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RECOGNIZING & RESPONDING TO

ACUTE LIVER FAILURE

By Mary G. McKinley, RN, CCRN, MSN

By quickly recognizing the signs and symptoms of acute liver failure, you can help your patient improve his odds of surviving this often-deadly condition.

CARING FOR A PATIENT with acute liver failure (ALF), a medical emergency, is complex and challenging. ALF affects about 2,000 people per year in the United States, killing 40% or more of them. This rare condition often strikes young people.[1,2]

In this article, I'll tell you how to recognize a patient with ALF, describe the pathophysiology, and discuss how to care for him to improve his chance of survival.

What's it all about: ALF

Weighing approximately 3 pounds (1.4 kg) in the adult, the liver is not only the largest visceral organ of the body, but also one of the most metabolically active. It has digestive, endocrine, excretory, and hematologic functions. (See *The liver wears many hats*.) In ALF, all of these functions can be disrupted, causing multiple signs and symptoms and management challenges.

Because the liver is involved in so many metabolic processes, it's vulnerable to various insults. According to the American Association for the Study of Liver Diseases (AASLD), "the most widely accepted definition of ALF includes evidence of coagulation abnormality, usually an international normalized ratio (INR) greater than or equal to 1.5, and any degree of mental alteration (encephalopathy) in a patient without preexisting cirrhosis and with an illness of less than 26 weeks' duration."[1]

According to the AASLD, the term *ALF* is preferred to other terms, such as *fulminant hepatic failure* and *fulminant hepatitis* or *necrosis*, for conditions lasting 26 weeks or less. In addition, the AASLD also recommends against using terms related to the illness's duration "such as hyperacute (less than 7 days), acute (7 to 21 days), and subacute (more than 21 days and less than 26 weeks)" because these terms, when used alone, don't offer clues to the patient's prognosis.[1]

Focusing on toxicity and infection

Although many conditions can lead to ALF, it's most commonly caused by toxic agents and infectious disorders. See *ALF: One condition, many causes*.

Acetaminophen overdose is the most common single cause of ALF in the United States and Europe.[1] Acetaminophen is directly hepatotoxic and causes necrosis of the liver parenchyma. The toxic dose is highly variable, but dosages of 150 mg/kg or approximately 7 grams in an adult have been

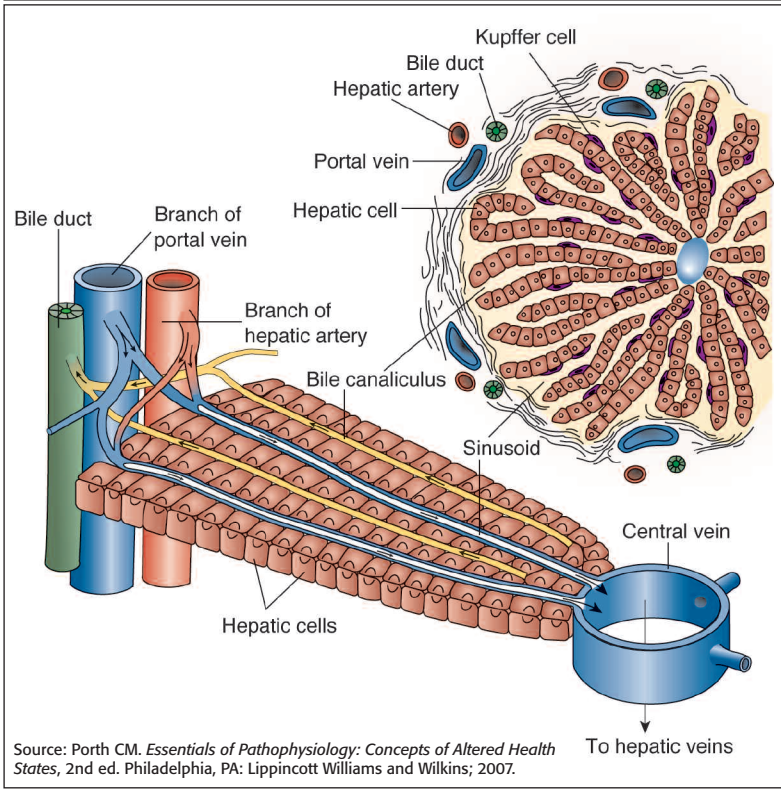
identified as toxic.[3]

Because acetaminophen is available over the counter, acetaminophen overdose is a common method of attempted suicide. Acetaminophen is also an active ingredient in many over-the-counter remedies (such as cold and flu products), which can lead to an unintentional overdose.

Even when taken in amounts below the recommended maximum dose for adults (4 grams in 24 hours), acetaminophen can trigger ALF in susceptible people. For example, those who chronically use alcohol, have preexisting liver disease, are malnourished, or who are fasting may be more vulnerable to toxicity at lower doses.

Hepatitis A and B viruses cause some cases of ALF in the United States.[1] Other forms of hepatitis have been associated with ALF less frequently.[4] (For other possible triggers, see *ALF: One condition, many causes.*) For about 20% of patients with ALF, the cause isn't known.[1]

Cross section of liver lobule



Source: Porth CM. *Essentials of Pathophysiology: Concepts of Altered Health States,* 2nd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2007.

Three keys to ALF

ALF involves these three pathophysiologic mechanisms:[5,6]

• ***Hepatocellular dysfunction that develops rapidly*** disrupts many normal liver functions, including elimination of bilirubin; synthesis of protein, glucose, and coagulation factors; and regulation of lactate. Because the liver stops synthesizing plasma proteins, capillary oncotic pressure decreases, causing fluid to shift from intravascular to interstitial or intraperitoneal space. Hormones such as aldosterone aren't inactivated due to this dysfunction. High circulating levels of aldosterone cause the kidneys to retain sodium and water and excrete potassium. The end result is further fluid and electrolyte imbalances.

• ***Blood flow through the liver is disrupted.*** Cellular inflammation and degeneration in the liver increase resistance to blood flow, resulting in portal hypertension. In turn, portal hypertension causes congestion and engorgement of venous circulation, particularly in the gastrointestinal (GI) and renal

The liver wears many hats

Digestive role	Endocrine role	Excretory role	Hematologic role
• Produces bile salts for fat digestion • Processes and stores fats, carbohydrates, and proteins • Processes and stores vitamins and minerals • Synthesizes cholesterol • Produces triglycerides	• Regulates the metabolism of carbohydrates, fats, and proteins • Metabolizes hormones such as mineralocorticoids, glucocorticoids, and sex hormones	• Excretes bile • Excretes cholesterol • Converts ammonia to urea • Detoxifies drugs, hormones, and other foreign substances	• Stores blood • Synthesizes all but two clotting factors • Synthesizes bilirubin

Source: Alspach J, 2006.[9]

ALF: One condition, many causes

Here are some of the many causes of ALF.

Infections	Toxins	Injury	Parenchymal disease	Other processes
• Hepatitis A and B viruses • Herpes simplex virus • Varicella-zoster virus • Epstein-Barr virus • Cytomegalovirus	• Drugs: acetaminophen, halothane, methyldopa, isoniazid, chronic alcohol abuse, ecstasy • Other toxins: sea anemone, mushroom poisoning, carbon tetrachloride	• Ischemia after cardiac arrest, shock, or severe heart failure	• Malignant infiltration: lymphoma, melanoma, breast cancer • Primary liver tumor • Cirrhosis • Wilson's disease	• Vascular abnormalities such as hepatic veno-occlusive disease (Budd-Chiari syndrome) • Fatty liver of pregnancy • Primary graft nonfunction following liver transplant

Sources: Polson J, Lee WM, 2005;[1] Sood GK, 2008;[2] Feldman M, Friedman LS, Sleisenger MH, 2002;[5] Sass DA, Shakil AO, 2005.[6]

systems. This engorgement can lead to esophageal varices and bleeding. The patient may also develop ascites, which occurs when protein-rich fluid leaks from the vascular space to the peritoneal cavity.

• ***Cerebral edema and intracranial hypertension*** are considered the most serious complications of ALF.[1] Although the pathogenesis of cerebral edema in ALF is poorly understood, it may result from the release of neurotoxins (such as ammonia) from the GI system. Neurotoxins not cleared by the dysfunctional liver then accumulate in the systemic circulation.

The two mechanisms driving cerebral edema are brain cell swelling (cytotoxic edema) and disruption of the blood-brain barrier (vasogenic edema). Progressive cerebral edema produces intracranial hypertension, which impairs cerebral perfusion and can lead to irreversible neurologic damage. Other factors that may contribute to encephalopathy include hypoglycemia, sepsis, hypoxemia, and seizures.[5,6]

Reaching a diagnosis

Because of the vital functions of the liver, the patient may have many signs and symptoms, including these:

• weakness, fatigue, or malaise from alterations in metabolism of fats, proteins, and glucose

• anorexia or poor nutritional status from poor venous blood flow through the GI tract (portal hypertension)

• bleeding and bruising from altered synthesis of clotting factors

• jaundice from decreased bilirubin uptake and conjugation

• encephalopathy characterized by central nervous system disturbances ranging from a lack of mental alertness to confusion and coma. These changes are caused by abnormal protein metabolism, the inability of the liver to convert ammonia to urea, and the accumulation of neurotoxins in the brain.

• hypotension and fluid and electrolyte imbalance from decreased production of plasma proteins in the liver, which reduces capillary oncotic pressure causing fluid to shift from the vascular to the interstitial space, and possible blood loss from clotting abnormalities.

In search of a cause

When assessing a patient with ALF, the most important step is to identify and treat the underlying cause. For instance, acetaminophen toxicity can be treated with N-acetylcysteine; ALF precipitated by herpesvirus may respond to acyclovir.[5]

Early in the course of therapy, the healthcare team, the patient, and his family should consider liver transplantation because many patients with ALF will experience rapid deterioration and the onset of serious complications.[6] With early consideration for transplant, he can be placed on appropriate transplant lists and transferred to a liver transplantation center as needed. (See *Considering transplant*.)

Managing patient care

As with any patient, clinical priorities are to maintain the ABCs (airway, breathing, and circulation). Elevate the head of the bed to help prevent aspiration and facilitate breathing and administer oxygen as needed. Prepare to assist with endotracheal intubation and initiate mechanical ventilation if indicated.

Antiepileptic drugs may be needed to treat or prevent seizures. Seizures may acutely increase cerebral metabolic rate, cause cerebral hypoxia, and further contribute to cerebral edema.[7]

Ongoing care depends on the patient's condition. Initiate appropriate nursing interventions for these common signs and symptoms of ALF.

Weakness, fatigue, malaise. Encourage the patient to rest by organizing his care, pacing his activities, and minimizing external stimuli. Rest

reduces the demand on the liver so it can recover function.[6] Take steps to prevent potential complications of immobility.

- Prevent pneumonia by encouraging coughing, deep breathing, ambulating, and using incentive spirometry, and by following evidence-based infection control guidelines.
- Prevent venous thromboembolism by implementing thromboprophylaxis according to the American College of Chest Physicians evidenced-based clinical practice guidelines.
- Prevent pressure ulcers by identifying at-risk patients and implementing strategies such as meticulous skin care, optimal nutrition and hydration, and minimizing pressure by turning and repositioning patients every 2 hours and using pressure-relieving surfaces.

Anorexia, poor nutritional status. Improving the patient's nutritional status will help him regain strength and become more active. Routine assessment includes measuring and recording daily weights, abdominal girth, and intake and output and monitoring his serum glucose and electrolytes, as well as serum albumin, transferrin, and prealbumin levels.

If possible, the patient's diet should have an adequate amount of protein (60 grams daily), supplemented by vitamins A, C, and K, B complex vitamins, and folic acid. Arrange for a nutritional consult. It was once routine to restrict protein to 0.6 grams/kg for patients with ALF because ammonia is a by-product of protein and amino acid metabolism and increased ammonia is associated with hepatic encephalopathy, but this may not be necessary.[2] Initiate enteral feedings early in the course of treatment. Don't restrict protein severely; usually 60 grams/day of protein is reasonable. If your patient can't have enteral feedings, he may need parenteral nutrition.[1]

Carefully monitor his electrolyte balance, and modify his diet as ordered. For example, if he has ascites, the healthcare provider may order sodium restriction; if renal function is compromised, he may restrict potassium. If the patient develops signs and symptoms of increasing encephalopathy, protein restriction may be indicated.

If the patient is anorexic, he may tolerate frequent small meals better than three larger meals. He may need oral supplements or, if he isn't eating, a specialized enteral or parenteral formula. For example, HepatAmine, a hypertonic solution containing crystalline amino acids, can be administered I.V. to provide adequate protein and supplemental requirements of vitamins and minerals such as potassium. Vivonex Plus, an elemental formula feeding (100% free amino acids), can be given orally or through a nasogastric (NG) tube and is easily absorbed, providing needed nutritional support.[8]

Because patients with ALF commonly develop hypoglycemia, monitor your patient's blood glucose levels. Hypoglycemia should be managed with continuous glucose infusions.[1]

Coagulation problems. To monitor the patient for possible occult bleeding, assess his vital signs, check his stools and urine for blood, and assess lab work results, including complete blood cell count, platelets, prothrombin time, INR, and bleeding times.

To prevent GI bleeding, initiate aggressive preventive treatments as ordered. For example, administer a histamine receptor antagonist or proton pump inhibitor and insert an NG tube to monitor for bleeding and check gastric pH.[9] Vitamin K is routinely administered subcutaneously for coagulopathy.[1]

You may need to administer clotting factors, such as fresh frozen plasma or platelet transfusions, if the patient is hemorrhaging or prior to an invasive procedure. Giving an infusion of plasma to a patient with heart failure or renal dysfunction can be challenging because this can lead to volume overload and respiratory failure. The healthcare provider will carefully weigh the associated risks and benefits.

To minimize the risk of injury and bleeding, take these steps to protect your patient:

- Assess his fall risk and institute fall precautions if indicated.
- Institute seizure precautions as indicated.

Considering transplant

Before deciding on transplant, the healthcare team must balance the likelihood of spontaneous recovery with the risks associated with transplantation. The King's College Criteria, identified in 1989, are the validated criteria for liver transplant. The following variables are significant criteria for transplant: disease etiology, age of patient, jaundice to coma interval, serum bilirubin level, prothrombin time, arterial pH, and serum creatinine.[6] These criteria are readily available and could be used to expedite transfer to a center and early listing for a transplant. Living donor transplantation is also a possibility made necessary by the scarcity of organs from deceased donors.

Artificial liver support devices such as the bioartificial liver are being studied. In this device, blood flows through an extracorporeal circuit lined with hepatocytes. Although these devices may serve as a bridge to transplantation, so far no mechanical devices have favorably impacted the outcome of ALF.[6] "Currently available liver support systems aren't recommended outside of clinical trials; their future in the management of ALF remains unclear."[1]

Before liver transplantation was available, as few as 15% of patients with ALF survived.[1] The refinement of transplant surgery, immunosuppressive agents, and comprehensive care for these patients has increased the survival rate post-transplant to 65% or even 80%.[6]

• Teach him to use an electric razor instead of a safety razor and to use a soft-bristled toothbrush.
• Administer stool softeners, as prescribed.
• Apply pressure to all puncture sites until hemostasis is achieved.

Esophageal or gastric varices. If bleeding from varices is a risk, the healthcare provider may consider esophagogastroduodenoscopy and sclerotherapy, which involves injecting a sclerosant (such as sodium morrhuate) into the bleeding varix and causing it to thrombose. This procedure has a 90% success rate.[10]

Pharmacologic approaches to treat bleeding varices include octreotide and vasopressin given in conjunction with nitroglycerin. Octreotide is a synthetic somatostatin that diminishes blood flow to the portal system due to vasoconstriction, thus decreasing variceal bleeding. Vasopressin is a potent splanchnic vasoconstrictor. Reducing blood flow to the splanchnic organs decreases portal inflow and portal pressure. Nitroglycerin is used with vasopressin to reduce the detrimental effects of vasopressin while preserving its beneficial effects.[11]

A treatment used as a temporary, lifesaving or emergency measure is the Sengstaken-Blakemore tube; similar tubes include the Minnesota and the Linton-Nachlas. These tubes, which provide tamponade at the site of bleeding, aren't generally the treatment of choice because of their potential for complications, such as airway compromise or recurrent bleeding following clot disruption upon removal.[10,11]

Skin integrity compromise. Pruritus and edema are often associated with liver failure. Take these steps to prevent injury, reduce itching, and maintain skin integrity.
• Inspect the patient's skin daily and document assessment findings.
• Keep the patient's fingernails short.
• Avoid alcohol-based skin products and scented lotions and soaps, which can be drying.
• Use tepid rather than hot water for bathing and use emollients or gentle cleansers.
• Minimize pressure, especially over bony prominences. Redistribute the pressure on the skin by turning and repositioning him every 2 hours and using pressure-relieving surfaces, such as alternating-pressure mattresses or low-air-loss beds.
• Maintain function with active and passive range-of-motion exercises and elevate edematous extremities whenever possible.
• Optimize nutrition and hydration.

Encephalopathy. Frequently assess your patient's level of consciousness to help identify changes early and assist in directing care. Watch for subtle changes in mentation and abnormal involuntary movements that may indicate seizure activity. Assess for reversible causes of mental status alterations, such as hypoglycemia or hypoxemia, and manage them appropriately.

Intracranial pressure (ICP) monitoring may be used to detect elevated ICP and reduced cerebral perfusion pressure. However, placing invasive intracranial monitoring devices in a critically ill patient increases his risk of bleeding and infection.[1] Treatment goals for a patient with encephalopathy include eliminating excess blood ammonia, which is a neurotoxin. Lactulose is a synthetic disaccharide that decreases blood ammonia concentrations and reduces the degree of encephalopathy. Studies have shown that lactulose reduces the blood ammonia levels by 25% to 50%, which is usually accompanied by an improvement in mental status.[12] However, using lactulose in these cases may lead to gaseous abdominal distension that may cause an upcoming transplant procedure to be technically challenging.[1] The healthcare provider may also order drugs such as neomycin to reduce intestinal flora, which contribute to encephalopathy by increasing ammonia production.

Because the occurrence of cerebral edema and intracranial hypertension is related to the severity of encephalopathy, focus your nursing care on interventions that can help to reduce ICP.
• Elevate the head of the bed and position the patient to allow maximum cerebral venous outflow (no neck flexion).
• Be prepared to assist with endotracheal intubation and initiation of mechanical ventilation.
• Monitor vital signs frequently, especially BP, to maintain adequate cerebral perfusion pressure.
• Administer antiepileptic drugs as prescribed because seizure activity can cause acute elevations in ICP.
• Group nursing interventions to minimize stimulation and keep the patient's environment quiet.

The healthcare provider may order mannitol, an osmotic diuretic, to reduce cerebral edema and promote diuresis. Another option is administering a short-acting barbiturate to reduce cerebral metabolic rate when severe intracranial hypertension doesn't respond to other measures, making sure that adequate mean arterial pressure is maintained.[1]

Inducing mild to moderate hypothermia to decrease cerebral metabolic rate and reduce intracerebral hypertension is also controversial. No prospective control trial has defined the specifics of achieving therapeutic hypothermia (for instance, the optimal temperature, length of treatment, or rewarming procedure) or determined whether it should be performed prophylactically or therapeutically.[6]

Fluid and electrolyte imbalances. Monitor serum chemistry results and report abnormalities to the patient's healthcare provider. As part of your hemodynamic assessment for volume deficits, assess the patient's vital signs frequently. Hemodynamically unstable patients may need to have a pulmonary artery catheter inserted to guide appro-

priate fluid replacement therapy. Continuous renal replacement therapy may be needed for patients in acute renal failure.[1]

To maintain fluid balance, you may need to administer colloids to improve the capillary oncotic pressure and reduce third-space fluid shifts. However, give protein-based colloids (such as albumin) judiciously to patients with hepatic encephalopathy and ascites because they may increase cerebral compromise and ascitic fluid.[6]

Aldosterone antagonist diuretics or potassium-sparing diuretics may be ordered to decrease fluid retention, thus slowing the development of ascites and reducing the workload of the heart. If these diuretics lose their effectiveness over time, the healthcare provider may order loop diuretics instead.[7] Because hypovolemia related to diuretic therapy is also a concern, monitor your patient to prevent volume depletion. Watch for these signs and symptoms of fluid overload: weight gain, hypertension, peripheral edema, dyspnea, tachypnea, orthopnea, neck vein distension, or pulmonary crackles. Signs and symptoms of dehydration include hypotension, tachycardia, orthostasis, flat neck veins, poor skin turgor, and thirst.

Facing challenges

Teach your patient and his family about ALF and its management. Many patients with ALF are very unstable, require admission to the ICU, and face rapidly progressive liver failure, requiring liver transplantation.

ALF, with its wide range of pathophysiologic processes and signs and symptoms, is a challenging disease that can be rapidly fatal. When you recognize a patient with ALF early and provide aggressive and appropriate treatment, you improve his chances of survival. ✧

REFERENCES

1. Polson J, Lee WM, American Association for the Study of Liver Disease. AASLD position paper: the management of acute liver failure. *Hepatology*. 2005;41(5):1179-1197.
2. Sood GK. Acute liver failure. Updated June 10, 2008. http://www.emedicine.com/MED/topic990.htm.
3. Farrell SE Acetaminophen toxicity. Updated October 3, 2007. http://www.emedicine.com/emerg/topic819.htm.
4. Ostapowicz G, Fontana RJ, Schiødt FV, et al. U.S. Acute Liver Failure Study Group. Results of a prospective study of acute liver failure at 17 tertiary care centers in the United States. *Ann Intern Med*. 2002;137(12):947-954.
5. Feldman M, Friedman LS, Sleisenger MH. *Sleisenger and Fordtran's Gastrointestinal and Liver Disease: Pathophysiology/Diagnosis/Management*, vol. 2, 7th ed. St. Louis, MO: W.B. Saunders; 2002.
6. Sass DA, Shakil AO. Fulminant hepatic failure. *Liver Transpl*. 2005;11(6):594-605.
7. Dennison RD. *Pass CCRN!*, 3rd ed. St Louis, MO: Mosby Elsevier; 2007.
8. American Dietetic Association. *Nutrition Care Manual*. http://www.nutritioncaremanual.org.
9. Alspach J. *Core Curriculum for Critical Care Nursing*, 6th ed. St. Louis, MO: Saunders Elsevier; 2006.
10. Azer SA. Esophageal varices. Updated April 12, 2006. http://www.emedicine.com/med/topic745.htm.
11. Treger R, Graham TP, Dea SK. Sengstaken-Blakemore tube. Updated August 26, 2008. http://www.emedicine.com/proc/topic81020.htm.
12. *Mosby's Drug Consult 2007*. St. Louis, MO: C.V. Mosby; 2007.

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Recognizing and responding to acute liver failure

GENERAL PURPOSE To provide nurses with an overview of acute liver failure (ALF). **LEARNING OBJECTIVES** After reading the preceding article and taking this test, you should be able to: **1.** List the causes of ALF. **2.** Describe the pathophysiology of ALF. **3.** Identify nursing interventions for patients with ALF.

1. The liver performs all of the following functions *except*
a. converting ammonia to urea.
b. synthesizing clotting factors.
c. producing triglycerides.
d. producing hemoglobin.

2. The accepted definition of ALF includes all of the following *except*
a. preexisting cirrhosis.
b. evidence of a coagulation abnormality.
c. some degree of encephalopathy.
d. illness lasting less than 26 weeks.

3. ALF is most commonly caused by toxic agents and
a. allergic disorders.
b. infectious disorders.
c. abdominal trauma.
d. severe dehydration.

4. The most common cause of ALF in the United States is
a. acetaminophen overdose.
b. abdominal trauma.
c. cirrhosis.
d. viral infection.

5. Which of the following *doesn't* make a person more susceptible to acetaminophen toxicity?
a. obesity
b. chronic alcohol use
c. fasting
d. malnutrition

6. What's considered the most serious complication of ALF?
a. GI bleeding
b. jaundice
c. intracranial hypertension
d. fluid volume overload

7. The two mechanisms driving cerebral edema in ALF are cytotoxic edema and
a. traumatic brain injury.
b. interstitial edema.
c. coagulopathies.
d. vasogenic edema.

8. Progressive cerebral edema may produce
a. neurotoxin accumulation.
b. hyperglycemia.
c. depletion of aldosterone levels.
d. irreversible neurologic damage.

9. What causes anorexia in patients with ALF?
a. portal hypertension
b. decreased plasma protein production
c. decreased capillary oncotic pressure
d. altered protein metabolism

10. Decreased bilirubin uptake and conjugation in ALF can lead to
a. encephalopathy.
b. jaundice.
c. bruising.
d. portal hypertension.

11. ALF caused by acetaminophen toxicity can be treated with
a. calcium chloride.
b. sodium bicarbonate.
c. N-acetylcysteine.
d. cation-exchange resin.

12. ALF precipitated by herpesvirus may be treated with
a. acyclovir.
b. N-acetylcysteine.
c. gentamicin.
d. ampicillin.

13. Which of the following *isn't* a significant criterion for a liver transplant?
a. the jaundice to coma interval
b. disease etiology
c. serum glucose
d. serum creatinine

14. Which of the following statements about nutrition and ALF is correct?
a. Potassium should be restricted for a patient with ascites.
b. Vivonex Plus may be given I.V. for nutritional support.
c. Protein intake should be severely restricted.
d. Serum electrolytes should be carefully monitored.

15. Octreotide is best described as
a. a synthetic somatostatin.
b. a fibrinolytic.
c. a sclerosant.
d. an anticoagulant.

16. Lactulose is given to patients with ALF to
a. induce abdominal distension to prepare for and facilitate transplantation.
b. decrease blood ammonia concentrations and reduce encephalopathy.
c. reduce blood flow to the splanchnic organs to treat varices.
d. reduce interstitial edema and promote diuresis.

17. Which sign or symptom might indicate fluid overload?
a. hypotension
b. bradycardia
c. orthopnea
d. orthostasis

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