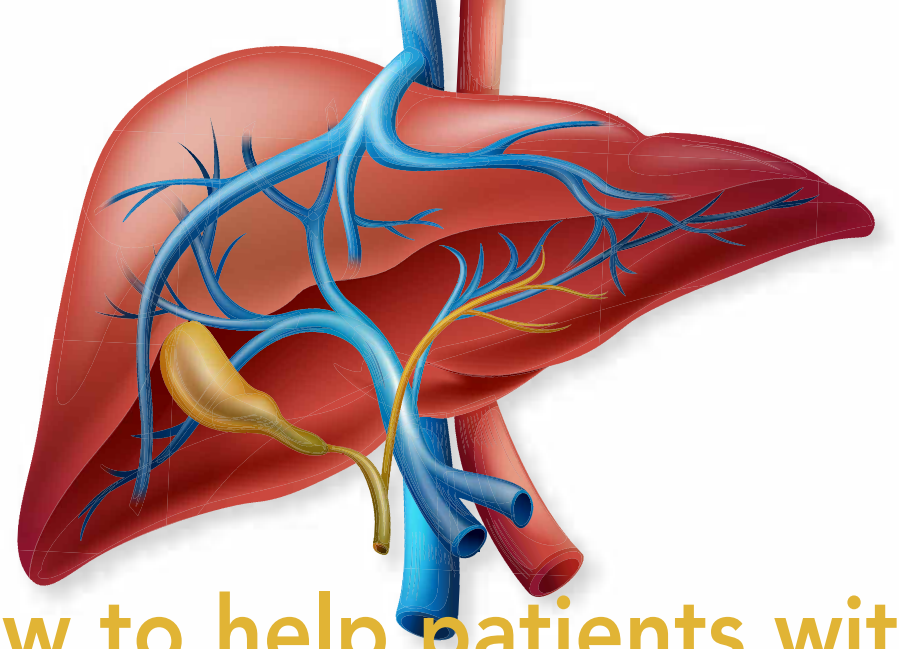

How to help patients with liver failure

Your astute assessment and management can help patients survive this life-threatening condition.

By Shari J. Lynn, MSN, RN

A LIFE-THREATENING condition, liver failure results from liver damage. In many cases, the damage is gradual and silent, causing few symptoms until the organ loses all of its functions.

Liver failure can be acute or take the form of chronic liver disease (CLD), which leads to irreversible end-stage liver disease (ESLD).

- *CLD* typically stems from alcohol abuse, hepatitis or, less often, hemochromatosis (excess iron stores) and malnutrition.
- *Acute liver failure (ALF)* is less common than CLD. In the developing world, it usually results from hepatitis B or E; in Europe and the United States, from acetaminophen overdose. Other causes include illicit drug use, herpes simplex virus, cytomegalovirus, Epstein-Barr virus, adenovirus, and parvovirus.

ALF is a medical emergency when it occurs in someone with no history of liver disease. Although sometimes reversible, it may warrant a liver transplant. In a patient who does have a history of liver disease, ALF is called acute-on-chronic liver failure. Nonalcoholic steatohepatitis is a form of CLD caused by fat buildup in the liver; its exact cause is unknown.

ALF can be classified as hyperacute, acute, or subacute based on the patient's prognosis and time from onset of jaundice to encephalopathy. (See *Classifying ALF.*)

Both ALF and CLD can lead to multisystem organ failure—quickly with ALF and gradually with CLD. To understand the effects of a failing liver, you need to comprehend the mechanisms involved. (See *Understanding liver functions.*)

Assessment

ALF and CLD share many signs and symptoms, including:

- decreased appetite
- nausea
- jaundice
- fatigue
- asterixis (flapping tremor)
- peripheral edema
- coagulopathy
- dark urine
- muscle wasting
- bleeding esophageal varices
- cerebral edema
- sepsis
- hepatic encephalopathy.

Abnormal liver function test results include an increased serum alanine level and an aminotransferase level that's 10 to 100 times the upper limit of normal. Typically, serum bilirubin and prothrombin times are increased and the albumin level is decreased.

Make sure to assess patients for signs and symptoms of hepatic encephalopathy and note their progression. Keep in mind that increased confusion suggests hepatic encephalopathy and cerebral edema. (See *Grading hepatic encephalopathy*).

Treatment

Interventions depend on whether

the patient has ALF or CLD, as well as the specific dysfunctions present. (See *Common dysfunctions in ALF.*)

ALF treatment

Treatment of ALF focuses on correcting the cause. Patients require urgent treatment and monitoring, including serial blood and coagulation tests. Specific measures may vary with the systemic dysfunctions present. Expect invasive monitoring for patients with cardiovascular problems, such as hypotension and intravascular volume depletion, along with volume depletion correction, vasopressors, and inotropic support, if needed. Patients who've ingested acetaminophen require I.V. acetylcysteine.

To reduce the risk of aspiration pneumonitis, patients with a decreased level of consciousness are likely to be intubated. Those with metabolic and renal dysfunctions require fluid management and blood glucose normalization, in addition to possible renal replacement therapy (such as dialysis).

Encephalopathy necessitates treatment of fever and hyponatremia, as well as sepsis screening. Some patients may require transcranial ultrasonography and intracranial pressure (ICP) monitoring. High-grade encephalopathy warrants endotracheal intubation, with partial pressure of carbon dioxide maintained between 31 and 44 mm Hg and serum sodium between 145 and 150 mmol/L. Intracranial hypertension calls for osmotherapy, temperature control, and rescue therapy (such as indomethacin and thiopentone). Antibiotic prophylaxis may be given to patients at high sepsis risk.

Understanding liver functions

A highly vascular organ, the liver is perfused by 1,500 mL of blood per minute. About 75% of its blood supply comes from the portal vein and 25% from the hepatic artery. Portal-vein blood is rich in nutrients and toxins, as it comes from the GI tract and spleen.

The liver detoxifies substances in the body and produces endothelin and nitric oxide. Endothelin causes vasoconstriction; nitric oxide induces vasodilation. Working together, these two mediators regulate hepatic blood flow.

In liver disease, portal-vein pressure rises, resulting in portal hypertension and an imbalance of endothelin and nitric oxide. Excessive nitric oxide production leads to splanchnic blood-vessel dilation, causing blood to shunt away from major organs and increasing blood flow to the liver—in particular, the portal vein. This shunting reduces kidney perfusion, in turn activating the renin-angiotensin-aldosterone system (which regulates blood pressure) as the kidneys try to increase their own blood volume.

Aldosterone activation increases sodium absorption in the loop of Henle, causing water retention and increased blood volume. Excess nitric oxide leads to dilation of abdominal vessels; at the same time, blood pressure in the kidneys decreases. Sodium and water continue to be reabsorbed, causing circulatory volume to rise—to the point that fluid leaks from the intravascular space and overloads the lymphatic system. Eventually, this fluid leaks into the peritoneal cavity, resulting in ascites, a hallmark of liver failure.

In chronic liver disease, cirrhosis occurs as fibrotic tissue develops. The liver atrophies and shrinks as more scar tissue forms. These effects are irreversible.

Classifying ALF

Acute liver failure (ALF) can be classified as shown below. Keep in mind, though, that these classifications have limited usefulness in clinical practice because they don't necessarily indicate prognosis.

Classification	Time from jaundice to encephalopathy	Risk of cerebral edema
Hyperacute	Less than 7 days	Common (occurs in more than 70% of patients)
Acute	8-28 days	Common (occurs in more than 55% of patients)
Subacute	5-12 weeks	Low (occurs in fewer than 15% of patients))

CLD treatment

Treatment focuses on stopping additional liver damage and slowing disease progression, as well as managing symptoms. It includes a healthy, low-sodium diet; abstaining from alcohol and illicit drugs; and addressing medical problems as they occur. Ultimately, patients may need a liver transplant.

Liver transplantation

Patients ages 12 and older who progress to the point of needing a liver transplant are assessed using the Model for End-Stage Liver Disease (MELD) scoring system; those younger than age 12 are assessed using the Pediatric End-Stage Liver Disease (PELD) system. Blood tests used to determine MELD scores are serum bilirubin, serum creatinine, serum sodium, and international normalized ratio (INR). Blood tests used to determine PELD scores are bilirubin, albumin, and INR. The MELD score also considers the patient's renal function and whether he or she is on dialysis; the PELD takes into account the child's age and degree of failure to grow.

The patient's results are entered into a formula that yields a score. MELD scores range from 6 (less ill) to 40 (gravely ill); PELD scores, from negative numbers to 99. A patient's MELD or PELD score can change as his or her condition worsens. Besides reflecting the patient's disease severity, the MELD or PELD score determines how urgently he or she will need a liver transplant in the next 3 months. Thus, it determines the patient's placement on the liver-transplant waiting list.

Nursing care

Nursing care for patients with liver failure focuses on supporting body systems, managing signs and symptoms of decreased liver function, and avoiding worsening cerebral edema.

- Monitor level of consciousness, blood pressure, volume status, blood and coagulation tests, and signs and symptoms.
- Keep the head of the bed elevated 30 degrees, with the patient's head in the neutral position.
- Decrease stimulation, such as frequent suctioning.
- Stay alert for hypercapnia and hypoxia; correct these conditions as indicated and ordered.
- Manage fever aggressively with a fan, cooling blanket, or both.
- Watch for signs and symptoms of infection and possible sepsis; administer antibiotics, as needed and ordered.
- Maintain strict glucose monitoring for possible hypoglycemia or hyperglycemia.
- Provide nutritional support as ordered.

Patients who reach an acuity level that warrants close monitoring (such as those in an intensive care unit with grade 3 or 4 hepatic encephalopathy) require ICP monitoring.

Postoperative care

After liver transplantation, assess the patient for such complications

Grading hepatic encephalopathy

Hepatic encephalopathy occurs when an impaired liver can no longer remove toxins from the blood, which causes serum ammonia levels to rise. Osmotic disturbances then occur in the brain, along with inflammation and increased cerebral blood flow.

Patients with hepatic encephalopathy exhibit confusion, hyperthermia, respiratory and circulatory problems, and increased intracranial pressure (ICP). Treatment may include lactulose given by mouth or as an enema to remove ammonia through the GI tract, titrated to three to four soft stools in 24 hours. Rifaximin, an antibiotic, may be used to treat infection; sodium benzoate, to decrease ammonia production; and hypertonic saline solution, to decrease elevated ICP.

Various scales have been developed to diagnose and classify hepatic encephalopathy. The table below shows a system with four grades of increasing severity.

Grade	Signs and symptoms
Grade 1	• Mild lack of awareness • Euphoria or anxiety • Shortened attention span • Impaired performance of addition or subtraction
Grade 2	• Lethargy or apathy • Minimal disorientation of time or place • Subtle personality changes • Inappropriate behavior • Impaired performance of addition or subtraction
Grade 3	• Somnolence to semi-stupor but responsive to verbal stimuli • Confusion • Gross disorientation
Grade 4	• Coma (unresponsive to verbal or noxious stimuli)

Common dysfunctions in ALF

Acute liver failure (ALF) leads to alterations in many body systems, as shown below.

Body system	Common dysfunctions
Cardiovascular	• Hypotension • Intravascular volume depletion • Low cardiac output • Right ventricular failure • Vasodilation
Central nervous system	• Intracranial hypertension • Progressive encephalopathy
Hematologic	• Coagulopathy
Immunologic	• Increased risk of sepsis
Metabolic	• Hyperammonemia • Hypoglycemia • Hyponatremia • Impaired drug metabolism • Lactic acidosis
Renal	• Renal dysfunction
Respiratory	• Increased risk of aspiration pneumonitis

as bleeding, infection, and rejection. Monitor the patient's temperature, urine output, neurologic status and hemodynamic pressures. Also provide education about immunosuppressive drugs.

Managing ascites

Liver failure commonly causes ascites—fluid buildup in the peritoneal cavity. As indicated, take appropriate steps to manage ascites, which can affect multiple body systems. The physician may order diuretics and a low-sodium diet. Aldosterone antagonists (such as spironolactone) may be used to treat ascites instead of loop diuretics (such as furosemide) because they spare potassium and address ascites pathophysiology. Although patients with ascites may be placed on fluid restrictions, studies fail to show this intervention is effective. Be aware that patients who undergo paracentesis to treat ascites may need follow-up fluid replacement and albumin administration.

Take steps to *manage* ascites, which can affect multiple body systems.

Rising to the challenge

With continuing medical advances and more patients waiting for liver donors, you're likely to encounter more patients with liver failure. To optimize outcomes, make sure you're familiar with the signs and symptoms of liver failure, ascites, and hepatic encephalopathy. Finally, be sure to provide emotional support to patients and families. ★

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