.

Type of alcohol	Standard measure in ounces	Alcohol content
Beer	12 oz	5%
Malt liquor	8 oz	7%
Wine	5 oz	12%
80-proof distilled spirits or liquor	1.5 oz	40%

Source: Centers for Disease Control and Prevention. Fact sheets: alcohol use and health. 2012. http://www.cdc.gov/alcohol/fact-sheets/alcohol-use.htm.

Basic liver functions: A quick review

The largest visceral organ in the body, the liver serves numerous functions critical to maintaining homeostasis.



Function	Manifestations of altered function
Production of bile salts	Malabsorption of fat and fat-soluble vitamins
Elimination of bilirubin	Elevation in serum bilirubin and jaundice
Metabolism of steroid hormones	
Sex hormones	Disturbances in gonadal function, including gynecomastia in the male
Glucocorticoids	Signs of increased cortisol levels (such as Cushing syndrome)
Aldosterone	Signs of hyperaldosteronism (for example, sodium retention and hypokalemia)
Metabolism of drugs	Decreased drug metabolism
Carbohydrate metabolism	Hypoglycemia may develop when glycogenolysis and gluconeogenesis are impaired
Stores glycogen and synthesizes glucose from amino acids, lactic acid, and glycerol	Abnormal glucose tolerance curve may occur because of impaired uptake and release of glucose by the liver
Fat metabolism	
Formation of lipoproteins	Impaired synthesis of lipoproteins
Conversion of carbohydrates and proteins to fat Synthesis, recycling, and elimination of cholesterol Formation of ketones from fatty acid	Altered cholesterol levels
Protein metabolism	
Deamination of proteins	
Formation of urea from ammonia	Elevated blood ammonia levels
Synthesis of plasma proteins	Decreased levels of plasma proteins, particularly albumin, which contributes to edema formation Decreased plasma binding of drugs because of decreased albumin production
Synthesis of clotting factors (fibrinogen, prothrombin, factors V, VII, IX, X)	Bleeding tendency
Storage of minerals and vitamins	Signs of deficiency of fat-soluble and other vitamins that are stored in the liver
Filtration of blood and removal of bacteria and particulate matter by Kupffer cells	Increased exposure of the body to colonic bacteria and other foreign matter

Source: Porth CM. *Essentials of Pathophysiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2011:730,732.

develop and impair breathing as well.[6,8]

Management of portal hypertension aims to prevent gastroesophageal varices and subsequent hemorrhage using endoscopic procedures such as sclerotherapy or variceal ligation.[9] Antihypertensive drugs, diuretics, and a low-sodium diet may also be prescribed. Nursing management includes assessment for GI bleeding and emergent care as described below.

• **Varices.** Defined as veins that are permanently elongated and dilated from portal hypertension, varices are most common in the GI tract, specifically in the esophagus and rectum, and the abdomen. Because of their thin walls, esophageal varices are especially prone to rupture.[6]

An estimated 35% to 80% of patients diagnosed with cirrhosis will present with esophageal varices.[8,10] One study found that these patients have a 6% risk of a variceal bleed within the first year of being diagnosed and a 1-year mortality of 20%.[11]

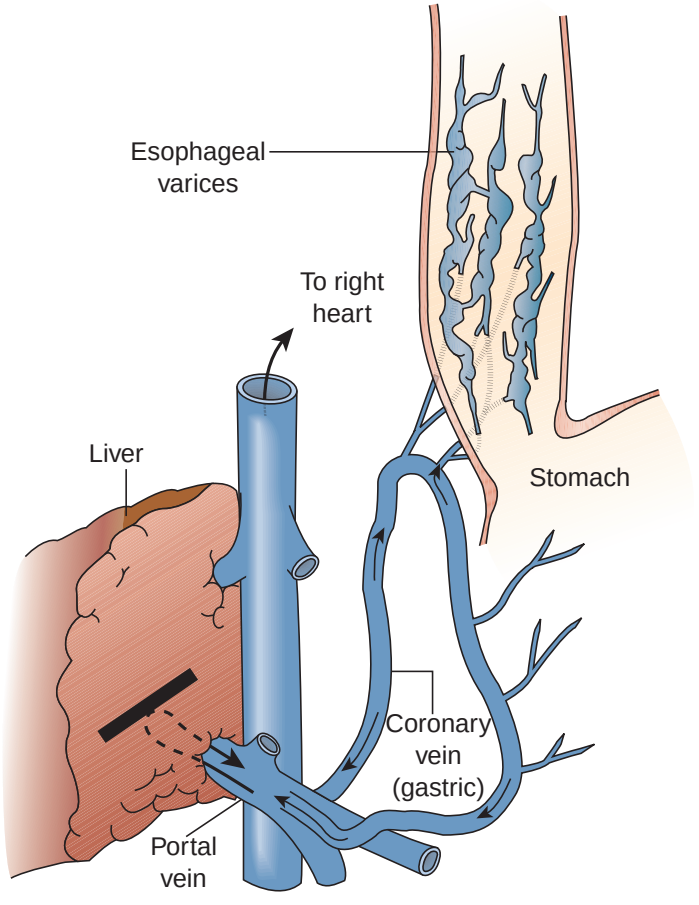
As blood circumvents the portal circulation, rising pressure dilates smaller veins within the GI tract that are also at risk for rupture under hypertensive pressures. Hemorrhage is complicated by a decrease in clotting factors as hepatic function declines.

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.[12] However, propranolol may be contraindicated in patients with refractory ascites. Closely monitor BP and renal function in patients with ascites taking propranolol or another beta-blocker.[13]

Vasoactive medications such as octreotide, somatostatin, and vasopressin may also be prescribed to treat acute variceal hemorrhage by reducing portal blood flow.

Understanding 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.



Source: Porth CM. *Essentials of Pathophysiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams and Wilkins; 2011:747-748.

Endoscopic variceal ligation, which involves placing small elastic bands around varices in the esophagus, or endoscopic sclerotherapy may be performed to prevent rupture or to stop bleeding.[13,14]

Patients with hemorrhage refractory to pharmacologic therapy and endoscopic procedures may benefit from a transjugular intrahepatic portosystemic shunt (TIPS), which creates a low-resistance channel between the intrahepatic portion of the portal vein and the hepatic vein. This decompresses the portal vein and varices associated with portal hypertension.[15] With TIPS, blood from the GI tract bypasses the liver. Because this increases circulating toxins, the patient faces a greater risk for HE.[16]

• **Ascites**. An excess of peritoneal cavity fluid, ascites is caused by portal hypertension, systemic vasodilation, hypoalbuminemia, and renal dysfunction.[17] The increase in collateral circulation to shunt the pressure in portal hypertension causes fluid to migrate into the abdomen, lower extremities, and scrotum or vulva. Signs and symptoms include progressive abdominal distension, weight gain, shortness of breath, early satiety, and dyspnea.[18] Ascites is the most common complication of cirrhosis.[13]

Nursing management is focused on increasing patient comfort; monitoring daily weights, intake and output, and serum electrolytes; and preparing the patient for a possible paracentesis. To promote effective breathing, position the patient with the head of the bed elevated or assist the patient out of bed into a chair. Medical management may include treating the underlying cause, avoiding medications known to be nephrotoxic, and oxygen therapy.[13] A diet low in sodium and high in calories, protein, and carbohydrates is indicated. Pharmacologic management includes diuretic therapy with furosemide and/or spironolactone, and antimicrobials if infection develops.[17,19]

• **Hepatic encephalopathy (HE)**. If a patient with ALD is confused, disoriented, or drowsy, HE is the likely etiology. HE is a brain dysfunction caused by hepatic dysfunction and/or portosystemic shunting.[16] The term covers a wide spectrum of neurologic disturbances that affect motor function, cognition, personality, and level of consciousness, including coma. The exact pathogenesis is unclear but it's linked to the accumulation of ammonia and other toxins. Normally, ammonia is cleared by the liver. When the liver is dysfunctional, however, toxic levels accumulate in the blood. Ammonia crosses the blood-brain barrier, leading to central nervous system toxicity.[20]

With progressive liver failure, altered mental status and coma are significant findings. HE occurs in 30% to 40% of patients with cirrhosis at some time during their clinical course.[16] Watch for subtle changes in mentation, slurred speech, agitation, and abnormal involuntary movements. Assess the patient for disorientation and asterixis (also known as flapping tremor), which are early indicators of HE.[16] (See *Assessing for asterixis*.)

In noncomatose patients with HE, hypertonia, hyperreflexia, and a positive Babinski sign can be observed. In comatose patients, deep tendon reflexes may be diminished.[16]

Medications such as lactulose, a synthetic disaccharide, and rifaximin, a nonabsorbable antibiotic, are prescribed to reduce serum ammonia levels. During this therapy, closely monitor the patient for dehydration and electrolyte abnormalities. As prescribed, titrate the lactulose dose to achieve two to three soft stool evacuations daily.[20] Be aware that patients may be incontinent of stool, and are often embarrassed and ashamed by the loss of control of bodily functions. Establishing a caring and trusting relationship will help to preserve their dignity.

Supportive care for patients with HE includes providing nutritional support and maintaining fluid and electrolyte balance. Ensure a safe environment and initiate fall prevention interventions for disoriented patients.[20]

• **Spontaneous bacterial peritonitis (SBP).** Patients with ALD are at high risk for infections stemming from numerous sources. The most dangerous, SBP, is an ascitic fluid infection that may develop when bacteria from the intestines are translocated to mesenteric lymph nodes. Under stress related to portal hypertension, lymphatic vessels carrying contaminated fluid may rupture, infecting ascitic fluid. Alternately, bacteria may infect ascitic fluid via movement from the mesenteric lymphatic system into the systemic circulation.[21] Suspect SBP if a patient with ALD presents with fever, abdominal pain or tenderness, altered mental status, or hypotension.[22]

SBP is diagnosed from analysis of fluid collected from the peritoneal cavity via paracentesis. As prescribed, begin empiric antibiotic therapy immediately after culture specimens are obtained.

• **Alcohol withdrawal syndrome (AWS) and delirium tremens (DT)**. Patients who've developed a biological dependence on alcohol are at risk for developing alcohol withdrawal syndrome (AWS), which is defined as the presence of two or more of the following signs or symptoms after cessation or reduction of alcohol use: diaphoresis; tachycardia; tremor; insomnia; nausea or vomiting; transient visual, tactile, or auditory hallucinations or illusions; psychomotor agitation; anxiety; and tonic-clonic seizures.[23] Onset of AWS can occur 6 to 48 hours after the last alcoholic drink as the blood alcohol level declines.[24]

Although rare, DT is the most serious complication of alcohol withdrawal. Characterized by severe autonomic hyperactivity and

cognitive dysfunction, it includes delirium, tachycardia, hypertension, low-grade fever, diaphoresis, agitation, and hallucinations.[23,25] Onset occurs from 72 to 96 hours after the last drink and can persist for up to 7 days.[24] Although DT occurs in only about 5% of patients with AWS, about 50% of them require admission to the ICU.[23,25,26]

Historically, mortality from DT was about 40%, but appropriate medical care improves the odds significantly. Today, with early recognition and prompt treatment, mortality has dropped from 5% to 20% to approximately 1% to 5%.[26]

Diagnosing ALD

Because no single lab marker definitively establishes alcohol as the etiology of liver disease, ALD is diagnosed on the basis of a patient history of excessive alcohol consumption and evidence of liver disease supported by diagnostic study results. 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). Due to altered synthesis of coagulation proteins, a prolonged PT has been shown to correlate with the degree of liver fibrosis.[27]

A liver panel, including serum ALT, AST, GGT, INR, and albumin levels, can be used to evaluate changes in ALD. AST and ALT are liver enzymes released into the bloodstream from damaged hepatocytes. ALD is suspected when the ratio of AST to ALT is greater than 2:1, as was the case with Ms. J.[1,3,28]

Diagnostic imaging is essential for diagnosing ALD and its complications. A chest X-ray can reveal pleural effusions and an abdominal ultrasound can help clinicians assess the patency of hepatic blood vessels and monitor for ascites. A contrast-enhanced computed tomography (CT) scan can show portal vein flow, thrombosis, and parenchymal distortion caused by cirrhosis.[27] Ultrasonography, CT, and magnetic resonance imaging can all be used to assess steatosis.[3,28]

The degree of liver disease can be evaluated with several assessment tools. The Child-Turcotte-Pugh (CTP) calculator (also called the Child-Pugh score) uses ascites, encephalopathy, total serum bilirubin, serum albumin, and PT or INR to grade the severity of cirrhosis.[29,30] Considered more accurate than the CTP calculator, the Model for End-Stage Liver Disease (MELD) quantifies liver dysfunction based on serum bilirubin, INR, and serum creatinine levels. MELD is currently used by the United Network for Organ Sharing to allocate priority for organ transplantation.[3,7,29]

Assessing and managing AWS

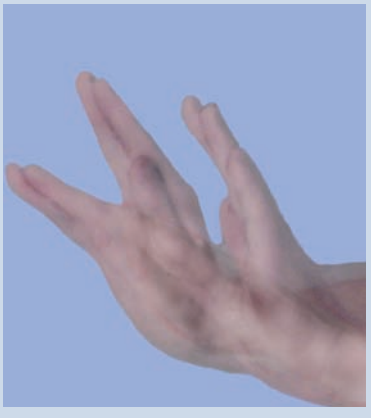
Nurses can significantly improve patient outcomes by assessing for AWS in patients with ALD. A valid and reliable tool to assess the severity and progression of AWS is the Clinical Institute Withdrawal Assessment for Alcohol, Revised (CIWA-Ar) scale.[25,31] Designed for patients who've been recently drinking and can respond to questions, the CIWA-Ar consists of 10 sections with questions that the nurse can ask the patient to make a rapid assessment at the bedside. The sections are nausea/vomiting, anxiety, paroxysmal sweats, tactile disturbances, visual disturbances, tremor, agitation, orientation and clouding of sensorium, auditory disturbances, and headache and fullness in head. The nurse assigns a score for each section depending on the patient's responses. Each section can be scored from 0 to 7 points except for orientation and clouding of sensorium, which is scored on a 0-to-4 scale. The highest possible score is 67 points, with higher scores indicating more severe withdrawal. The patient's score helps guide treatment decisions. For an example of the CIWA-AR form, see https://umem.org/files/uploads/1104212257_CIWA-Ar.pdf.

If medication is indicated, benzodiazepines are considered the first-line treatment for AWS because of their efficacy for reducing both withdrawal symptoms and the risk of seizures and/or DT.[32] Benzodiazepines should be administered as prescribed when the patient shows early signs of withdrawal to prevent symptom progression.[33] This underscores the need to screen patients for AWS using the CIWA-Ar scale early on.

Long-acting benzodiazepines such as chlordiazepoxide and diazepam may produce a smoother course of withdrawal and superior efficacy in prevention of delirium, but drugs with an intermediate half-life such as

Assessing for asterixis

To assess for HE, ask the patient to "stop traffic" by extending and cocking up both hands with fingers spread. Encourage the patient to maintain this position for 1 to 2 minutes. Watch for brief, sudden, nonrhythmic flexion of the hands and fingers, indicating asterixis. Besides liver disease, uremia and hypercapnia are possible causes of asterixis.



Source: Bickley LS. *Guide to Physical Examination and History Taking.* Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2009:704.

lorazepam or oxazepam are safer for older adults and those with preexisting liver or renal disease.[26,34]

Patients who abuse alcohol chronically are likely to be malnourished and can experience serious fluid and electrolyte imbalances during alcohol withdrawal. To address these problems, multivitamins supplemented with folate and fluid and electrolyte replacement therapy are administered along with benzodiazepines to manage AWS.[25] Monitor the patient for hypovolemia, hypokalemia, hypomagnesemia, and hypophosphatemia. As prescribed, administer glucose and thiamine to treat or prevent Wernicke encephalopathy. Because GI function is impaired in patients who chronically abuse alcohol, the I.V. route is indicated initially.[24]

Ms. J scored a 10 (mild withdrawal) on the CIWA-AR scale. Although medication is optional for patients with scores of 9 to 14, lorazepam was prescribed for Ms. J because of her preexisting hepatic disease. Patients with scores less than 8, indicating very mild AWS, normally don't require pharmacologic management.[25] A score above 20 is considered severe withdrawal.[31]

Nursing care for a patient with AWS is aimed at maintaining the safety of the patient and others. Keeping the patient calm can be challenging. If a benzodiazepine has been prescribed, routinely monitor for oversedation using a valid and reliable sedation scale. Maintain a quiet, low-stimulus environment with relaxing music in the background, if possible. Instruct staff to keep extraneous noise to a minimum. Interactions with the patient should be limited to simple explanations for care with few questions unless necessary. Directly confronting abnormal behavior may provoke the patient, increase agitation, and hinder cooperation.[35]

With the patient's consent, include the family in the plan of care. Reassure the patient and family that staff will provide compassionate and nonjudgmental care. Invite a family member who has a calming effect on the patient to sit at the bedside.

Assessing and managing nutritional deficits

Patients with advanced ALD should be screened for nutritional deficiencies. The patient's appetite may be poor due to early satiety caused by ascites, a salt-restricted unpalatable diet, or GI disturbances caused by reduced peristalsis or bile stasis. Studies have demonstrated that all patients with alcoholic hepatitis have some form of malnutrition because they derive 50% of their total caloric intake from alcohol.[36] Protein-calorie malnutrition is common in patients with ALD due to impaired caloric intake and increased catabolism.[3] Malabsorption syndrome and dietary insufficiency are also serious concerns for patients with ALD due to the liver's significant catabolic role in processing carbohydrates, lipids, vitamins, and other nutrients. Those with severe disease require aggressive supplementation and nutritional support.[1]

The evidence is mixed regarding the value of early enteral nutrition. Short-term effects of supplemental nutrition produce no improvement in mortality; however, long-term follow-up reveals significantly lower mortality and fewer infections.[3,37]

Pharmacologic management of ALD

Research-based evidence on the benefits of drug therapy for patients with ALD is equivocal.

• ***Corticosteroids.*** Thirteen trials over a 40-year period have yielded conflicting evidence regarding the effectiveness of corticosteroids in reducing mortality. Five studies suggest a decrease in short-term mortality in steroid-treated patients, while eight found no effect.[1]

The most recent systematic review of steroid therapy in liver disease failed to show statistically significant differences in mortality when patients were treated with corticosteroids.[38] However, in an earlier, placebo-controlled, double blind trial, Mathurin et al. found that corticosteroids improved short-term survival for patients with ALD.[39] Current recommendations for steroid therapy in ALD suggest a 2- to 4-week course of prednisolone to be tapered over 2 to 4 weeks or stopped, depending on the patient's clinical status.[1]

• ***Pentoxifylline***. This drug reduces blood viscosity and improves microcirculation. In ALD, it also exerts antioxidant effects and inhibits production of tumor necrosis factor. In several clinical trials, pentoxifylline increased 6-month survival and reduced the incidence of hepatorenal syndrome (renal dysfunction related to decreased perfusion with hepatic disease).[36,40,41] However, a randomized controlled trial found that although pentoxifylline doesn't improve survival, it does reduce complications associated with ALD.[42] A more recent large randomized controlled study of 270 patients with severe alcoholic hepatitis failed to result in an improved 6-month survival rate when combining prednisolone and pentoxifylline.[43] Guidelines from both the American Association for the Study of Liver Diseases and the American College of Gastroenterology recommend pentoxifylline daily for 4 weeks for patients with severe ALD.[1]

Medications to assist with abstinence

The most effective treatment for ALD is abstinence from alcohol.[44,45] Complete abstinence from alcohol is associated with a 60% 5-year survival in patients with cirrhosis.[46]

FDA-approved medications for treatment of alcoholism are disulfiram

(Antabuse), naltrexone, and acamprosate.[45] Given the hepatotoxicity of these anticraving drugs, however, they're recommended only for patients with early-stage ALD.[44]

Disulfiram causes extreme nausea and vomiting when patients consume alcohol after taking the medication. Due to these adverse reactions, disulfiram isn't effective at promoting abstinence and has been largely replaced by more tolerable agents.[1]

The opioid antagonist naltrexone has been noted to decrease cravings and reduce heavy drinking in chronic alcohol users.[45]

Acamprosate, which has properties that mimic the action of GABA, has been shown to reduce alcohol cravings and may reduce alcohol withdrawal symptoms.[1,47] Baclofen, a GABA receptor agonist, has also been shown to be safe and effective for reducing alcohol intake, promoting alcohol abstinence, and preventing relapse drinking.[44,47-49]

Pain management and analgesic use

Pain is common in patients with liver disease, but it's not well understood. Many disease variables contribute to pain in patients with liver disease; for example, patients with advanced liver disease may have ascites, causing abdominal and lower back pain, and gynecomastia leading to mastalgia.[13,50,51] One study found that pain and opioid use in patients with chronic liver disease were strongly associated with psychological symptoms.[51]

Pain management raises special safety concerns due to associated risks of harm from certain analgesics.[50,51] Nonsteroidal anti-inflammatory drugs (NSAIDs) are contraindicated in ALD because they can induce GI bleeding and renal failure, two complications already associated with decompensated liver function.[50,52] Aspirin alone or taken with other NSAIDS has been associated with variceal bleeding.[50]

Acetaminophen is considered a safer option than NSAIDs or opioids for pain management in patients with ALD. Although acetaminophen is metabolized by the liver, it can be administered in low doses (less than 2 g/day), provided patients abstain from alcohol.[50,52] (The recommended maximum dosage is 3 g/day for those with normal liver function.)

Opioid analgesics should be used with caution because these medications generally are metabolized in the liver. If an opioid is prescribed, monitor the patient's respiratory and cognitive status regularly, and assess pain using a validated pain intensity rating scale. Neuropathic pain, common in ALD, is often treated with tricyclic antidepressants, antiepileptic drugs, GABA analogues, and lidocaine patches.[50,52]

Screening for alcohol abuse with the CAGE questionnaire

Consisting of only four questions, this simple assessment tool is widely used to screen patients for alcohol abuse. Two yes answers indicate that the patient may have a problem with alcohol and needs further evaluation.

1. Have you ever felt you should Cut Down on your drinking?
2. Have people Annoyed you by criticizing your drinking?
3. Have you ever felt bad or Guilty about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (Eye-opener)?

Source: http://pubs.niaaa.nih.gov/publications/inscage.htm.

Patient screening and education

Many patients admitted to hospitals for various medical conditions have a problem with alcohol dependency. Nurses can play an important role in identifying patients with this condition.[33] Patients presenting with clinical manifestations consistent with ALD should be screened for alcohol use with validated tools such as the CAGE Questionnaire, a widely used tool consisting of four questions referring to signs of alcohol dependency and abuse. (See *Screening for alcohol abuse with the CAGE questionnaire*.)

Assess how patients perceive alcohol use and their willingness to quit. Provide relevant, educational information regarding the impact of alcohol on the liver and its short- and long-term consequences. Teach patients and families to recognize early signs of deterioration in liver function, such as mood changes, mild confusion, changes in sleeping habits, musty/sweet odor of breath, drowsiness, and itchy skin. Provide resources for how and when to seek medical attention.

Absolute abstinence from alcohol is crucial for preventing disease progression and complications related to ALD.[45,46] Encourage patients with ALD to participate in counseling programs and peer support groups.[53] Although not all nurses are highly skilled at counseling, studies have suggested that even "brief interventions" such as handing the patient a pamphlet on alcohol cessation programs can have a positive impact on reducing alcohol consumption.[36,54]

Research published by the National Institute on Alcohol Abuse and Alcoholism suggests that smoking damages the liver and can intensify liver disease when alcohol abuse is present.[55] Refer patients who smoke to a smoking cessation program or suggest speaking to a healthcare

provider about available products that aid in smoking cessation, such as nicotine patches, gum, or nasal spray.[56]

All patients with ALD should be vaccinated against hepatitis A and B viruses to prevent further liver damage. Similarly, they should receive annual influenza vaccines as well as the pneumococcal vaccine to prevent pneumonia, bacteremia, and meningitis. Ms. J's nurse reminds her that she'll need the influenza vaccination because she hasn't yet received it this season.

Providing compassionate nursing care

Patients with ALD may be stigmatized by healthcare providers. To preserve human dignity, nurses must remain objective when providing care and refrain from judging patients. To accomplish this, nurses can employ self-reflection strategies to explore and clarify values and to identify sources of potential bias that hinder patient care. Caring theories support accepting all patients for who they are, regardless of self-destructive behaviors such as substance abuse.[57]

Providing care for patients with ALD can be challenging for nurses. Because a patient's clinical status can deteriorate very rapidly, vigilant nursing assessments and interventions are required along with thorough patient and family education. Regardless of the circumstances surrounding a patient's ALD, the goal is to slow progression of the disease, manage signs and symptoms, and help the patient achieve the best-possible quality of life, however defined by the patient.

Ms. J's sobering moment came when her healthcare providers explained that if she didn't stop drinking, her disease would continue to progress and potentially lead to an early death. Ms. J's nurse used motivational interviewing, an evidence-based counseling technique, to help Ms. J explore and resolve her ambivalence about healthy lifestyle choices.[54] Following this interaction with her nurse, Ms. J attended a 12-step program meeting that was offered in the inpatient setting. This peer support group emphasized working toward abstinence from alcohol and increasing self-esteem. ■

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The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NURSE.0000454950.13699.e0

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