



# Nursing Management of Acute Liver Failure: A Clinical Review

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## Abstract

Acute Liver Failure (ALF) is a rapid and severe clinical syndrome defined by the onset of liver dysfunction and encephalopathy within eight weeks of symptoms in healthy individuals, or two weeks following jaundice in those with pre-existing disease. ALF arises from diverse etiologies. Nursing management is a cornerstone of ALF care, requiring intensive monitoring of neurological status, hemodynamic stability, and fluid-electrolyte balance. Specific interventions include managing cerebral edema, preventing hemorrhage through safety precautions, and providing nutritional support tailored to prevent ammonia accumulation. ALF presents a high-acuity challenge requiring specialized nursing care

**Keywords:** Acute Liver Failure; Liver; Hepatic Encephalopathy; Nursing Management

## Introduction and Pathophysiology

The liver functions as the body's "chemical factory," playing a vital role in metabolic homeostasis. It is responsible for the metabolism of carbohydrates (converting sugars to glycogen), proteins (synthesizing coagulation factors and antibodies), and lipids. Furthermore, it facilitates the absorption of fat-soluble vitamins (A, D, E, K) through bile production and serves as a storage site for B-complex vitamins. In addition to its metabolic roles, the liver detoxifies substances such as ammonia and inactivates various hormones and drugs [1, 2].

Acute Liver Failure (ALF) is a rapid and severe impairment of these functions. It is clinically defined as the development of liver damage and encephalopathy within eight weeks of symptom onset in previously healthy individuals, or within two weeks of jaundice in those with pre-existing disease [3]. Without a timely liver transplant, the mortality rate for this condition reaches approximately 80% [4].

## Etiology and Diagnostic Indicators

The causes of ALF are diverse (Table 1), though in approximately 20% of cases, the etiology remains unknown. Diagnosis is typically achieved through a combination of patient history, laboratory markers, and imaging such as Brain CT [5, 6].

## Clinical Presentation

Early symptoms are often non-specific and include weakness, fatigue, nausea, and dizziness. As the disease progresses, clinical signs of liver failure become more apparent. Jaundice is a hallmark sign, characterized by yellowing of the skin and conjunctiva, dark urine, and pale stools, occurring when bilirubin levels exceed 2.5 mg/dl. Other signs include coagulopathy (leading to bruising), hepatic foetor, and skin manifestations like spider angiomas or palmar erythema [6-8].

## Complications and Grading of Encephalopathy

One of the most severe complications is hepatic encephalopathy. Hepatic encephalopathy is a reversible neuropsychiatric syndrome characterized by a spectrum of brain dysfunction resulting from severe liver insufficiency and/or blood bypassing the liver (portosystemic shunting). In this state, the liver is unable to effectively filter neurotoxins—most notably ammonia—from the bloodstream. These toxins eventually cross the blood-brain barrier, disrupting central nervous system (CNS) function and leading to a range of cognitive, motor, and psychiatric disturbances [9, 10] (Table 2).

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Table 1: ALF causes.

Category	Causes	Diagnostic/Clinical Approach
Viral	Hepatitis B, D, CMV, Epstein-Barr, Herpes Simplex	Hepatitis markers, laboratory indices, biopsy
Metabolic	Wilson's Disease, Acute Fatty Liver of Pregnancy	Serum Ceruloplasmin, copper levels, monitoring for preeclampsia
Autoimmune	Autoimmune Hepatitis	Specific immunological markers, liver biopsy
Infiltrative	Metastatic neoplasms (colon, prostate), Leukemia, Lymphoma	Liver biopsy, chemotherapy evaluation
Toxic	Acetaminophen toxicity, Mushroom poisoning	History, blood levels of toxic substances
Vascular	Hypoperfusion/Ischemia	History of hypotension or reduced hepatic blood flow

Table 2: Hepatic encephalopathy.

Stage	Clinical Manifestations
Stage 0	Subclinical phase with subtle decreases in mental function
Stage I	Apathy, restlessness, sleep disturbances, impaired concentration
Stage II	Lethargy, drowsiness, disorientation, asterixis (flapping tremor)
Stage III	Stupor, hyperreflexia, response only to major stimuli
Stage IV	Coma; response only to painful stimuli

Table 3: Nursing interventions for ALF patients.

Nursing priority	Interventions	Clinical rationale
Neurological	Glasgow Coma Scale (GCS) monitoring; Elevate head of bed 20-30°.	Monitors for intracranial hypertension and reduces cerebral edema.
Safety	Use of soft toothbrushes, electric razors, and prolonged pressure on puncture sites.	Prevents hemorrhage in patients with severely reduced clotting factors.
Nutrition	Small meals, 40-60g protein/day, Vitamin A, B, C, K supplements.	Supports liver regeneration while limiting ammonia production.
Integumentary	Repositioning every 2 hours; avoid alcohol-based soaps; elevate edematous limbs.	Prevents pressure ulcers and manages pruritus/skin breakdown.
Fluid/Electrolyte	Monitor for fluid overload (edema, dyspnea) or dehydration; limit sodium for ascites.	Manages the third-spacing of fluids and monitors renal function.

Other life-threatening complications include cerebral edema (managed with mannitol and head elevation), GI bleeding, hypoglycemia, and multisystem organ failure [11].

## Nursing Implications and Interventions

The nursing care of a patient with ALF requires high-intensity monitoring, usually in an Intensive Care Unit (ICU). Nurses must focus on hemodynamic stability, neurological assessments, and the prevention of secondary complications. Given the high mortality rate without transplant, nurses must prioritize early evaluation for liver transplantation as it remains the only definitive therapy [12-14].

ALF presents a high-acuity challenge requiring specialized nursing care that bridges neurological, metabolic, and hemodynamic monitoring. The following implications and interventions are critical for nursing practice:

- **Neurological vigilance:** Frequent assessment of the central nervous system (CNS) and mental level is mandatory to detect early signs of hepatic encephalopathy or increased intracranial pressure (ICP) [15].
- **Safety and injury prevention:** Nurses must implement strict bleeding precautions due to reduced synthesis of clotting factors. This includes using soft toothbrushes, electric razors, and maintaining direct pressure on all puncture sites until hemostasis is achieved [16].
- **Metabolic and nutritional regulation:** Management requires a delicate balance of protein intake, typically 40–60g per day, to support recovery while preventing ammonia accumulation. Monitoring for hypoglycemia is a priority, as the liver can no

longer maintain glucose levels [17, 18].

- **Hemodynamic stability:** Continuous monitoring of blood pressure and central venous pressure is essential, often requiring the administration of fluids and vasopressors like dopamine to maintain cardiac output [17, 19].
- **Integumentary protection:** Patients are at high risk for skin breakdown due to edema and pruritus. Nursing care should include using lukewarm water, avoiding alcohol-based soaps, and utilizing alternating pressure mattresses [18, 20].
- **Respiratory support:** Because ALF often leads to respiratory failure or coma, nurses must be prepared for mechanical ventilation and airway management [17, 18].

## Conclusion

Nurses serve as the primary safeguard against the rapid deterioration characteristic of ALF, where the clinical status can shift from stable to life-threatening within hours. Because mortality is high without a transplant, the importance of clinical vigilance, quick response to complications and constant awareness from the nurse are the most significant factors in patient stabilization and bridge-to-transplant success.

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