

Management of the Complications of Cirrhosis

- Paul Wisniewski, DO
- Trauma Director
- Surgical Critical Care Fellowship Director



Disclosures

- None



Learning Objectives

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1. Understand the key complications that arise from cirrhosis.
2. Identify the management strategies for each complication.
3. Understand the role of medical, pharmacological, and interventional treatments.
4. Recognize the importance of early diagnosis and timely intervention to improve patient outcomes.



Introduction Complications

Ascites: Occurs in 50-60% of patients, with 20-30% mortality over 1 year.

Hepatic Encephalopathy: Occurs in 30-45% of patients, with 50-70% mortality in severe cases.

Portal Hypertension/Varices: Occurs in 60-70%, with 20-30% mortality following variceal bleeding.

Gastroesophageal Variceal Bleeding: Occurs in 30-40%, with 20-30% mortality after bleeding.

Infections: Occurs in 20-40%, with 30-40% mortality from SBP and infections.

Hepatorenal Syndrome: Occurs in 5-10%, with 50-80% mortality if untreated.



Pathophysiology of Acute Liver Failure

Portal Hypertension

- **Definition:** Increased pressure in the portal vein due to liver scarring.
- **Management:**
 - Non-selective beta-blockers (e.g., propranolol) to reduce portal pressure.
 - Endoscopic variceal ligation (EVL) or sclerotherapy for varices.
 - TIPS (Transjugular Intrahepatic Portosystemic Shunt) procedure in refractory cases.



Portal Hypertension

Portal Hypertension (including Varices)

- **Occurrence:** About **60-70%** of cirrhosis patients develop portal hypertension, which can lead to the formation of varices.
- **Mortality:** The risk of bleeding from varices is significant. **10-15%** of cirrhotic patients with varices will experience a bleeding episode. Mortality after a variceal bleed is high, with a **1-year mortality rate of 20-30%** following an acute bleeding episode.



Causes of Acute Liver Failure

Ascites

- **Definition:** Accumulation of fluid in the abdomen due to portal hypertension and hypoalbuminemia.
- **Management:**
 - Sodium restriction and diuretics (spironolactone, furosemide).
 - Paracentesis for large-volume ascites or symptomatic relief.
 - Albumin infusion for large-volume paracentesis.
 - Consideration for TIPS in refractory cases.



Ascites

Ascites

- **Occurrence:** 50-60% of cirrhosis patients will develop ascites during the course of the disease.
- **Mortality:** Ascites itself is not directly fatal, but it significantly impacts quality of life and is a major risk factor for other complications. The 1-year mortality rate for patients with ascites is around **20-30%**.



Causes of Acute Liver Failure

Hepatic Encephalopathy

- **Definition:** Decline in brain function due to liver failure, characterized by confusion, altered mental state.
- **Management:**
 - Lactulose to reduce ammonia levels.
 - Rifaximin to decrease gut-derived toxins.
 - Address underlying triggers (e.g., infections, GI bleeding).



Hepatic Encephalopathy

Hepatic Encephalopathy

- **Occurrence:** Approximately **30-45%** of patients with cirrhosis will develop hepatic encephalopathy at some point.
- **Mortality:** Acute episodes of hepatic encephalopathy have a high mortality rate, with mortality rates reaching **50-70%** for patients with severe encephalopathy, especially if untreated or complicated by infections.



Clinical Presentation of Acute Liver Failure

Gastroesophageal Varices

- **Definition:** Dilated veins in the esophagus or stomach due to portal hypertension, at risk of rupture and bleeding.
- **Management:**
 - Primary prophylaxis with beta-blockers to reduce bleeding risk.
 - Acute bleeding: Endoscopic band ligation (EBL) or sclerotherapy.
 - Secondary prophylaxis with beta-blockers and EVL or TIPS.



Gastroesophageal Variceal Bleeding

Gastroesophageal Variceal Bleeding

- **Occurrence:** Occurs in **30-40%** of patients with cirrhosis, usually when portal hypertension becomes significant.
- **Mortality:** The mortality rate for an episode of variceal bleeding is **15-20%** in the first year after the bleeding event. The 6-week mortality rate after acute variceal bleeding is about **20-30%**.



Treatment of Acute Liver Failure

Hepatorenal Syndrome (HRS)

- **Definition:** Renal failure due to severe liver disease.
- **Management:**
 - Vasoconstrictors (e.g., terlipressin) to improve renal perfusion.
 - Albumin infusions.
 - Liver transplantation in severe cases.



Hepatorenal Syndrome (HRS)

Hepatorenal Syndrome (HRS)

- **Occurrence:** Hepatorenal syndrome occurs in about **5-10%** of patients with cirrhosis, particularly in advanced stages or in those with ascites.
- **Mortality:** Hepatorenal syndrome carries a very poor prognosis, with a **1-year mortality rate of 50-80%** if left untreated. The only definitive treatment is liver transplantation.



Treatment of Acute Liver Failure

Infections

- 1. Definition:** Increased risk of infections in cirrhosis due to immune dysfunction and ascitic fluid.
- 2. Management:**
 1. Antibiotic prophylaxis for spontaneous bacterial peritonitis (SBP).
 2. Prompt treatment with broad-spectrum antibiotics for infections.
 3. Monitoring and managing infections aggressively, especially in patients with ascites.



Spontaneous Bacterial Peritonitis (SBP)

Infections (including Spontaneous Bacterial Peritonitis)

- **Occurrence:** Cirrhotic patients, especially those with ascites, have a high susceptibility to infections, with **20-40%** of patients developing infections.
- **Mortality:** Infections in cirrhosis, including spontaneous bacterial peritonitis (SBP), have a significant mortality risk. The overall mortality rate for SBP is **30-40%**, and in general, infections can contribute to a **higher overall mortality** in cirrhosis.



Spontaneous Bacterial Peritonitis (SBP)

- The treatment of **spontaneous bacterial peritonitis (SBP)** in patients with cirrhosis generally involves **empiric antibiotic therapy** while awaiting culture results. The choice of antibiotics is aimed at covering the most likely pathogens, primarily **gram-negative bacteria** such as *Escherichia coli* and other enteric organisms, as well as **gram-positive cocci** like *Streptococcus pneumoniae*.



Spontaneous Bacterial Peritonitis (SBP)

First-Line Antibiotics for SBP:

1. Ceftriaxone (2 g IV every 24 hours)

1. A broad-spectrum **third-generation cephalosporin** commonly used for empiric treatment.

2. Cefotaxime (2 g IV every 8-12 hours)

1. Another **third-generation cephalosporin** with a similar spectrum of activity as ceftriaxone.

3. Piperacillin-tazobactam (3.375 g IV every 6 hours)

1. A **broad-spectrum beta-lactam/beta-lactamase inhibitor combination** that may be used if there's concern for multi-drug resistant organisms.

4. Carbapenems (e.g., meropenem 1 g IV every 8 hours)

1. Considered for use in patients with risk factors for **multidrug-resistant (MDR)** organisms or healthcare-associated infections.



Spontaneous Bacterial Peritonitis (SBP)

Second-Line Options (if resistance or treatment failure occurs):

1. Fluoroquinolones (e.g., ciprofloxacin 400 mg IV every 12 hours)

1. Can be used if there is a history of **fluoroquinolone-sensitive** organisms but should be avoided in areas with high resistance rates.

2. Ampicillin-sulbactam (3 g IV every 6 hours)

1. An alternative to ceftriaxone in certain settings, particularly if gram-positive organisms are suspected.



Spontaneous Bacterial Peritonitis (SBP)

When to Use Antibiotics for SBP:

- **Empiric treatment** is started immediately after obtaining ascitic fluid for culture. Delay in initiating antibiotics increases mortality rates.
- After identification of the causative pathogen, antibiotic therapy should be tailored to sensitivities.

Duration of Therapy:

- Typically, **5 days** of antibiotic therapy is sufficient, although longer treatment may be necessary in cases of treatment failure or complicated SBP.

Prophylaxis:

- **Secondary prophylaxis** (to prevent recurrence of SBP) often involves **oral ciprofloxacin** (500 mg daily) in patients who have recovered from an episode of SBP and have a high risk of recurrence.
- In summary, **ceftriaxone** or **cefotaxime** is often the first-line empiric therapy for SBP, with adjustments based on culture results and local resistance patterns.



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