Currently, NAFLD is increasing because of the growing number of people who are obese. NAFLD occurs in 90% to 95% of severely obese children and adults. NASH occurs in 8% to 20% of obese persons with NAFLD.[3] NAFLD should be considered in patients with risk factors, including obesity, diabetes, hyperlipidemia, and hypertension (also known as *metabolic syndrome*).

Elevated liver function tests (ALT, AST) are often the first sign of NAFLD. Ultrasound and CT scans can be used to diagnose NAFLD. Definitive diagnosis is by a liver biopsy.

There is no currently approved medication for NAFLD.[10] The goal of therapy is weight loss of at least 10% of body weight, if overweight or obese and exercise. Reducing risk factors, including hyperlipidemia, hypertension, and diabetes, is important.

PROMOTING HEALTH EQUITY

Liver, Pancreas, and Gallbladder Disorders

Hepatitis
- Hepatitis C has a higher incidence among blacks than whites.
- Hepatitis B has a higher incidence among Asian Americans and Pacific Islanders.
- Deaths caused by hepatitis C are more common in blacks.

Liver and Pancreatic Cancer
- Primary liver cancer has a highest incidence in Hispanics, followed by blacks and whites.
- Pancreatic cancer occurs more often among blacks than whites.

Gallbladder Disease
- Whites and Native Americans have a higher incidence of gallbladder disease than blacks or Asian Americans

CIRRHOSIS

Cirrhosis is the end stage of liver disease. Cirrhosis is characterized by extensive degeneration and destruction of the liver cells. This results in the replacement of liver tissue by fibrosis (scar tissue) and regenerative nodules that occur from the liver's attempt to repair itself (Fig. 43.4). The development of cirrhosis usually happens after decades of chronic liver disease.

Etiology and Pathophysiology

Any chronic liver disease, including disease from excess alcohol use and NAFLD, can cause cirrhosis. The most common causes of cirrhosis in the United States are chronic hepatitis C infection and alcohol-induced liver disease. In patients with alcohol-induced liver disease, controversy exists as to the degree to which malnutrition adds to the damage caused by the alcohol itself. Some cases of nutrition-related cirrhosis have resulted from extreme dieting, malabsorption, and obesity. Environmental factors and genetic predisposition may lead to the development of cirrhosis, regardless of dietary or alcohol intake.

Around 20% of patients with chronic hepatitis C and 25% of those with chronic hepatitis B develop cirrhosis.[3] Chronic inflammation and cell necrosis from viral hepatitis can result in progressive fibrosis and cirrhosis. Chronic hepatitis combined with alcohol use has a synergistic effect in accelerating liver damage.

Biliary causes of cirrhosis include primary biliary cholangitis (PBC) and primary sclerosing cholangitis (PSC). Both are described earlier in this chapter.

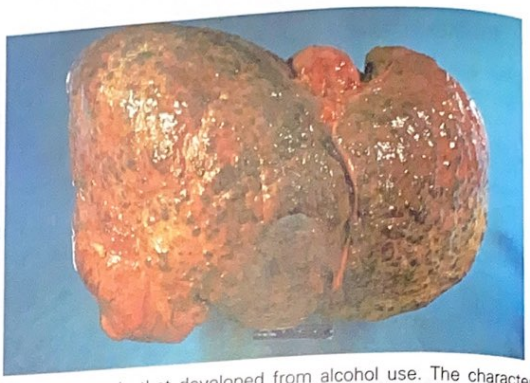

FIG. 43.4 Cirrhosis that developed from alcohol use. The characteristic diffuse nodularity of the surface is due to the combination of regeneration and scarring of the liver. (From Kumar V, Abbas AK, Aster JC, et al. *Robbins and Cotran pathologic basis of disease*, ed 8, Philadelphia, 2010, Saunders.)

Cardiac cirrhosis includes a spectrum of hepatic problems that result from long-standing, severe, right-sided heart failure. It causes hepatic venous congestion, parenchymal damage, necrosis of liver cells, and fibrosis over time. Treatment is aimed at managing the patient's underlying heart failure.

In cirrhosis, the liver cells try to regenerate, but the regenerative process is disorganized. This results in abnormal blood vessel and bile duct architecture. The overgrowth of new and fibrous connective tissue distorts the liver's normal lobular structure, resulting in lobules of irregular size and shape with impeded blood flow. Eventually, irregular and disorganized liver regeneration, poor cellular nutrition, and hypoxia (from inadequate blood flow and scar tissue) result in decreased liver function.

Clinical Manifestations

Early Manifestations. Patients may be unaware of their liver condition because there are few symptoms in early-stage disease. If a person does have symptoms, these may include fatigue or an enlarged liver. Blood tests may show normal liver function (compensated cirrhosis). The diagnosis of cirrhosis is often made later when a patient presents with symptoms of more advanced liver disease.

Late Manifestations. Late manifestations result from liver failure and portal hypertension (Fig. 43.5). Jaundice, peripheral edema, and ascites develop gradually. Other late manifestations include skin lesions, hematologic problems, endocrine problems, and peripheral neuropathies (Fig. 43.6). In the advanced stages, the liver becomes small and nodular. Liver function is dramatically impaired.

Jaundice. Jaundice results from decreased ability to conjugate and excrete bilirubin into the small intestines (Table 43.3). There is an overgrowth of connective tissue in the liver, which compresses the bile ducts and leads to an obstruction. This results in an increase in the bilirubin in the vascular system, and jaundice occurs. The jaundice may be minimal or severe, depending on the degree of liver damage.

Skin Lesions. Various skin manifestations often occur with cirrhosis. **Spider angiomas** (*telangiectasia* or *spider nevi*) are small, dilated blood vessels with a bright red center point and spiderlike branches. They occur on the nose, cheeks, upper trunk, neck, and shoulders. *Palmar erythema* (a red area that blanches with pressure) occurs on the palms of the hands. Both lesions are due to an increase in circulating estrogen due to the damaged liver's inability to metabolize steroid hormones.

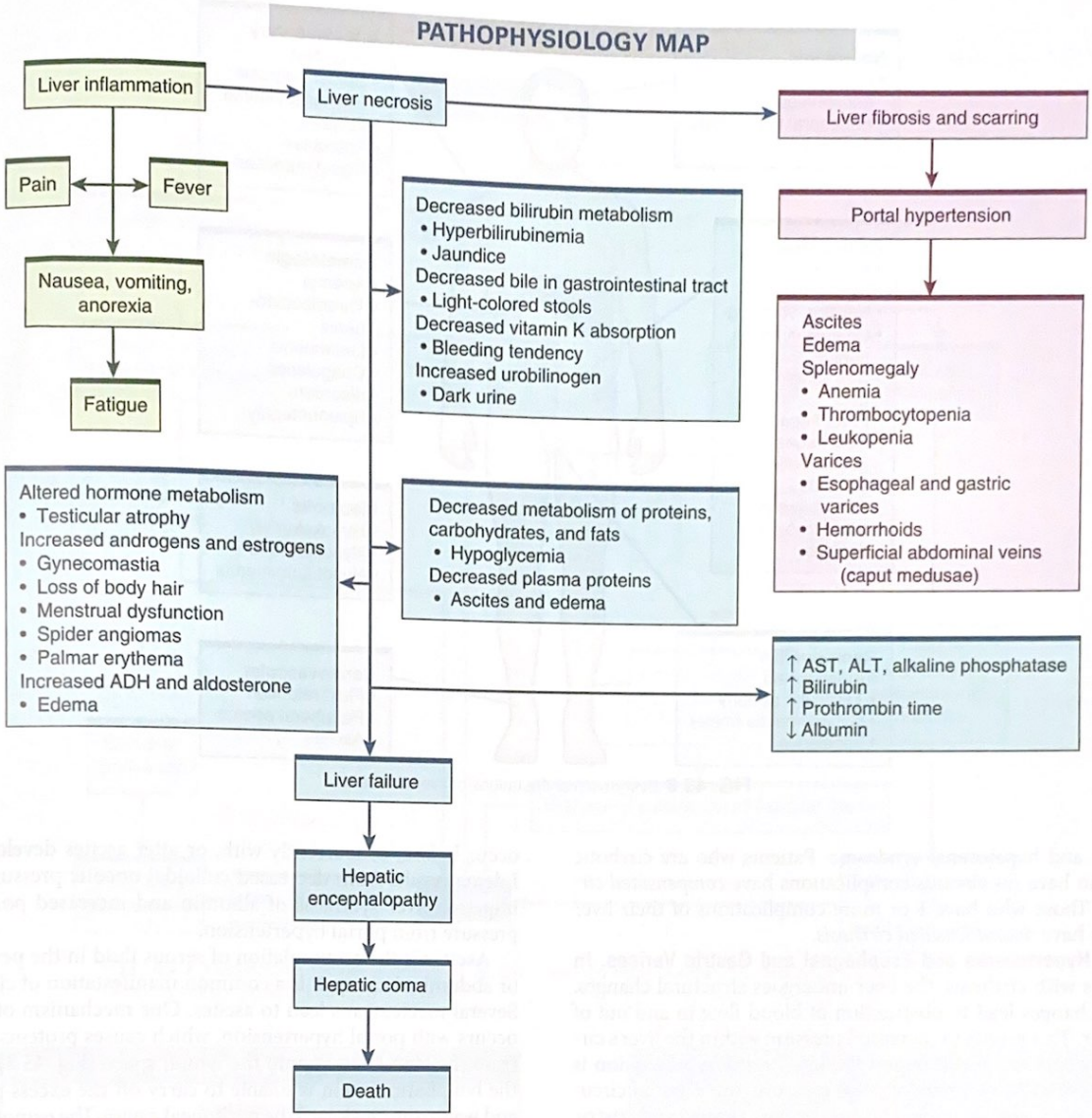

FIG. 43.5 Continuum of liver dysfunction in cirrhosis and resulting manifestations. (Adapted from Huether SE, McCance KL: *Understanding pathophysiology,* ed 5, St Louis, 2012, Mosby.)

Hematologic Problems. Hematologic problems include thrombocytopenia, leukopenia, anemia, and coagulation disorders. We think thrombocytopenia, leukopenia, and anemia are caused by the splenomegaly that results from backup of blood from the portal vein into the spleen (portal hypertension). Overactivity of the enlarged spleen results in increased removal of blood cells from circulation. Anemia can result from inadequate red blood cell (RBC) production and survival, poor diet, poor absorption of folic acid, and bleeding from varices.

The coagulation problems result from the liver's inability to make prothrombin and other factors essential for blood clotting. Manifestations of coagulation problems (bleeding tendencies) include epistaxis, purpura, petechiae, easy bruising, gingival bleeding, and heavy menstrual bleeding.

Endocrine Problems. The liver plays a vital role in the metabolism of hormones, such as estrogen and testosterone. In men with cirrhosis, gynecomastia (benign growth of the glandular tissue of the male breast), loss of axillary and pubic hair, testicular atrophy, and impotence with loss of libido may occur because of increased estrogen levels. Younger women with cirrhosis may develop amenorrhea, and older women may have vaginal bleeding. If the liver does not metabolize aldosterone properly, it can lead to hyperaldosteronism with sodium and water retention and potassium loss.

Peripheral Neuropathy. Peripheral neuropathy is a common finding in alcoholic cirrhosis. It is probably due to a dietary deficiency of thiamine, folic acid, and cobalamin. The neuropathy usually results in sensory and motor symptoms, but sensory symptoms may predominate.

Complications

Major complications of cirrhosis are portal hypertension, esophageal and gastric varices, peripheral edema, abdominal ascites, hepatic encephalopathy (mental status changes, including

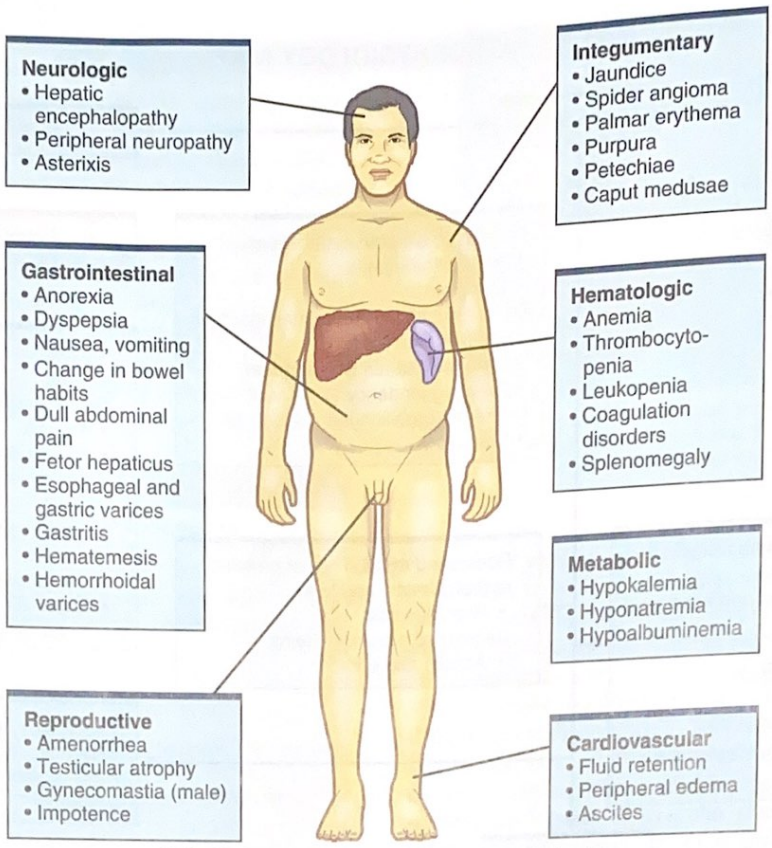

FIG. 43.6 Systemic manifestations of liver cirrhosis.

coma), and hepatorenal syndrome. Patients who are cirrhotic but who have no obvious complications have *compensated cirrhosis*. Those who have 1 or more complications of their liver disease have *decompensated cirrhosis*.

Portal Hypertension and Esophageal and Gastric Varices. In patients with cirrhosis, the liver undergoes structural changes. These changes lead to obstruction of blood flow in and out of the liver. This results in increased pressure within the liver's circulatory system (portal hypertension). **Portal hypertension** is characterized by increased venous pressure in the portal circulation, splenomegaly, large collateral veins, ascites, and gastric and esophageal varices.

As a way of reducing pressure, the body develops alternate circulatory pathways, referred to as *collateral circulation*. The collateral channels often form in the lower esophagus, anterior abdominal wall, parietal peritoneum, and rectum. Varicosities (distended veins) develop in areas where the collateral and systemic circulations communicate, resulting in esophageal and gastric varices, *caput medusae* (ring of varices around the umbilicus), and hemorrhoids.

Esophageal varices are a complex of tortuous, enlarged veins at the lower end of the esophagus. **Gastric varices** are found in the upper part of the stomach. These varices are fragile and do not tolerate high pressure, so they can bleed easily. Large varices are more likely to bleed. Esophageal varices can cause variceal hemorrhages with a 5-year mortality of 20%.[11] The patient may present with melena or hematemesis. Ruptured esophageal varices are the most life-threatening complication of cirrhosis and considered a medical emergency.

Peripheral Edema and Ascites. Peripheral edema occurs in the lower extremities and presacral area. Peripheral edema can occur before, concurrently with, or after ascites development. Edema results from decreased colloidal oncotic pressure from impaired liver synthesis of albumin and increased portacaval pressure from portal hypertension.

Ascites is the accumulation of serous fluid in the peritoneal or abdominal cavity. It is a common manifestation of cirrhosis. Several mechanisms lead to ascites. One mechanism of ascites occurs with portal hypertension, which causes proteins to shift from the blood vessels into the lymph space (Fig. 43.7). When the lymphatic system is unable to carry off the excess proteins and water, they leak into the peritoneal cavity. The osmotic pressure of the proteins pulls more fluid into the peritoneal cavity (Table 43.10).

A second mechanism of ascites formation is hypoalbuminemia resulting from the liver's decreased ability to synthesize albumin. The hypoalbuminemia results in decreased colloidal oncotic pressure.

A third mechanism of ascites is hyperaldosteronism, which occurs when the damaged hepatocytes metabolize aldosterone. The increased aldosterone level causes increased sodium reabsorption by the renal tubules. Sodium retention, combined with an increase in antidiuretic hormone in blood, leads to further water retention and edema. Edema decreases intravascular volume with decreased renal blood flow and glomerular filtration.

Ascites is manifested by abdominal distention with weight gain (Fig. 43.8). If the ascites is severe, the increase in abdominal pressure from the fluid accumulation may cause eversion of the umbilicus. Abdominal striae with distended abdominal wall veins may be present. Patients may have signs of dehydration (e.g., dry tongue and skin, sunken eyeballs, muscle weakness) and a decrease in urine output. Hypokalemia is common. It is

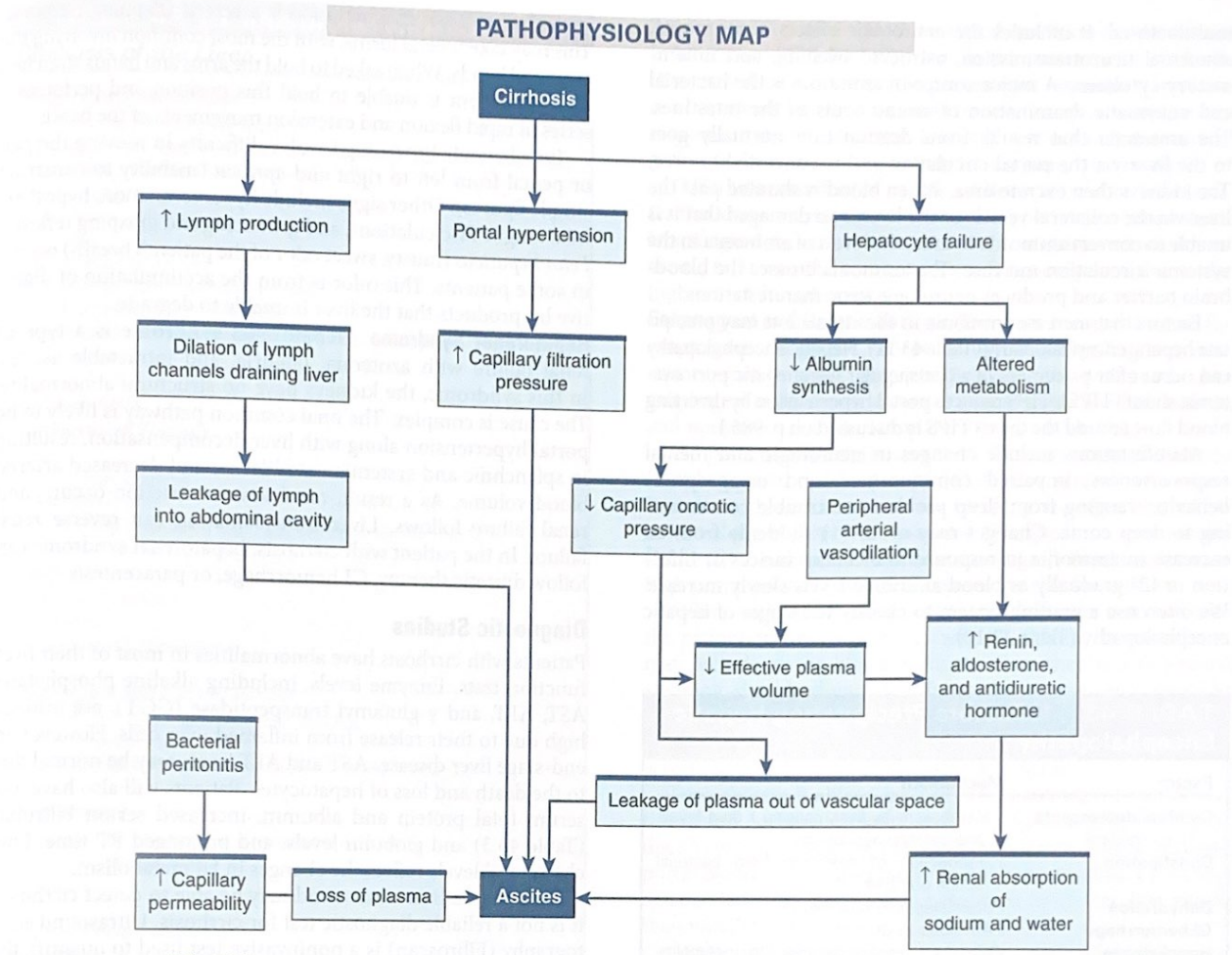

FIG. 43.7 Mechanisms for development of ascites. (Adapted from Huether SE, McCance KL: *Understanding pathophysiology,* ed 5, St Louis, 2012, Mosby.)

TABLE 43.10 Factors Involved in Ascites

Factor	Mechanism
Decreased serum colloidal oncotic pressure	Impaired liver synthesis of albumin Loss of albumin into peritoneal cavity
Hyperaldosteronism	↑ Aldosterone secretion stimulated by ↓ renal blood flow ↓ Liver catabolism of circulating aldosterone
Impaired water excretion	↑ Antidiuretic hormone stimulated by ↓ renal blood flow
Increased flow of hepatic lymph	Leaking of protein-rich lymph from surface of cirrhotic liver
Portal hypertension	↑ Resistance of blood flow through liver

FIG. 43.8 Gross ascites. (From Butcher GP: *Gastroenterology: An illustrated colour text,* London, 2004, Churchill Livingstone.)

due to an excessive loss of potassium caused by hyperaldosteronism. Low potassium levels can also result from diuretic therapy used to treat the ascites.

Because of decreased immune function associated with cirrhosis, patients with ascites are at risk for *spontaneous bacterial peritonitis* (SBP). SBP is a bacterial infection of the ascitic fluid. In SBP, bacteria normally found in the intestines move into the peritoneal space. The bacteria most often responsible for the infection are a gram-negative enteric pathogen, such as *Escherichia coli.* SBP is a common complication of hospitalized patients with cirrhosis and ascites. Worsening vasodilation contributes to the development of SBP.[11]

Hepatic Encephalopathy. **Hepatic encephalopathy** is a neuropsychiatric manifestation of liver disease. The pathogenesis is

multifactorial. It includes the neurotoxic effects of ammonia, abnormal neurotransmission, astrocyte swelling, and inflammatory cytokines. A major source of ammonia is the bacterial and enzymatic deamination of amino acids in the intestines. The ammonia that results from deamination normally goes to the liver via the portal circulation and is converted to urea. The kidneys then excrete urea. When blood is shunted past the liver via the collateral vessels or the liver is so damaged that it is unable to convert ammonia to urea, the levels of ammonia in the systemic circulation increase. The ammonia crosses the blood-brain barrier and produces neurologic toxic manifestations.

Factors that increase ammonia in the circulation may precipitate hepatic encephalopathy (Table 43.11). Hepatic encephalopathy can occur after placement of a transjugular intrahepatic portosystemic shunt (TIPS). TIPS reduces portal hypertension by diverting blood flow around the liver. (TIPS is discussed on p. 986.)

Manifestations include changes in neurologic and mental responsiveness; impaired consciousness; and inappropriate behavior, ranging from sleep problems to trouble concentrating to deep coma. Changes may occur (1) suddenly from an increase in ammonia in response to bleeding varices or infection or (2) gradually as blood ammonia levels slowly increase. We often use a grading system to classify the stages of hepatic encephalopathy (Table 43.12).

TABLE 43.11 Factors Precipitating Hepatic Encephalopathy

Factor	Mechanism
Cerebral depressants (e.g., opioids)	↓ Metabolism by liver, causing ↑ drug levels and cerebral depression
Constipation	↑ Production of ammonia from bacterial action on feces
Dehydration	Potentiates ammonia toxicity
GI hemorrhage	↑ Ammonia in GI tract
Hypokalemia	Potassium needed by brain to metabolize ammonia
Hypovolemia	↑ Blood ammonia because of hepatic hypoxia Impaired cerebral, hepatic, and renal function because of ↓ blood flow
↑ Metabolism	↑ Workload of liver
Infection	↑ Metabolic rate and cerebral sensitivity to toxins
Metabolic alkalosis	Facilitation of transport of ammonia across blood-brain barrier Increased renal production of ammonia
Paracentesis	Loss of sodium and potassium ions ↓ Blood volume
Uremia (renal failure)	Retention of nitrogenous metabolites

A characteristic manifestation is **asterixis** (flapping tremors). This may take several forms, with the most common involving the arms and hands. When asked to hold the arms and hands stretched out, the patient is unable to hold this position and performs a series of rapid flexion and extension movements of the hands.

Impairments in writing involve difficulty in moving the pen or pencil from left to right and *apraxia* (inability to construct simple figures). Other signs include hyperventilation, hypothermia, tongue fasciculations, and grimacing and grasping reflexes. *Fetor hepaticus* (musty, sweet odor of the patient's breath) occurs in some patients. This odor is from the accumulation of digestive by-products that the liver is unable to degrade.

Hepatorenal Syndrome. **Hepatorenal syndrome** is a type of renal failure with azotemia, oliguria, and intractable ascites. In this syndrome, the kidneys have no structural abnormality. The cause is complex. The final common pathway is likely to be portal hypertension along with liver decompensation, resulting in splanchnic and systemic vasodilation and decreased arterial blood volume. As a result, renal vasoconstriction occurs, and renal failure follows. Liver transplantation can reverse renal failure. In the patient with cirrhosis, hepatorenal syndrome can follow diuretic therapy, GI hemorrhage, or paracentesis.

Diagnostic Studies

Patients with cirrhosis have abnormalities in most of their liver function tests. Enzyme levels, including alkaline phosphatase, AST, ALT, and γ-glutamyl transpeptidase (GGT), are initially high due to their release from inflamed liver cells. However, in end-stage liver disease, AST and ALT levels may be normal due to the death and loss of hepatocytes. Patients will also have low serum total protein and albumin, increased serum bilirubin (Table 43.3) and globulin levels, and prolonged PT time. Low cholesterol levels reflect the changes in fat metabolism.

Although a liver ultrasound may be able to detect cirrhosis, it is not a reliable diagnostic test for cirrhosis. Ultrasound elastography (Fibroscan) is a noninvasive test used to quantify the degree of liver fibrosis. A liver biopsy, which may be done to identify liver cell changes, is the gold standard for a definitive diagnosis of cirrhosis.

Interprofessional Care

The goal of treatment is to slow the progression of cirrhosis and to prevent and treat any complications. Interprofessional care measures are listed in Table 43.13. Management of specific problems associated with cirrhosis is described next.

Ascites. Management of ascites focuses on sodium restriction, diuretics, and fluid removal.[12] Patients may need to limit sodium intake to 2 g/day. Very low sodium intake can result in reduced

TABLE 43.12 Grading Scale for Hepatic Encephalopathy

Grade	Level of Consciousness	Intellectual Function	Neurologic Findings
0	Normal to minimal change	Subtle to no change in personality, behavior, memory, concentration	Asterixis absent May have abnormal psychometric test
1	Lack of awareness, sleep disturbance	Short attention span, impaired computational skills, personality change, decrease in short-term memory, mild confusion, depression	Incoordination, asterixis may be absent
2	Lethargy, drowsiness	Disoriented to time, inappropriate behavior, deficits in executive function	Asterixis, abnormal reflexes
3	Somnolent, arousable	Disoriented to time, loss of meaningful conversation, marked confusion, incomprehensible speech	Asterixis, abnormal reflexes
4	Not arousable, comatose	Absent	Decerebrate May be responsive to painful stimuli

TABLE 43.13 Interprofessional Care

Cirrhosis of the Liver

Diagnostic Assessment

- History and physical examination
- Liver function tests (ALT, AST, alkaline phosphatase, bilirubin, γ-glutamyl transpeptidase [GGT])
- Serum albumin
- Serum electrolytes
- PT time
- Complete blood count
- Liver biopsy (percutaneous needle)
- Liver ultrasound (e.g., FibroScan)
- Upper endoscopy (esophagogastroduodenoscopy)
- CT scan, MRI

Management

Conservative Therapy

- Rest
- B-complex vitamins
- Avoiding alcohol
- Minimizing or avoiding aspirin, acetaminophen, and NSAIDs

Ascites

- Low-sodium diet
- Diuretics
- Paracentesis (if needed)

Esophageal and Gastric Varices

- Endoscopic band ligation or sclerotherapy
- Balloon tamponade
- Transjugular intrahepatic portosystemic shunt (TIPS)

Drug Therapy

- Nonselective β-blocker (e.g., propranolol [Inderal])
- octreotide (Sandostatin)
- vasopressin

Hepatic Encephalopathy

Drug Therapy

- Antibiotics (rifaximin [Xifaxan])
- lactulose

nutritional intake and malnutrition. The patient is usually not on restricted fluids unless severe ascites develops. When caring for patients with ascites, accurately monitor fluid and electrolyte balance. An albumin infusion may help maintain intravascular volume and adequate urine output by increasing plasma colloid oncotic pressure.

Diuretic therapy is an important part of management. Often a combination of drugs that work at multiple sites of the nephron is more effective than a single agent. Spironolactone (Aldactone) is an effective diuretic, even in patients with severe ascites. Spironolactone is also an antagonist of aldosterone and is potassium sparing. Other potassium-sparing diuretics include amiloride (Midamor) and triamterene (Dyrenium). A high-potency loop diuretic (e.g., furosemide [Lasix]), is often used in combination with a potassium-sparing drug.

Tolvaptan (Samsca), a vasopressin-receptor antagonist, can correct hyponatremia, a common problem in patients with cirrhosis. It causes an increase in water excretion, resulting in an increase in serum sodium concentration.

A **paracentesis** is a sterile procedure in which a catheter is used to withdraw fluid from the abdominal cavity. This procedure can diagnose a medical condition or relieve pain, pressure, or difficulty breathing. In the patient with cirrhosis, this procedure is done for the person with impaired respiration or abdominal discomfort caused by severe ascites who does not respond to diuretic therapy. It is only a temporary measure of palliation because the fluid tends to reaccumulate rapidly.[12]

TIPS (discussed later in this section) is used to treat ascites that does not respond to diuretics. A peritoneovenous shunt is a surgical procedure that provides continuous reinfusion of ascitic fluid into the venous system. It is rarely used due to the high rate of complications.

Esophageal and Gastric Varices. The main therapeutic goal for esophageal and gastric varices is to prevent bleeding and variceal rupture by reducing portal pressure. The patient who has esophageal and/or gastric varices should avoid alcohol, aspirin, and nonsteroidal antiinflammatory drugs (NSAIDs).

All patients with cirrhosis should have an upper endoscopy (esophagogastroduodenoscopy [EGD]) to screen for varices. Patients with varices at risk for bleeding are often started on a nonselective β-blocker (nadolol [Corgard] or propranolol [Inderal]) to reduce the risk of hemorrhage. β-Blockers decrease high portal pressure, which decreases the risk for rupture.

When variceal bleeding occurs, the first step is to stabilize the patient and manage the airway. IV therapy is started and may include giving blood products. Care then moves toward stopping the bleeding, identifying the source, and applying interventions to prevent further bleeding. Management that involves a combination of drug therapy and endoscopic therapy is more successful than either approach alone.

Drug therapy for bleeding varices may include the somatostatin analog octreotide (Sandostatin) or vasopressin. Both produce vasoconstriction of the splanchnic arterial bed, decrease portal blood flow, and decrease portal hypertension. Currently, octreotide is used more often because it has fewer side effects than vasopressin.

At the time of endoscopy, band ligation or sclerotherapy of varices may be used to prevent rebleeding. Endoscopic variceal ligation (EVL, or "banding") is done by placing a small rubber band (elastic O-ring) around the base of the *varix* (enlarged vein). Sclerotherapy involves injecting a sclerosing solution into the swollen veins through a needle placed through the endoscope.

Balloon tamponade is an option when endoscopy does not control acute esophageal or gastric variceal hemorrhage. Balloon tamponade controls the hemorrhage by mechanical compression of the varices. Several types of tubes are available. The Sengstaken-Blakemore tube has 2 balloons, gastric and esophageal, with 3 lumens: 1 for the gastric balloon, 1 for the esophageal balloon, and 1 for gastric aspiration. Two other types of balloons are the Minnesota tube (a modified Sengstaken-Blakemore tube with an esophageal suction port above the esophageal balloon) and the Linton-Nachlas tube.

SAFETY ALERT Balloon Tamponade

- Label each lumen to avoid confusion.
- Secure the tube to prevent movement of the tube that could result in occlusion of the airway.
- Deflate balloons for 5 minutes every 8 to 12 hr per agency policy to prevent tissue necrosis.

Supportive measures during an acute variceal bleed include giving fresh frozen plasma and packed RBCs, vitamin K, and

proton pump inhibitors (PPIs; e.g., pantoprazole). Lactulose and rifaximin (Xifaxan) may be given to prevent hepatic encephalopathy from breakdown of blood and the release of ammonia in the intestine. Antibiotics are given to prevent bacterial infection.

Because of the high incidence of recurrent bleeding with each bleeding episode, continued therapy is necessary. Long-term management of patients who have had an episode of bleeding includes nonselective β-blockers, repeated band ligation of the varices, and portosystemic shunts in patients who develop recurrent bleeding.

Shunting Procedures. Nonsurgical and surgical methods of shunting blood away from the varices are available. Shunting procedures tend to be done more after a second major bleeding episode than during an initial bleeding episode. *Transjugular intrahepatic portosystemic shunt (TIPS)* is a nonsurgical procedure in which a tract (shunt) between the systemic and portal venous systems is created to redirect portal blood flow. A catheter is placed in the jugular vein and then threaded through the superior and inferior vena cava to the hepatic vein. The wall of the hepatic vein is punctured, and the catheter is directed to the portal vein. Stents are positioned along the passageway, overlapping in the liver tissue and extending into both veins.

This procedure reduces portal venous pressure and decompresses the varices, thus controlling bleeding. TIPS does not interfere with a future liver transplantation. Limitations of TIPS include the increased risk for hepatic encephalopathy (toxin-containing blood bypasses the liver) and stenosis of the stent.

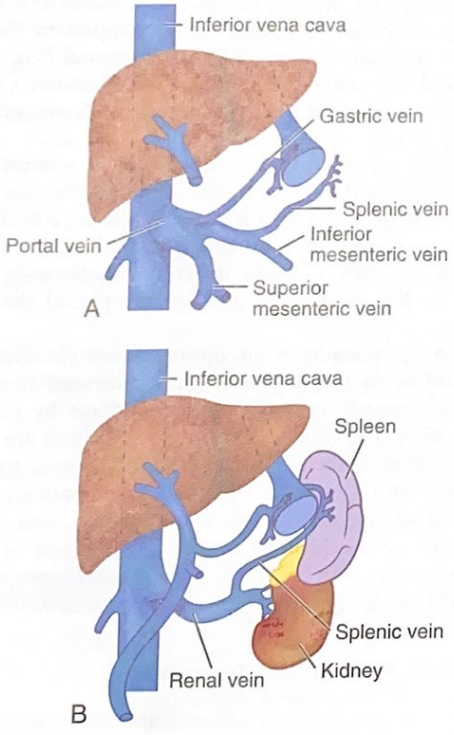

FIG. 43.9 Portosystemic shunts. A, Portacaval shunt. The portal vein is anastomosed to the inferior vena cava, diverting blood from the portal vein to the systemic circulation. B, Distal splenorenal shunt. The splenic vein is anastomosed to the renal vein. The portal venous flow stays intact while esophageal varices are selectively decompressed. (The short gastric veins are decompressed.) The spleen conducts blood from the high pressure of the esophageal and gastric varices to the low-pressure renal vein.

TIPS is contraindicated in patients with severe hepatic encephalopathy, liver cancer, severe hepatorenal syndrome, and portal vein thrombosis.

Various surgical shunting procedures can decrease portal hypertension by diverting some of the portal blood flow while allowing adequate liver perfusion. Currently, the surgical shunts most often used are the portacaval shunt and the distal splenorenal shunt (Fig. 43.9).

Hepatic Encephalopathy. The goal of management of hepatic encephalopathy is to reduce ammonia formation. Lactulose, a drug that traps ammonia in the gut, reduces ammonia formation in the intestines. We can give it orally, as an enema, or through a nasogastric (NG) tube. The drug's laxative effect expels the ammonia from the colon. Antibiotics, such as rifaximin, also may be given, especially in patients who do not respond to lactulose. Regular and frequent bowel movements are necessary to minimize the ammonia buildup, so use measures to prevent constipation.

Control of hepatic encephalopathy also involves treatment of precipitating causes (Table 43.10). This includes lowering dietary protein intake, preventing and controlling GI bleeds, and, in the case of a bleed, removing the blood promptly from the GI tract to decrease the protein accumulation in the gut.

Drug Therapy. There is no specific drug therapy for cirrhosis. However, several drugs are used to treat symptoms and complications of advanced liver disease (Table 43.14).

Nutritional Therapy. The diet for the patient who has cirrhosis without complications is high in calories (3000 cal/day). It is high in carbohydrate content with moderate to low levels of fat. Protein restriction may be needed for some patients right after a severe flare of symptoms (i.e., episodic hepatic encephalopathy). However, protein restriction is rarely needed in patients with cirrhosis and persistent hepatic encephalopathy. For many, malnutrition is a more serious clinical problem than hepatic encephalopathy.[3]

TABLE 43.14 Drug Therapy

Cirrhosis

Drug	Mechanism of Action
Diuretics	
furosemide (Lasix)	Acts on distal tubule and loop of Henle to ↓ reabsorption of sodium and water
spironolactone (Aldactone)	Blocks actions of aldosterone, potassium sparing
Other Therapy	
lactulose	Acidifies feces in bowel and traps ammonia, causing its elimination in feces
magnesium sulfate	Corrects hypomagnesemia that may occur with liver dysfunction
nadolol (Corgard) propranolol (Inderal)	Reduces portal venous pressure and esophageal variceal bleeding
neomycin sulfate rifaximin (Xifaxan)	↓ Bacterial flora, thus reducing ammonia formation
octreotide (Sandostatin) vasopressin	Hemostasis and control of bleeding in esophageal and gastric varices, constricts splanchnic arterial bed
PPIs (e.g., pantoprazole [Protonix])	↓ Gastric acidity
Vitamin K	Corrects clotting abnormalities from decreased vitamin K levels

A patient with alcoholic cirrhosis often has protein-calorie malnutrition. Oral nutritional supplements containing protein from branched-chain amino acids that are metabolized by the muscles may be needed. These supplements provide protein that the liver can more easily metabolize. Parenteral nutrition or EN are used with severe cases of malnutrition (see Chapter 39).

The patient with ascites and edema is placed on a low-sodium diet. The degree of sodium restriction depends on the patient's condition. Teach the patient and caregiver about the degree of restriction. Table salt is a well-known source of sodium. Other foods high in sodium include canned soups and vegetables, many frozen foods, salted snacks (e.g., potato chips), nuts, smoked meats and fish, crackers, breads, baking soda, olives, pickles, ketchup, and beer. Teach the patient to read labels for sodium content (see Fig. 34.5). Offer suggestions about how to make the diet more palatable. Seasonings, like garlic, parsley, onion, lemon juice, and spices may make food more appetizing. Collaborate with a dietitian about dietary strategies.

NURSING MANAGEMENT: CIRRHOSIS

Nursing Assessment

Subjective and objective data that should be obtained from a person with cirrhosis are outlined in Table 43.15.

Nursing Diagnoses

Nursing diagnoses for the patient with cirrhosis may include:

- Impaired nutritional status
- Ineffective tissue perfusion
- Activity intolerance
- Fluid imbalance

Additional information on nursing diagnoses and interventions for the patient with cirrhosis is presented in eNursing Care Plan 43.2 available on the website for this chapter.

Planning

The overall goals are that the patient with cirrhosis will (1) have relief of discomfort, (2) have minimal to no complications (ascites, esophageal varices, hepatic encephalopathy), and (3) return to as normal a lifestyle as possible.

Nursing Implementation

Health Promotion. Common risk factors for cirrhosis include alcohol use, malnutrition, viral hepatitis, biliary obstruction, obesity, and right-sided heart failure. Prevention and early treatment of cirrhosis focus on reducing or eliminating these risk factors. Urge patients to abstain from alcohol. Encourage those with chronic alcohol use to enroll in support programs that help patients maintain sobriety. (The treatment of alcohol use is discussed in Chapter 10.)

Adequate nutrition, especially for the person who uses alcohol and other people at risk for cirrhosis, is essential to promote normal liver regeneration. Identify and treat acute hepatitis early so that it does not progress to chronic hepatitis and cirrhosis. Bariatric surgery for morbidly obese persons reduces the incidence of NAFLD.

Acute Care. Nursing care for the patient with cirrhosis focuses on conserving the patient's strength while maintaining muscle strength and tone. When the patient needs complete bed rest, implement measures to prevent pneumonia, thromboembolic problems, and pressure injuries. Modify the activity and rest schedule according to signs of improvement (e.g., decreasing jaundice, improvement in liver function studies).

Anorexia, nausea and vomiting, pressure from ascites, and poor eating habits all interfere with adequate intake of nutrients. Oral hygiene before meals may improve the patient's taste sensation. Make between-meal snacks available so that the patient can eat them at times when food is best tolerated. Offer preferred foods whenever possible. Explain the reason for any dietary restrictions to the patient and caregiver.

Nursing assessment and care should include the patient's physical status. Is jaundice present? Where is it seen—sclera, skin, hard palate? What is the progression of jaundice? If the pruritis accompanies jaundice, use measures to relieve itching. Cholestyramine or hydroxyzine (Atarax) may help. Other

TABLE 43.15 Nursing Assessment

Cirrhosis

Subjective Data

Important Health Information

Past health history: Viral, toxic, or idiopathic hepatitis. Alcohol use, metabolic syndrome, chronic biliary obstruction and infection, severe right-sided heart failure

Medications: Adverse reaction to any medication. Use of anticoagulants, aspirin, NSAIDs, acetaminophen

Functional Health Patterns

Health perception–health management: Chronic alcohol use. Weakness, fatigue

Nutritional-metabolic: Anorexia, weight loss, dyspepsia, nausea and vomiting, gingival bleeding. Dry, yellow skin, bruising

Elimination: Dark urine, decreased urine output, light-colored or black stools, flatulence, change in bowel habits.

Cognitive-perceptual: Dull, right upper quadrant or epigastric pain. Numbness, tingling of extremities. Itching

Sexuality-reproductive: Impotence, amenorrhea

Objective Data

General

Fever, cachexia, wasting of extremities

Integumentary

Icteric sclera, jaundice, petechiae, ecchymoses, spider angiomas, palmar erythema, alopecia, loss of axillary and pubic hair, peripheral edema

Respiratory

Shallow, rapid respirations. Epistaxis

Gastrointestinal

Abdominal distention, ascites, distended abdominal wall veins, palpable liver and spleen, foul breath. Hematemesis. Black, tarry stools. Hemorrhoids

Neurologic

Altered mentation, asterixis

Reproductive

Gynecomastia, testicular atrophy, and impotence (men); loss of libido (men and women); amenorrhea or heavy menstrual bleeding (women)

Possible Diagnostic Findings

Anemia, thrombocytopenia; leukopenia. ↓ Serum albumin, potassium. Abnormal liver function studies. ↑ INR, ↓ platelets, ↑ ammonia, ↑ bilirubin levels. Abnormal abdominal ultrasound, CT, or MRI

TABLE 43.16 Nursing Management

Care of the Patient Undergoing Paracentesis

Preprocedure

- Have the patient void or insert an indwelling catheter.
- Obtain baseline vital signs and pulse oximetry. Weigh patient, inspect and palpate abdomen, and assess abdominal girth. Assess bladder for distention and determine last voiding.
- Assess baseline laboratory values (e.g., CBC, electrolytes, coagulation studies).
- Give any sedation or analgesia, if ordered.
- Teach patient to remain immobile during the procedure.
- Help the patient to a high-Fowler (sitting) position with feet on the floor.

Postprocedure

- Perform assessment and compare to baseline: vital signs, pulse oximetry, abdominal girth, abdominal pain. Note any signs of hypovolemia.
- Have the patient sit on the side of the bed or place in high-Fowler's position.
- Label and send the fluid for laboratory analysis.
- Check the dressing for bleeding and/or leakage of ascitic fluid.
- Give IV fluid and/or albumin as ordered.
- Measure any drainage and describe the collected fluid.
- Reweigh the patient and monitor intake and output.
- Maintain bedrest per agency protocol.

measures to relieve itching include baking soda or moisturizing bath oils (Alpha Keri), lotions containing calamine, antihistamines, soft or old linens, and control of the temperature (not too hot and not too cold). Keep the patient's nails short and clean. Teach patients to rub with their knuckles rather than scratch with their nails when they cannot resist scratching.

Note the color of urine and stools and assess for improvement or normalization of color. When jaundice is present, the urine is often dark brown, and the stool is gray or tan.

Edema and ascites require your assessment and intervention. Accurate calculation and recording of intake and output, daily weights, and measurements of extremities and abdominal girth help in the ongoing assessment of the location and extent of the edema. Mark the abdomen with a permanent marker so that you measure the girth at the same location each time.

Immediately before a paracentesis, have the patient void to prevent puncturing of the bladder during the procedure. Other nursing care associated with a paracentesis is outlined in Table 43.16.

Dyspnea is a frequent problem for the patient with severe ascites and can lead to pleural effusions. A semi-Fowler's or Fowler's position allows for maximal respiratory efficiency. Use pillows to support the arms and chest to increase the patient's comfort and ability to breathe.

Meticulous skin care is essential because the edematous tissues are prone to breakdown. Use an alternating-air pressure mattress or other special mattress. A turning schedule (minimum of every 2 hours) must be adhered to rigidly. Support the abdomen with pillows. If the abdomen is taut, cleanse it gently. The patient will tend to avoid moving because of abdominal discomfort and dyspnea. Range-of-motion exercises are helpful. Implement measures such as coughing and deep breathing to prevent respiratory problems. The lower extremities may be elevated. If scrotal edema is present, a scrotal support gives some comfort.

When the patient is taking diuretics, monitor serum sodium, potassium, chloride, and bicarbonate levels. Monitor renal function (blood urea nitrogen [BUN], serum creatinine) routinely and with any change in the diuretic dosage. Observe for signs of fluid and electrolyte imbalance, especially hypokalemia. Dysrhythmias, hypotension, tachycardia, and generalized muscle cramping, weakness, lethargy, and confusion may be present with hyponatremia from water excess.

Observe for and provide nursing care for any hematologic problems. These include bleeding tendencies, anemia, and increased susceptibility to infection.

Assess the patient's response to altered body image resulting from jaundice, spider angiomas, palmar erythema, ascites, and gynecomastia. The patient may have anxiety and embarrassment about these changes. Explain these phenomena and be a supportive listener. Provide nursing care with concern and encouragement to help the patient maintain his or her self-esteem.

CHECK YOUR PRACTICE

You are caring for a 69-yr-old male patient with advanced cirrhosis who just underwent banding for esophageal varices. The UAP tells you that the patient's BP is 80/60 mm Hg and he is hard to arouse.

- What is your concern?
- What would you do?

Bleeding Varices. If the patient has esophageal or gastric varices, observe for any signs of bleeding from the varices, such as hematemesis and melena. If hematemesis occurs, assess the patient for hemorrhage, call the HCP, and be ready to transfer the patient to the endoscopy suite and/or assist with equipment to control the bleeding. Maintain the patient's airway. Patients with bleeding varices are usually admitted to the intensive care unit (ICU).

Balloon tamponade is an option for patients who have bleeding that is unresponsive to band ligation or sclerotherapy. When balloon tamponade is used, explain to the patient and caregiver the use of the tube and how the balloon is inserted. Check the balloons for patency. It is usually the HCP's responsibility to insert the tube by either the nose or mouth. Then the gastric balloon is inflated with 250 mL of air, and the tube is retracted until resistance (lower esophageal sphincter) is felt. The tube is secured by placing a piece of sponge or foam rubber at the nostrils (nasal cuff). For continued bleeding, the esophageal balloon is then inflated. A sphygmomanometer is used to measure and maintain the desired pressure at 20 to 40 mm Hg. An x-ray verifies the balloon's position.

Nursing care includes monitoring for complications of rupture or erosion of the esophagus, regurgitation and aspiration of gastric contents, and occlusion of the airway by the balloon. If the gastric balloon breaks or is deflated, the esophageal balloon will slip upward, obstructing the airway and causing asphyxiation. If this happens, cut the tube or deflate the esophageal balloon. Keep scissors at the bedside. Minimize regurgitation by oral and pharyngeal suctioning and by keeping the patient in a semi-Fowler's position.

The patient is unable to swallow saliva because the inflated esophageal balloon occludes the esophagus. Encourage the patient to expectorate and provide an emesis basin and tissues. Frequent oral and nasal care offers relief from the taste of blood and irritation from mouth breathing.

ETHICAL/LEGAL DILEMMAS

Rationing

Situation

T.H., a 43-yr-old female patient with cirrhosis of the liver, is frequently admitted to the hospital. She has been told that her continued alcohol use will inevitably lead to her death. She now has GI bleeding and needs blood transfusions. She has a rare blood type that is hard to match. Should you ask for an ethics consultation?

Ethical/Legal Points for Consideration

- *Rationing,* or the controlled distribution of scarce resources, is a difficult ethical problem. The needs of an individual patient or group of patients are weighed against the needs of many patients, who may have a greater chance of recovery, and the availability of the necessary resources.
- Health interests can supersede the interests or rights of a person. For example, in anticipation of an anthrax attack, the government could confiscate all relevant antibiotics and restrict their use to treat the disease.
- Two individual rights that must be considered regarding rationing are the (1) constitutional right to privacy and (2) right to consent to or refuse medical procedures and therapy.
- The competent adult is the only person who may consent to or refuse treatment for his or her health care problems.
- If T.H. consents to a blood transfusion, an intervening party may be allowed to refuse that treatment only given substantial intervening circumstances and not as a threat to compel adherent future behavior.
- If involved parties cannot reach an agreement, legal intervention by way of a court order may become necessary.

Discussion Questions

1. Do you think patients with diseases that have a behavioral component, like substance use, deserve aggressive treatment?
2. Would you request an ethics committee consultation in T.H.'s case?

Hepatic Encephalopathy. Nursing care of the patient with hepatic encephalopathy focuses on maintaining a safe environment, sustaining life, and assisting with measures to reduce the formation of ammonia. Patients with hepatic encephalopathy may be confused and at risk for falls or other injuries. Assess the patient's (1) level of responsiveness (e.g., reflexes, pupillary reactions, orientation), (2) sensory and motor abnormalities (e.g., hyperreflexia, asterixis, motor coordination), (3) fluid and electrolyte imbalances, (4) acid-base imbalances, and (5) response to treatment measures.

Assess the neurologic status, including an exact description of the patient's behavior, at least every 2 hours. Plan your care of the patient based on the severity of the encephalopathy. In patients with altered levels of consciousness or whose airway may become compromised, have emergency equipment readily available. Any GI bleeding may worsen encephalopathy. Institute measures to prevent falls or injuries.

. Control factors known to precipitate encephalopathy as much as possible, including anything that may cause constipation (e.g., dehydration, opioid drugs). Measures to minimize constipation are important to reduce ammonia production. Give drugs, laxatives, and enemas as ordered. Encourage fluids, if not contraindicated. Assess the patient taking lactulose for diarrhea and excessive fluid and electrolyte losses.

Ambulatory Care. The patient with cirrhosis may be faced with a prolonged course and the chance of life-threatening problems and complications. The patient and caregiver need to understand the importance of continual health care and medical supervision.

TABLE 43.17 Patient & Caregiver Teaching

Cirrhosis

When teaching the patient and caregiver about management of cirrhosis, do the following:

1. Explain that cirrhosis is a chronic illness and requires continual health care.
2. Teach the symptoms of complications and when to seek medical attention to enable prompt treatment.
3. Teach the patient to avoid potentially hepatotoxic over-the-counter drugs, because the diseased liver is unable to metabolize them.
4. Encourage abstinence from alcohol because continued use increases the rate of liver disease progression and risk for liver complications.
5. Teach the patient with esophageal or gastric varices to avoid aspirin and NSAIDs to prevent hemorrhage.
6. Teach the patient with portal hypertension and varices that straining at stool, coughing, sneezing, and retching and vomiting may increase the risk for variceal hemorrhage.

Supportive measures include proper diet, rest, avoiding potentially hepatotoxic OTC drugs, such as acetaminophen in high doses, and abstaining from alcohol. Abstinence from alcohol is important and results in improvement in most patients. However, some patients find abstinence extremely hard and need emotional support. Explore your own attitude toward the patient whose cirrhosis is from chronic alcohol use. Always provide care without being condescending or judgmental. Treat patients with respect and concern for their well-being (see Chapter 10).

Cirrhosis is a chronic disease, and people can live many years with symptoms and complications from cirrhosis. The patient is affected not only physically but also psychologically, socially, and economically. Major lifestyle changes may be needed, especially if chronic alcohol use is the primary cause. Provide information about community support programs, such as Alcoholics Anonymous, for help with chronic alcohol use.

Teach the patient and caregiver about complications and when to seek medical attention (Table 43.17). Include instructions about adequate rest periods, how to detect early signs of complications, skin care, drug therapy side effects, observation for bleeding, and protection from infection.

Referral to a community or home health nurse may help ensure patient adherence to prescribed therapy. Home care for the patient with cirrhosis focuses on helping the patient with activities of daily living while maintaining the highest level of wellness possible.

◆ Evaluation

Expected outcomes are that the patient with cirrhosis will

- Maintain food and fluid intake adequate to meet nutritional needs
- Maintain skin integrity with relief of edema and itching
- Have normal fluid and electrolyte balance
- Acknowledge and get treatment for a substance use problem

ACUTE LIVER FAILURE

Acute liver failure, or *fulminant hepatic failure,* is a potentially life-threatening clinical syndrome.[3] It is characterized by a rapid onset of severe liver dysfunction in someone with no history of liver disease. It is often accompanied by hepatic encephalopathy.

The most common cause of acute liver failure is drugs, usually acetaminophen. Other drugs that can cause acute liver

failure include isoniazid, sulfa-containing drugs, and anticonvulsants. Drugs can cause hepatocyte damage by disrupting essential intracellular processes or causing an accumulation of toxic metabolic products. Other causes can include viral hepatitis, especially HBV. Hepatitis A is a less common cause.

Outcomes depend on the cause. Cerebral edema, cerebellar herniation, and brainstem compression are the most common causes of death. Treatment of cerebral edema is described in Chapter 56. Liver transplantation is associated with a significant survival benefit in patients with a low probability of spontaneous recovery.

Clinical Manifestations and Diagnostic Studies

Manifestations of acute liver failure include jaundice, coagulation abnormalities, and encephalopathy. Changes in cognitive function are often the first clinical sign. Patients are susceptible to a wide variety of complications, including cerebral edema, renal failure, hypoglycemia, metabolic acidosis, sepsis, and multiorgan failure.

Serum bilirubin is high, and the PT time is prolonged. Liver enzyme levels (AST, ALT) are often markedly increased. Other laboratory tests include blood chemistries (especially glucose, since hypoglycemia may be present and need correction), complete blood count (CBC), acetaminophen level, screening for other drugs and toxins, viral hepatitis serology (especially HAV and HBV), serum ceruloplasmin (enzyme synthesized in liver) and α_1-antitrypsin levels, iron levels, ammonia levels, and autoantibodies (ANAs and ASMAs).

CT or MRI can provide information about the liver size and contour, presence of ascites or tumors, and patency of the blood vessels.

◆ Interprofessional and Nursing Care

Since acute liver failure may progress rapidly, with hour-by-hour changes in consciousness, the patient is usually transferred to the ICU once the diagnosis is made. Planning for transfer to a transplant center should begin in patients with grade 1 or 2 encephalopathy because they may worsen rapidly. Early transfer is important because the risks involved with patient transport may increase or even prevent transfer if stage 3 or 4 encephalopathy develops (Table 43.12).

Renal failure is a frequent complication of liver failure. It may be due to dehydration, hepatorenal syndrome, or acute tubular necrosis. The frequency of renal failure may be even greater with acetaminophen overdose or other toxins with which direct renal toxicity occurs. Although few patients die of renal failure alone, it often increases the mortality risk and may worsen the prognosis. Protect renal function by maintaining adequate fluid balance, avoiding nephrotoxic agents (e.g., aminoglycosides, NSAIDs), and promptly identifying and treating infection.

Monitoring and management of hemodynamic and renal function, as well as glucose, electrolytes, and acid-base status, are critical. Conduct frequent neurologic evaluations for signs of increased intracranial pressure. Position the patient with the head elevated at 30 degrees. Avoid excessive patient stimulation. Maneuvers that cause straining or Valsalva-like movements may increase intracranial pressure (ICP). ICP monitoring is discussed in Chapter 56.

Assess the patient regularly for baseline level of consciousness and orientation and report any changes to the HCP. Avoid the use of any sedatives due to their effects on mental status. The effects can be confused with worsening encephalopathy. Only minimal doses of benzodiazepines should be used due to their delayed metabolism by the failing liver. Closely observe the patient to prevent injuries and pad bedrails to avoid injury from possible seizures. Monitor intake and output for renal function and provide good skin and oral care to avoid breakdown and infection.

Changes in level of consciousness may compromise nutritional intake. Many patients receive vitamin supplementation. Other factors, such as coagulation problems, may influence whether EN is started. An NG tube may be irritating to the nasal and esophageal mucosa and cause bleeding.

LIVER CANCER

Primary liver cancer starts in the liver. The most common types of liver cancer are hepatocellular carcinoma (HCC) (75% of cases) and intrahepatic cholangiocarcinoma (bile duct cancer). In 2018 there were about 40,710 cases of HCC and 28,920 HCC deaths in the United States. Worldwide liver cancer is the fifth most common cancer and second most common cause of cancer death.[13]

Liver cancer is the most common cause of death in patients with cirrhosis. Cirrhosis caused by hepatitis C is the most common cause of HCC in the United States, followed by NAFLD. About 2% of patient with cirrhosis develop liver cancer each year.

In primary liver cancer, lesions may be singular or numerous and nodular or diffusely spread over the entire liver. Some tumors infiltrate other organs, such as the gallbladder, or move into the peritoneum or the diaphragm. Primary liver cancer often metastasizes to the lung.

Metastatic cancer in the liver is more common than primary liver cancer (Fig. 43.10). The liver is a common site of metastatic

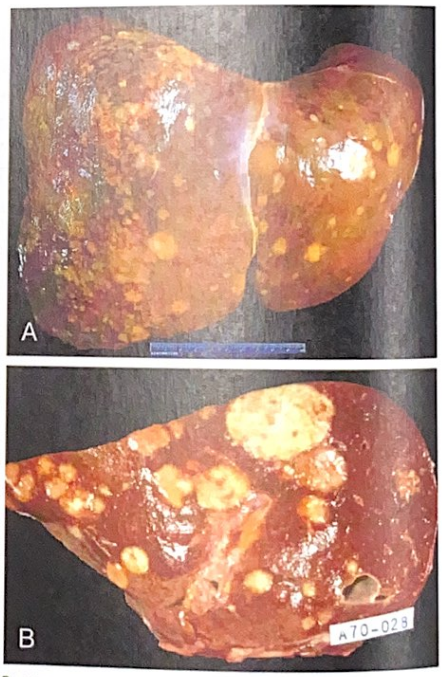

FIG. 43.10 Multiple hepatic metastases from a primary colon cancer. A, Gross specimen showing outside of liver. B, Liver section showing metastatic lesions. (*A*, From Kumar V, Abbas AK, Fausto N: *Robbins and Cotran pathologic basis of disease*, ed 7, Philadelphia, 2005, Saunders. *B*, From Kumar V, Abbas AK, Aster JC, et al: *Robbins and Cotran pathologic basis of disease*, ed 8, Philadelphia, 2010, Saunders.)

growth because of its high rate of blood flow and extensive capillary network. Cancer cells in other parts of the body are often carried to the liver via the portal circulation.

Clinical Manifestations and Diagnostic Studies

The manifestations of early liver cancer can be absent or subtle. They are often a result of the underlying cirrhosis rather than from the actual liver tumor(s). The patient may present with hepatomegaly, splenomegaly, fatigue, peripheral edema, ascites, and other complications from portal hypertension. In late stages, patients will often have fever/chills, jaundice, anorexia, weight loss, palpable mass, and right upper quadrant pain.

Diagnostic and screening for liver cancer are ultrasound, CT, and MRI. Recent advancements in MRI scanning have allowed for accurate diagnosis of liver cancers without the need for a percutaneous biopsy.[13] Sometimes, a biopsy may be done when the results of diagnostic imaging studies are inconclusive or tissue is needed to guide treatment. Risks of a biopsy include bleeding and potential tumor cell seeding along the needle tract. Therefore a biopsy is generally not done unless a diagnosis cannot be made by CT or MRI and clinical presentation. Serum α-fetoprotein (AFP) levels combined with ultrasound have a high rate of detection of early-stage HCC. (AFP is discussed in Chapter 15.)

Interprofessional and Nursing Care

Prevention of liver cancer focuses on identifying and treating chronic hepatitis B and C viral infections. Treatment of chronic alcohol use may lower the risk for liver cancer. Screening for at-risk patients (e.g., those with cirrhosis) usually involves a combination of serum AFP and CT, MRI, or ultrasound imaging of the liver.

Treatment of liver cancer depends primarily on the stage of cancer: number, size, and location of tumors; involvement of any blood vessels; patient age and overall health; and extent of underlying liver disease. Surgical liver resection (partial hepatectomy) offers the best chance for a cure. However, only about 15% of people have enough healthy liver tissue for this to be an option. The underlying cirrhosis and portal hypertension often compromise liver function and may cause liver failure after surgery. Furthermore, many patients are diagnosed at an advanced stage of cancer when surgery is not an option. For those patients who have early-stage liver cancer and impaired liver function, liver transplantation offers a good prognosis.

Nonsurgical therapies include percutaneous ablation, chemoembolization, radioembolization, and sorafenib (Nexavar) oral therapy.[13] In ablation, a thin needle is inserted into the core of the tumor. Then various substances can be injected (ethanol, acetic acid) and the temperature of the probe (radiofrequency, microwave, cryotherapy) can be altered to destroy the tumor. This procedure can be done percutaneously, laparoscopically, or through an open incision. It is typically limited by the number, size, and location of liver tumors. It is usually offered to patients with early-stage liver cancer. Although complications are not common, they can include infection, bleeding, dysrhythmias, and skin burn.

In patients with multinodular HCC or intermediate-stage liver cancer, embolization of the tumors is another intervention. There are 2 options typically used: transarterial chemoembolization (TACE) or transarterial radioembolization (TARE). TACE and TARE are minimally invasive procedures done by interventional radiologists. A catheter is placed via the femoral artery or radial artery and advanced to the arterial blood supply of the tumors in the liver. Either a chemotherapy drug (TACE) or radioactive beads (TARE) along with embolizing agents are then injected into the arteries of the tumor(s) region. TACE works by shutting off the blood supply to the tumors and exposing liver tumor cells to the chemotherapy agent. TARE destroys the tumor(s) by slowly releasing radioactive material directly to the site of the tumor. It can take up to 3 months for complete results.

In patients with advanced HCC, sorafenib (Nexavar) is typically the first-line treatment. It is a kinase inhibitor, a type of targeted therapy, that blocks certain proteins (kinases) that play a role in tumor growth and cancer progression (see Table 15.13). This drug has the potential to slow tumor progression and prolong life.

Nursing interventions focus on keeping the patient as comfortable as possible. Since these patients have the same problems as any patient with advanced liver disease, the nursing interventions discussed for cirrhosis of the liver apply to these patients (see p. 987).

Although the prognosis for patients with liver cancer is poor, it is improving with early screening and surveillance programs for those with chronic hepatitis and/or cirrhosis. The cancer often progresses rapidly, with patients having complications from the advancing cancer and declining liver function. Without treatment, death may occur within 6 to 12 months, most often from hepatic encephalopathy or massive blood loss from GI bleeding.

LIVER TRANSPLANTATION

Liver transplantation has become an option for many people with end-stage liver disease or localized HCC. Liver disease related to chronic viral hepatitis is the leading reason for liver transplantation. Other reasons include congenital biliary abnormalities (biliary atresia), inborn errors of metabolism, sclerosing cholangitis, acute liver failure, and chronic end-stage liver disease. In 2018, 11,514 patients were listed for a liver transplant in the United States with 8250 patients undergoing a transplant.[3]

Liver transplant candidates must go through a rigorous transplant evaluation prior to being placed on the transplant list. This is done to confirm the diagnosis of end-stage liver disease and to assess for other co-morbid conditions (e.g., cardiovascular disease, chronic kidney disease) that may affect the patient's surgical outcome. The evaluation includes physical examination, laboratory tests (CBC, liver function tests), cardiac and pulmonary evaluations, endoscopy, CT scan, and psychologic testing. Potential recipients receive counseling about cigarette smoking and alcohol abstinence. Contraindications for liver transplant include severe extrahepatic disease, advanced HCC or other cancer, ongoing drug or chronic alcohol use, and inability to understand or adhere with the rigorous posttransplant care.

Liver transplantation is done using both deceased (cadaver) and live donor livers. The live donor liver transplant was first developed for children whose parents wanted to serve as donors. Today, some liver transplant centers are performing live liver transplant procedures for adults. In this procedure, the living person donates a part of his or her liver to another. However, live liver donation poses potential risks to the donor, including biliary problems, hepatic artery thrombosis, wound infection, postoperative ileus, and pneumothorax.

growth because of its high rate of blood flow and extensive capillary network. Cancer cells in other parts of the body are often carried to the liver via the portal circulation.

Clinical Manifestations and Diagnostic Studies

The manifestations of early liver cancer can be absent or subtle. They are often a result of the underlying cirrhosis rather than from the actual liver tumor(s). The patient may present with hepatomegaly, splenomegaly, fatigue, peripheral edema, ascites, and other complications from portal hypertension. In late stages, patients will often have fever/chills, jaundice, anorexia, weight loss, palpable mass, and right upper quadrant pain.

Diagnostic and screening for liver cancer are ultrasound, CT, and MRI. Recent advancements in MRI scanning have allowed for accurate diagnosis of liver cancers without the need for a percutaneous biopsy.[13] Sometimes, a biopsy may be done when the results of diagnostic imaging studies are inconclusive or tissue is needed to guide treatment. Risks of a biopsy include bleeding and potential tumor cell seeding along the needle tract. Therefore a biopsy is generally not done unless a diagnosis cannot be made by CT or MRI and clinical presentation. Serum α-fetoprotein (AFP) levels combined with ultrasound have a high rate of detection of early-stage HCC. (AFP is discussed in Chapter 15.)

❖ Interprofessional and Nursing Care

Prevention of liver cancer focuses on identifying and treating chronic hepatitis B and C viral infections. Treatment of chronic alcohol use may lower the risk for liver cancer. Screening for at-risk patients (e.g., those with cirrhosis) usually involves a combination of serum AFP and CT, MRI, or ultrasound imaging of the liver.

Treatment of liver cancer depends primarily on the stage of cancer: number, size, and location of tumors; involvement of any blood vessels; patient age and overall health; and extent of underlying liver disease. Surgical liver resection (partial hepatectomy) offers the best chance for a cure. However, only about 15% of people have enough healthy liver tissue for this to be an option. The underlying cirrhosis and portal hypertension often compromise liver function and may cause liver failure after surgery. Furthermore, many patients are diagnosed at an advanced stage of cancer when surgery is not an option. For those patients who have early-stage liver cancer and impaired liver function, liver transplantation offers a good prognosis.

Nonsurgical therapies include percutaneous ablation, chemoembolization, radioembolization, and sorafenib (Nexavar) oral therapy.[13] In ablation, a thin needle is inserted into the core of the tumor. Then various substances can be injected (ethanol, acetic acid) and the temperature of the probe (radiofrequency, microwave, cryotherapy) can be altered to destroy the tumor. This procedure can be done percutaneously, laparoscopically, or through an open incision. It is typically limited by the number, size, and location of liver tumors. It is usually offered to patients with early-stage liver cancer. Although complications are not common, they can include infection, bleeding, dysrhythmias, and skin burn.

In patients with multinodular HCC or intermediate-stage liver cancer, embolization of the tumors is another intervention. There are 2 options typically used: transarterial chemoembolization (TACE) or transarterial radioembolization (TARE). TACE and TARE are minimally invasive procedures done by interventional radiologists. A catheter is placed via the femoral artery or radial artery and advanced to the arterial blood supply of the tumors in the liver. Either a chemotherapy drug (TACE) or radioactive beads (TARE) along with embolizing agents are then injected into the arteries of the tumor(s) region. TACE works by shutting off the blood supply to the tumors and exposing liver tumor cells to the chemotherapy agent. TARE destroys the tumor(s) by slowly releasing radioactive material directly to the site of the tumor. It can take up to 3 months for complete results.

In patients with advanced HCC, sorafenib (Nexavar) is typically the first-line treatment. It is a kinase inhibitor, a type of targeted therapy, that blocks certain proteins (kinases) that play a role in tumor growth and cancer progression (see Table 15.13). This drug has the potential to slow tumor progression and prolong life.

Nursing interventions focus on keeping the patient as comfortable as possible. Since these patients have the same problems as any patient with advanced liver disease, the nursing interventions discussed for cirrhosis of the liver apply to these patients (see p. 987).

Although the prognosis for patients with liver cancer is poor, it is improving with early screening and surveillance programs for those with chronic hepatitis and/or cirrhosis. The cancer often progresses rapidly, with patients having complications from the advancing cancer and declining liver function. Without treatment, death may occur within 6 to 12 months, most often from hepatic encephalopathy or massive blood loss from GI bleeding.

LIVER TRANSPLANTATION

Liver transplantation has become an option for many people with end-stage liver disease or localized HCC. Liver disease related to chronic viral hepatitis is the leading reason for liver transplantation. Other reasons include congenital biliary abnormalities (biliary atresia), inborn errors of metabolism, sclerosing cholangitis, acute liver failure, and chronic end-stage liver disease. In 2018, 11,514 patients were listed for a liver transplant in the United States with 8250 patients undergoing a transplant.[3]

Liver transplant candidates must go through a rigorous transplant evaluation prior to being placed on the transplant list. This is done to confirm the diagnosis of end-stage liver disease and to assess for other co-morbid conditions (e.g., cardiovascular disease, chronic kidney disease) that may affect the patient's surgical outcome. The evaluation includes physical examination, laboratory tests (CBC, liver function tests), cardiac and pulmonary evaluations, endoscopy, CT scan, and psychologic testing. Potential recipients receive counseling about cigarette smoking and alcohol abstinence. Contraindications for liver transplant include severe extrahepatic disease, advanced HCC or other cancer, ongoing drug or chronic alcohol use, and inability to understand or adhere with the rigorous posttransplant care.

Liver transplantation is done using both deceased (cadaver) and live donor livers. The live donor liver transplant was first developed for children whose parents wanted to serve as donors. Today, some liver transplant centers are performing live liver transplant procedures for adults. In this procedure, the living person donates a part of his or her liver to another. However, live liver donation poses potential risks to the donor, including biliary problems, hepatic artery thrombosis, wound infection, postoperative ileus, and pneumothorax.

Because of the limited number of donor livers, when a liver becomes available for transplant it may be divided into 2 parts (split liver transplant) and implanted into 2 recipients. The decision to use a split donor liver is based on the donor's size and health. The recipients of the split liver generally are smaller than the donor. The success rate associated with split liver transplantation is somewhat lower than that associated with whole organ transplantation.

Postoperative complications of liver transplant include bleeding, infection, and rejection. However, the liver is subject to a less aggressive immunologic attack than other organs, like the kidneys. Transplants and immunosuppressive therapy are discussed in Chapter 13.

Immunosuppressive therapy generally involves a combination of corticosteroids, a calcineurin inhibitor (cyclosporine or tacrolimus), and an antiproliferative agent (e.g., azathioprine). Tacrolimus is superior to cyclosporine in liver transplantation. Standard immunosuppressive regimens often change over the course of the recipient's life. Corticosteroid withdrawal may be done and is relatively safe to do in liver transplant recipients.

About 80% of patients live more than 5 years after liver transplant. Long-term survival depends on the cause of liver failure (e.g., localized HCC, chronic hepatitis B or C, biliary disease). Patients who have liver disease from hepatitis B or C often have reinfection of the transplanted liver. For patients with hepatitis B, treatment after surgery with IV HBIG and a nucleoside or nucleotide analog (used to treat HBV infection) has reduced the rates of reinfection of the transplanted liver. For patients with HCV, treatment with the new direct-acting antivirals (DAAs) that can cure HCV infection has provided the opportunity to use HCV-positive liver grafts. The need for liver transplant may decrease in the HCV population. Research is ongoing to decide if DAAs should be started before or after transplant.

The patient who had a liver transplant needs highly skilled nursing care, either in an ICU or other specialized unit. Postoperative nursing care includes assessing neurologic status; monitoring for signs of hemorrhage; preventing pulmonary complications; electrolyte levels, and urine output; and monitoring for manifestations of infection and rejection. Common respiratory problems are pneumonia, atelectasis, and pleural effusions. To prevent these complications, encourage the patient to cough, deep breathe, use incentive spirometry, and frequently reposition. Measure the drainage from the Jackson-Pratt drain, NG tube, and T tube and note the color and consistency of the drainage at regular intervals.

The first 2 months after surgery are critical for monitoring for infection. Causes of infection can be viral, fungal, or bacterial. Fever may be the only sign of infection. Adhering to the medication regimen can be hard, especially in the beginning. Emotional support and teaching for the patient and caregiver are essential to the success of the patient with a liver transplant.

Gerontologic Considerations: Liver Disease in the Older Adult

The incidence of liver disease increases with age. The liver's size and metabolic breakdown of drugs decrease, and hepatobiliary function is changed. The liver has a decreased capacity to respond to injury. This especially applies to regeneration after injury. Transplanted livers take longer to regenerate in the older adult compared with the younger adult.

Older adults are particularly vulnerable to drug-induced liver injury. This is due to several factors, including the increased use of multiple prescription and OTC drugs, which can lead to drug interactions and potential drug toxicity. Age-related decreases in liver function result in decreased drug metabolism and a decreased ability to recover from drug-induced injury.

A growing number of older adults have chronic hepatitis C and the resulting cirrhosis. Antibodies to HCV and elevated liver enzymes may be found during a routine health assessment in asymptomatic patients.

Lifetime health behaviors may influence the development of chronic liver disease in the older adult. Chronic alcohol u and obesity can contribute to cirrhosis, fatty liver inflammati (NASH), and liver failure. Because of many older adults' con comitant cardiovascular and lung diseases and possible anticoag ulant therapy, variceal bleeding can cause significant morbidity and mortality and needs immediate medical intervention. In the older adult with liver disease, hepatic encephalopathy is sometimes misdiagnosed as dementia and often overlooked.

Because older adults tend to have more co-morbid conditions, transplantation may have more risks for complications. Therefore older adults may not be good candidates for liver transplants.

DISORDERS OF THE PANCREAS

ACUTE PANCREATITIS

Acute pancreatitis is an acute inflammation of the pancreas. Spillage of pancreatic enzymes into surrounding pancreatic tissue causes autodigestion and severe pain. The degree of inflam mation varies from mild edema to severe hemorrhagic necros

Etiology and Pathophysiology

Many factors can cause injury to the pancreas. In the United States, the most common cause is gallbladder disease (gallstones), which is more common in women. The second most common cause is chronic alcohol use. This is more common in men.

Other less common causes include drug reactions, pancreatic cancer, and hypertriglyceridemia (serum levels over 1000 mg/dL).[14] Biliary sludge and microlithiasis, which is a mix of cholesterol crystals and calcium salts, can be present in patients with acute pancreatitis.

The most common pathogenic mechanism in acute pancreatitis is autodigestion of the pancreas (Fig. 43.11). The causative factors injure pancreatic cells or activate the pancreatic enzymes in the pancreas rather than in the intestine. This may be due to reflux of bile acids into the pancreatic ducts through an open or distended sphincter of Oddi. This reflux may be caused by blockage created by gallstones. Obstruction of pancreatic ducts results in pancreatic ischemia.

The exact mechanism by which chronic alcohol use predisposes a person to pancreatitis is not known. We think that alcohol increases the production of digestive enzymes in the pancreas.

The pathophysiologic involvement of acute pancreatitis is classified as either *mild pancreatitis* (also known as *edematous* or *interstitial pancreatitis*) or *severe pancreatitis* (also called *necrotizing pancreatitis*) (Fig. 43.12). In severe pancreatitis, about half the patients have permanent decreases in pancreatic endocrine and exocrine function. Patients with severe pancreatitis are at high risk for developing pancreatic necrosis, organ failure, and septic complications, resulting in an overall fatality rate of 5%.[14]

PATHOPHYSIOLOGY MAP

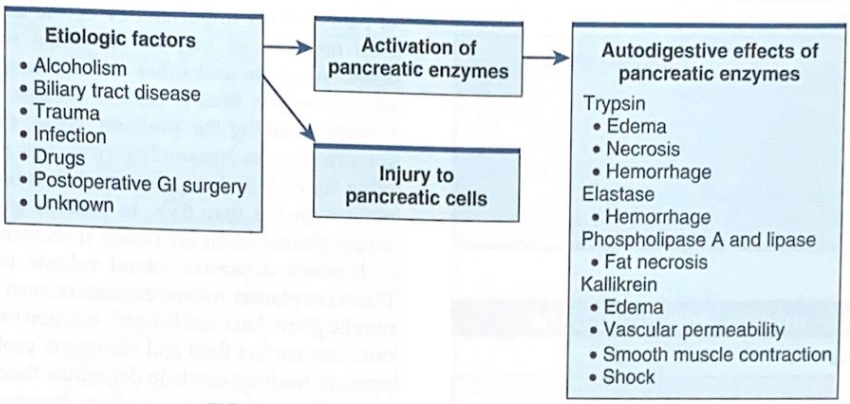

FIG. 43.11 Pathogenic process of acute pancreatitis.

FIG. 43.12 In acute pancreatitis, the pancreas appears edematous and is often hemorrhagic *(H)*. (From Stevens A, Lowe J: *Pathology: Illustrated review in colour,* ed 2, London, 2000, Mosby.)

Clinical Manifestations

Abdominal pain is the main manifestation of acute pancreatitis. The pain is due to distention of the pancreas, peritoneal irritation, and obstruction of the biliary tract. The pain is usually in the left upper quadrant, but it may be mid-epigastric. It often radiates to the back due to the retroperitoneal location of the pancreas. The pain has a sudden onset. It is described as severe, deep, piercing, and continuous or steady. Eating worsens the pain. It often starts when the patient is recumbent. Pain is not relieved by vomiting and may be accompanied by flushing, cyanosis, and dyspnea. The patient may assume various positions involving flexion of the spine to try to relieve the severe pain.

Other manifestations include nausea and vomiting, low-grade fever, leukocytosis, hypotension, tachycardia, and jaundice. Abdominal tenderness with muscle guarding is common.

Bowel sounds may be decreased or absent. Paralytic ileus may occur and causes marked abdominal distention. The lungs are often involved with crackles present. Intravascular damage from circulating trypsin (a proteolytic enzyme) may cause areas of cyanosis or greenish to yellow-brown discoloration of the abdominal wall. Other areas of ecchymoses are the flanks (*Grey Turner' spots* or *sign,* a bluish flank discoloration) and the periumbilical area (*Cullen's sign,* a bluish periumbilical discoloration). These result from seepage of bloodstained exudate from the pancreas and may occur in severe cases.

Shock may occur from hemorrhage into the pancreas, toxemia from the activated pancreatic enzymes, or hypovolemia due to fluid shift into the retroperitoneal space (massive fluid shifts).

Complications

The severity of acute pancreatitis depends on the extent of pancreatic destruction. Acute pancreatitis can be life threatening. Some patients recover completely, others have recurring attacks, and others develop chronic pancreatitis.

Two significant local complications of acute pancreatitis are pseudocyst and abscess. A *pancreatic pseudocyst* is an accumulation of fluid, pancreatic enzymes, tissue debris, and inflammatory exudates surrounded by a wall next to the pancreas. Manifestations are abdominal pain, palpable epigastric mass, nausea, vomiting, and anorexia. The serum amylase level is often high. CT, MRI, and endoscopic ultrasound (EUS) may detect a pseudocyst. The cysts usually resolve spontaneously within a few weeks but may perforate, causing peritonitis or rupture into the stomach or the duodenum. Treatment options include surgical drainage, percutaneous catheter placement and drainage, and endoscopic drainage.

When a pseudocyst gets infected, a *pancreatic abscess* results from extensive necrosis in the pancreas. It may rupture or perforate into adjacent organs. Manifestations of an abscess include upper abdominal pain, abdominal mass, high fever, and leukocytosis. Pancreatic abscesses need prompt surgical drainage to prevent sepsis.

The main systemic complications of acute pancreatitis are cardiovascular and pulmonary (pleural effusion, atelectasis, pneumonia, and acute respiratory distress syndrome [ARDS]). The pulmonary complications are due to the passage of exudate containing pancreatic enzymes from the peritoneal cavity through transdiaphragmatic lymph channels. Enzyme-induced inflammation of the diaphragm occurs, with the result being atelectasis caused by reduced diaphragm movement. Trypsin can activate prothrombin and plasminogen, increasing the patient's risk for intravascular thrombi, pulmonary emboli, and DIC. Hypotension can occur from fluid shifts and sepsis.

Tetany, which can be caused by hypocalcemia, is a sign of severe disease. It is due in part to the combining of calcium and fatty acids during fat necrosis. We do not understand the exact mechanisms of how or why hypocalcemia occurs. Patients with severe acute pancreatitis are at risk for abdominal compartment syndrome from intraabdominal hypertension and edema.

Diagnostic Studies

The primary diagnostic tests for acute pancreatitis are serum amylase and lipase (Table 43.18). The serum amylase level is

TABLE 43.18 Diagnostic Findings

Acute Pancreatitis

Laboratory Test	Abnormal Finding
Serum amylase	↑
Serum lipase	↑
Urinary amylase	↑
Blood glucose	↑
Serum calcium	↓
Serum triglycerides	↑

TABLE 43.19 Interprofessional Care

Acute Pancreatitis

Diagnostic Assessment
- History and physical examination
- Serum amylase and lipase
- Blood glucose
- Serum calcium
- Serum triglycerides
- Flat plate of the abdomen
- Abdominal ultrasound
- Endoscopic ultrasound (EUS)
- MRCP
- ERCP
- Contrast-enhanced CT of pancreas
- Chest x-ray

Management
- NPO with NG tube to suction
- Albumin (if shock present)
- IV calcium gluconate (10%) (if tetany present)
- Lactated Ringer's solution

Drug Therapy
- Pain medication (e.g., morphine)
- PPI (e.g., omeprazole [Prilosec])
- Antibiotics (if necrotizing pancreatitis)

usually high early and stays high for 24 to 72 hours. Serum lipase level, which is high in acute pancreatitis, is an important test because other disorders (e.g., mumps, cerebral trauma, renal transplantation) may increase serum amylase levels. Other serum findings include an increase in liver enzymes, triglycerides, glucose, and bilirubin and a decrease in calcium.

Diagnostic evaluation of acute pancreatitis is directed at determining the cause. An abdominal ultrasound, x-ray, or contrast-enhanced CT scan may identify pancreatic problems. CT scan is the best imaging test for pancreatitis and related complications, such as pseudocysts and abscesses. ERCP is an option (although ERCP can cause acute pancreatitis), along with EUS, magnetic resonance cholangiopancreatography (MRCP), and angiography. Chest x-rays may show atelectasis and pleural effusions.

Interprofessional Care

Goals of interprofessional care for acute pancreatitis include (1) relief of pain, (2) prevention or alleviation of shock, (3) reduction of pancreatic secretions, (4) correction of fluid and electrolyte imbalances, (5) prevention or treatment of infections, and (6) removal of the precipitating cause, if possible (Table 43.19).

Conservative Therapy. Treatment of acute pancreatitis is focused on supportive care, including aggressive hydration, pain management, management of metabolic complications, and minimization of pancreatic stimulation. Treatment and control of pain are very important. IV opioid analgesics may be given. Pain medications may be combined with an antispasmodic agent. Atropine and other anticholinergic drugs are avoided when paralytic ileus is present because they can decrease GI mobility, making the problem worse. Other drugs that relax smooth muscles (spasmolytics), such as nitroglycerin or papaverine, may be used. Supplemental O_2 is used to maintain O_2 saturation greater than 95%. In patients with severe pancreatitis, serum glucose levels are closely monitored for hyperglycemia.

If shock is present, blood volume replacements are used. Plasma or plasma volume expanders, such as dextran or albumin, may be given. Lactated Ringer's solution or other electrolyte solutions can correct fluid and electrolyte problems. Central venous pressure readings can help determine fluid replacement requirements. Vasoactive drugs, such as dopamine, may be needed to increase systemic vascular resistance in those with hypotension.

CHECK YOUR PRACTICE

You are a nurse working in the emergency department. Your patient is a 45-yr-old woman who reports acute abdominal pain in her left upper quadrant. She is diagnosed with acute pancreatitis. She tells you, "I've been waiting in this emergency room for 8 hours and I'm starving. Why can't I get something to eat? My pain is so bad; I think it's becoming worse because you won't give me food."
- How would you respond?
- What information and teaching would you give her?

It is important to reduce or suppress pancreatic enzymes to decrease stimulation of the pancreas and allow it to rest. This is achieved in several ways. First, the patient is NPO. Second, NG suction may be used to reduce vomiting and gastric distention and to prevent gastric acidic contents from entering the duodenum. Certain drugs are given to suppress gastric acid secretion (Table 43.20). With resolution of the pancreatitis, the patient resumes oral intake. For the patient with severe acute pancreatitis who does not resume oral intake, EN support may be started.

The inflamed and necrotic pancreatic tissue is a good medium for bacterial growth. In patients with acute necrotizing pancreatitis, infection is the leading cause of morbidity and mortality. Therefore it is important to prevent infections. Because many of the organisms come from the intestine, enteral feeding reduces the risk for necrotizing pancreatitis. Monitor the patient closely so that antibiotic therapy can be started early if necrosis and infection occur. Endoscopic- or CT-guided percutaneous aspiration with Gram stain and culture may be done.

Surgical Therapy. When the acute pancreatitis is related to gallstones, an urgent ERCP plus endoscopic *sphincterotomy* (severing of the muscle layers of the sphincter of Oddi) may be done. Laparoscopic cholecystectomy may follow ERCP to reduce the potential for recurrence. Surgical intervention may be needed when the diagnosis is uncertain and for patients who do not respond to conservative therapy.

Those with severe acute pancreatitis may need drainage of necrotic fluid collections. This is done surgically, under CT guidance, or endoscopically. Percutaneous drainage of a pseudocyst can be done, and a drainage tube left in place.

Drug Therapy. Several different drugs are used to prevent and treat problems associated with pancreatitis (Table 43.20). Currently, there are no drugs that cure pancreatitis.

TABLE 43.20 Drug Therapy
Acute and Chronic Pancreatitis

Drug	Mechanism of Action
Acute Pancreatitis	
Antacids	Neutralize gastric hydrochloric (HCl) acid secretion ↓ Production and secretion of pancreatic enzymes and bicarbonate
Antispasmodics (e.g., dicyclomine [Bentyl])	↓ Vagal stimulation, motility, pancreatic outflow (↓ volume and concentration of bicarbonate and enzyme secretion) Contraindicated in paralytic ileus
Carbonic anhydrase inhibitor (acetazolamide)	↓ Volume and bicarbonate concentration of pancreatic secretion
Morphine	Pain relief
PPIs (e.g., omeprazole [Prilosec])	↓ HCl acid secretion (HCl acid stimulates pancreatic activity)
Chronic Pancreatitis	
Insulin	Treat diabetes or hyperglycemia, if needed
Pancreatic enzyme products (pancrelipase [Pancrease, Zenpep, Creon, Viokace])	Replacement therapy for pancreatic enzymes

Nutritional Therapy. Initially, the patient with acute pancreatitis is on NPO status to reduce pancreatic secretion. Depending on the severity of the pancreatitis, EN is started. Because of infection risk, parenteral nutrition is reserved for patients who cannot tolerate EN (see Chapter 39). If IV lipids are given, monitor blood triglyceride levels. In cases of moderate to severe pancreatitis, the patient may need enteral feeding via a jejunal feeding tube.

When food is allowed, small, frequent feedings are given. The diet is high in carbohydrate content because that is the least stimulating to the exocrine part of the pancreas. Suspect intolerance to oral foods when the patient reports pain, has increasing abdominal girth, or has increased serum amylase and lipase levels. Supplemental fat-soluble vitamins may be given.

❖ NURSING MANAGEMENT: ACUTE PANCREATITIS

◆ Nursing Assessment

Subjective and objective data that should be obtained from a person with acute pancreatitis are outlined in Table 43.21.

◆ Nursing Diagnoses

Nursing diagnoses for the patient with acute pancreatitis may include:

- Acute pain
- Fluid imbalance
- Electrolyte imbalance
- Impaired nutritional intake

Additional information on nursing diagnoses and interventions for the patient with acute pancreatitis is presented in eNursing Care Plan 43.3 available on the website for this chapter.

◆ Planning

The overall goals are that the patient with acute pancreatitis will have (1) relief of pain, (2) normal fluid and electrolyte balance, (3) minimal to no complications, and (4) no recurrent attacks.

TABLE 43.21 Nursing Assessment
Acute Pancreatitis

Subjective Data

Important Health Information

Past health history: Biliary tract disease, alcohol use, abdominal trauma, duodenal ulcers, infection, metabolic disorders
Medications: Thiazides, NSAIDs
Surgery or other treatments: Surgical procedures on the pancreas, stomach, duodenum, or biliary tract. Endoscopic retrograde cholangiopancreatography (ERCP)

Functional Health Patterns

Health perception–health management: Chronic alcohol use, fatigue
Nutritional-metabolic: Nausea and vomiting, anorexia
Activity-exercise: Dyspnea
Cognitive-perceptual: Severe midepigastric or left upper quadrant pain that may radiate to the back, worsened by food and alcohol intake, unrelieved by vomiting

Objective Data

General

Restlessness, anxiety, low-grade fever

Integumentary

Flushing, diaphoresis, discoloration of abdomen and flanks, cyanosis, jaundice. Decreased skin turgor, dry mucous membranes

Respiratory

Tachypnea, basilar crackles

Cardiovascular

Tachycardia, hypotension

Gastrointestinal

Abdominal distention, tenderness, and muscle guarding. Decreased bowel sounds

Possible Diagnostic Findings

↑ Serum amylase and lipase, leukocytosis, hyperglycemia, hypocalcemia, abnormal ultrasound and CT scans of pancreas, abnormal ERCP or MRCP

◆ Nursing Implementation

◆ **Health Promotion.** The major factors involved in health promotion are (1) assessing the patient for predisposing and etiologic factors and (2) encouraging early treatment of these factors to prevent acute pancreatitis. Encourage the patient to cease alcohol intake, especially if they have had pancreatitis before. Recurrent attacks of pancreatitis may become milder or disappear if alcohol use is stopped. Encourage early diagnosis and treatment of biliary tract disease, such as gallstones.

◆ **Acute Care.** During the acute phase, it is important to monitor vital signs. Hypotension, fever, and tachypnea may compromise hemodynamic stability. Monitor the response to IV fluids. Closely monitor fluid and electrolyte balance. Frequent vomiting, along with gastric suction, may result in decreased chloride, sodium, and potassium levels.

Respiratory failure may develop in the patient with severe acute pancreatitis. Assess respiratory function (e.g., lung sounds, O_2 saturation levels). If ARDS develops, the patient may need intubation and mechanical ventilation support.

SAFETY ALERT Respiratory Distress in Acute Pancreatitis

- Assess for respiratory distress in the patient with severe acute pancreatitis.
- Listen to lung sounds and monitor O_2 saturation on a regular basis.

Because hypocalcemia can occur, observe for symptoms of tetany, including jerking, irritability, and muscular twitching. Numbness or tingling around the lips and in the fingers is an early sign of hypocalcemia. Assess the patient for a positive Chvostek's sign or Trousseau's sign (see Fig. 16.15). Give calcium gluconate (as ordered) to treat symptomatic hypocalcemia. Monitor serum magnesium levels since hypomagnesemia may develop.

Because abdominal pain is a primary symptom of pancreatitis, a major focus of your care is pain relief. Pain and restlessness can increase the metabolic rate and contribute to hemodynamic instability. Opioids may be used for pain relief. Assess and document the duration of pain relief. Comfortable positioning, frequent changes in position, and relief of nausea and vomiting help reduce the restlessness that usually accompanies the pain. Assuming positions that flex the trunk and draw the knees up to the abdomen may decrease pain. A side-lying position with the head elevated 45 degrees decreases tension on the abdomen and may help ease the pain.

For the patient who is on NPO status or has an NG tube, provide frequent oral and nasal care to relieve the dryness of the mouth and nose. Oral care is essential to prevent parotitis. If the patient is taking anticholinergics to decrease GI secretions, the mouth will be especially dry. If the patient is taking antacids to neutralize gastric acid secretion, they should be sipped slowly or inserted in the NG tube.

Observe for fever and other manifestations of infection in the patient with acute pancreatitis. Respiratory tract infections are common, which causes the patient to take shallow, guarded abdominal breaths. Measures to prevent respiratory tract infections include turning, coughing, deep breathing, and assuming a semi-Fowler's position.

Other important assessments are observation for signs of paralytic ileus, renal failure, and mental changes. Determine the blood glucose level to assess damage to the β cells of the islets of Langerhans in the pancreas.

If patients have surgery to drain necrotic fluid or treat a cyst, they may need special wound care for an anastomotic leak or a fistula. To prevent skin irritation, use skin barriers (e.g., Stomahesive, Karaya Paste), pouching, and drains. In addition to protecting the skin, pouching allows a more accurate determination of fluid and electrolyte losses and increases patient comfort. Sterile pouching systems are available. Consult with a clinical specialist or wound, ostomy, and continence nurse (WOCN).

Ambulatory Care. After acute pancreatitis, the patient may need home care follow-up. Because of loss of physical and muscle strength, physical therapy may be needed. Continued care to prevent infection and detect any complications is important. Counseling about abstinence from alcohol is important to prevent the patient from experiencing future attacks of acute pancreatitis and development of chronic pancreatitis. Because nicotine can stimulate the pancreas, smoking should be avoided.

Teach the patient and caregiver about the treatment plan, including the importance of taking the required medications and following the recommended diet. Dietary teaching should include fat restriction because fats stimulate the secretion of cholecystokinin, which then stimulates the pancreas. Encourage carbohydrates as they are less stimulating to the pancreas. Teach the patient to avoid crash and binge dieting because they can precipitate attacks.

Teach the patient and caregiver to recognize and report symptoms of infection, diabetes, or steatorrhea (foul-smelling, fatty stools). These changes indicate ongoing destruction of pancreatic tissue and pancreatic insufficiency. The patient may need exogenous enzyme supplementation.

◆ Evaluation

The expected outcomes are that the patient with acute pancreatitis will

- Have adequate pain control
- Maintain adequate fluid and electrolyte balance
- Be knowledgeable about the treatment plan to restore health
- Get help for alcohol use and smoking cessation (if needed)

CHRONIC PANCREATITIS

Chronic pancreatitis is a continuous, prolonged, inflammatory, and fibrosing process of the pancreas. The pancreas is progressively destroyed as it is replaced by fibrotic tissue. Strictures and calcifications may occur in the pancreas.

Etiology and Pathophysiology

Chronic pancreatitis can be due to chronic alcohol use; obstruction caused by gallstones, tumor, pseudocysts, or trauma; and systemic diseases (e.g., systemic lupus erythematosus), autoimmune pancreatitis, and cystic fibrosis. Some patients may not have an identifiable risk factor (idiopathic pancreatitis). Chronic pancreatitis may follow acute pancreatitis, but it may also occur in the absence of any history of an acute condition.

The most common cause of obstructive pancreatitis is inflammation of the sphincter of Oddi associated with gallstones. Cancer of the ampulla of Vater, duodenum, or pancreas can also cause this type of chronic pancreatitis.

The most common cause of nonobstructive pancreatitis (the most common type of chronic pancreatitis) is chronic alcohol use. There is inflammation and sclerosis, mainly in the head of the pancreas and around the pancreatic duct. In some people who drink alcohol, a genetic factor may predispose them to the direct toxic effect of the alcohol on the pancreas.

Clinical Manifestations

As with acute pancreatitis, a major manifestation of chronic pancreatitis is abdominal pain. The patient may have episodes of acute pain, but it usually is chronic (recurrent attacks at intervals of months or years). The attacks may become more frequent until they are almost constant, or they may decrease as pancreatic fibrosis develops. The pain is found in the same areas as in acute pancreatitis, but is usually described as a heavy, gnawing feeling or sometimes as burning and cramp-like. Food or antacids do not relieve the pain.

Other manifestations include symptoms of pancreatic insufficiency, including malabsorption with weight loss, constipation, mild jaundice with dark urine, steatorrhea, and diabetes. The steatorrhea may become severe, with voluminous, foul-smelling, fatty stools. Some abdominal tenderness may be present.

Chronic pancreatitis is associated with a variety of complications. These include pseudocyst formation, bile duct or duodenal obstruction, pancreatic ascites or pleural effusion, splenic vein thrombosis, pseudoaneurysms, and pancreatic cancer.

Diagnostic Studies

Confirming the diagnosis of chronic pancreatitis can be hard. The diagnosis is based on the patient's signs and symptoms, laboratory studies, and imaging. In chronic pancreatitis, serum amylase and lipase levels may be increased slightly or not at all, depending on the degree of pancreatic fibrosis. Serum bilirubin and alkaline phosphatase levels may be increased. There is usually mild leukocytosis and a high sedimentation rate.

ERCP can visualize the pancreatic and common bile ducts. Imaging studies, such as CT, MRI, MRCP, abdominal ultrasound, and EUS, can show a variety of changes, including calcifications, ductal dilation, pseudocysts, and enlargement of the pancreas.

Stool samples are examined for fecal fat content. Deficiencies of fat-soluble vitamins and cobalamin, glucose intolerance, and diabetes may occur in those with chronic pancreatitis. A secretin stimulation test can assess the degree of pancreatic dysfunction.

❖ Interprofessional and Nursing Care

When the patient with chronic pancreatitis has an acute attack, the therapy is identical to that for acute pancreatitis. At other times, the focus is on prevention of further attacks, relief of pain, and control of pancreatic exocrine and endocrine insufficiency. It sometimes takes frequent doses of analgesics (morphine, fentanyl patch [Duragesic]) to relieve the pain if dietary measures and enzyme replacement are not effective.

Diet, pancreatic enzyme replacement, and control of diabetes are ways to control the pancreatic insufficiency. Small, bland, frequent meals that are low in fat content are recommended to decrease pancreatic stimulation. Smoking is associated with accelerated progression of chronic pancreatitis. Teach the patient not to consume alcohol and caffeinated beverages. If the patient is dependent on alcohol, refer the patient to other resources as needed (see Chapter 10).

Pancreatic enzyme products, such as pancrelipase (Pancrease, Zenpep, Creon, Viokace), contain amylase, lipase, and trypsin. They are used to replace the deficient pancreatic enzymes. The enzymes are usually enteric coated to prevent their breakdown or inactivation by gastric acid. They are usually taken with meals and snacks. Teach the patient and caregiver to monitor stools for steatorrhea to help determine the effectiveness of the enzymes. Bile salts may be given to help with fat-soluble vitamin (A, D, E, and K) absorption and prevent further fat loss.

If diabetes develops, it is controlled with insulin (most often) or oral hypoglycemic agents. Teach the patient about testing blood glucose levels and drug therapy (see Chapter 48). While acid-neutralizing drugs (e.g., antacids) and acid-inhibiting drugs (e.g., H_2 receptor blockers, PPIs) may be given to control gastric acidity, they have little overall effect on patient outcomes. Antidepressants can reduce any neuropathic pain associated with chronic pancreatitis.

Treatment of chronic pancreatitis sometimes requires endoscopic therapy or surgery. When biliary disease is present or obstruction or pseudocyst develops, surgery may be needed. Surgical procedures can divert bile flow or relieve ductal obstruction. A choledochojejunostomy diverts bile around the ampulla of Vater, where there may be spasm or hypertrophy of the sphincter. In this procedure, the common bile duct is anastomosed into the jejunum. Another type of surgical diverting procedure is the Roux-en-Y pancreatojejunostomy, in which the pancreatic duct is opened and an anastomosis made with the jejunum. Pancreatic drainage procedures can relieve ductal obstruction and are often done with ERCP. Some patients may have an ERCP with sphincterotomy and/or stent placement at the site of obstruction. These patients need follow-up procedures, such as ERCP, to either exchange or remove the stent.

PANCREATIC CANCER

There is a high mortality rate of pancreatic cancer with the incidence almost equal to the mortality rates. The median age at diagnosis is around 71 years of age.[15] Most pancreatic tumors are adenocarcinomas that begin in the epithelium of the ductal system. More than half of the tumors occur in the head of the pancreas. As the tumor grows, the common bile duct becomes obstructed and obstructive jaundice develops. Tumors starting in the body or the tail often remain silent until their growth is advanced. Most cancers have metastasized at the time of diagnosis. The signs and symptoms of pancreatic cancer are similar to those of chronic pancreatitis. The prognosis of a patient with cancer of the pancreas is poor. Most patients die within 5 to 12 months of diagnosis. The 5-year survival rate is only 8%.[15]

Etiology and Pathophysiology

The cause of pancreatic cancer is unknown. Risk factors for pancreatic cancer include chronic pancreatitis, diabetes, age, cigarette smoking, family history of pancreatic cancer, high-fat diet, and exposure to chemicals, such as benzidine. Blacks have a higher incidence of pancreatic cancer than whites. The most established risk factor is cigarette smoking. Smokers are 2 to 3 times more likely to develop pancreatic cancer than nonsmokers. The risk is related to both the duration and number of cigarettes smoked.

Clinical Manifestations

Common manifestations include abdominal pain (dull, aching), anorexia, rapid and progressive weight loss, nausea, and jaundice. The most common manifestations with cancer of the head of the pancreas are pain, jaundice, and weight loss. In general, pain is common and is related to the cancer's location. The pain is often in the upper abdomen or left hypochondrium and often radiates to the back. It is often related to eating and occurs at night. Extreme, unrelenting pain is related to extension of the cancer into the retroperitoneal tissues and nerve plexuses. Pruritus may accompany obstructive jaundice. Weight loss is due to poor digestion and absorption caused by lack of digestive enzymes from the pancreas.

Diagnostic Studies

Abdominal ultrasound or EUS, spiral CT scan, ERCP, MRI, and MRCP are the most often used diagnostic imaging techniques for pancreatic diseases, including cancer. EUS involves imaging the pancreas with the use of an endoscope positioned in the stomach and duodenum. EUS also allows for fine-needle aspiration of the tumor for pathologic examination. CT scan is often the first study and gives information on metastasis and vascular involvement of the tumor. ERCP allows visualization of the pancreatic duct and biliary system. With ERCP, pancreatic secretions and tissue can be obtained for biopsy and analysis of tumor markers. MRI, PET, PET/CT scans and MRCP may be done to confirm a cancer diagnosis and determine staging. They can also monitor progress and response to therapy.

...s. Respiratory tract infec...

...nifestations of infec...

...the patient to take shallow, guar...

...sures to prevent respiratory tract infe...

...g, coughing, deep breathing, and assuming...

...s position.

...important assessments are observation for signs of ...ytic ileus, renal failure, and mental changes. Determine the ...ood glucose level to assess damage to the β cells of the islets of Langerhans in the pancreas.

If patients have surgery to drain necrotic fluid or treat a cyst, they may need special wound care for an anastomotic leak or a fistula. To prevent skin irritation, use skin barriers (e.g., Stomahesive, Karaya Paste), pouching, and drains. In addition to protecting the skin, pouching allows a more accurate determination of fluid and electrolyte losses and increases patient comfort. Sterile pouching systems are available. Consult with a clinical specialist or wound, ostomy, and continence nurse (WOCN).

Ambulatory Care. After acute pancreatitis, the patient may need home care follow-up. Because of loss of physical and muscle strength, physical therapy may be needed. Continued care to prevent infection and detect any complications is important. Counseling about abstinence from alcohol is important to prevent the patient from experiencing future attacks of acute pancreatitis and development of chronic pancreatitis. Because nicotine can stimulate the pancreas, smoking should be avoided.

Teach the patient and caregiver about the treatment plan, including the importance of taking the required medications and following the recommended diet. Dietary teaching should include fat restriction because fats stimulate the secretion of cholecystokinin, which then stimulates the pancreas. Encourage

...

w...

dire...

Clinica...

As with ...

pancreatiti...

of acute pain, ...

vals of months ...

until they are al...

atic fibrosis develo...

acute pancreatitis, b...

feeling or sometimes a...

ids do not relieve the pa...

Other manifestations i...

ficiency, including malabsorp...

mild jaundice with dark urine...

steatorrhea may become severe, ...

ing, fatty stools. Some abdominal te...

Chronic pancreatitis is associated w...

cations. These include pseudocyst formati...

denal obstruction, pancreatic ascites or pleur...

vein thrombosis, pseudoaneurysms, and ...