American Journal Of Biomedical Science & Pharmaceutical Innovation

VOLUME Vol.06 Issue02 2026
PAGE NO. 16-18
DOI 10.37547/ajbspi/Volume06Issue02-03

Clinical And Pathophysiological Aspects Of Liver Cirrhosis

Niyozova Yorqinoy Mirzaxamdamovna
Andijan State Medical Institute Assistant of the Department of Outpatient and Polyclinic Therapy, Uzbekistan

Received: 20 December 2025; **Accepted:** 11 January 2026; **Published:** 16 February 2026

Abstract: Diffuse fibrosis, nodule development, and loss of the hepatic parenchyma are the hallmarks of cirrhosis of the liver (CP), a chronic progressive illness that causes portal hypertension and hepatic failure. The illness is linked to a high rate of morbidity and death globally and is the last stage of many chronic liver illnesses. The origin, pathophysiology, clinical signs, diagnostic methods, therapeutic modalities, and prognosis of liver cirrhosis are all thoroughly covered in this article. The significance of early diagnosis and multidisciplinary care is emphasized in order to avoid consequences such hepatocellular cancer, ascites, variceal hemorrhage, and hepatic encephalopathy.

Keywords: Cirrhosis of the liver, fibrosis, portal hypertension, hepatic failure, hepatocellular carcinoma, pathogenesis.

Introduction: One of the most dangerous chronic liver disorders, cirrhosis continues to be a major worldwide health concern. More than one million fatalities are attributed to cirrhosis each year, making it one of the top 20 causes of mortality globally, according to the World Health Organization (WHO) [1]. Numerous chronic liver illnesses, such as autoimmune hepatitis, persistent alcohol use, nonalcoholic fatty liver disease (NAFLD), viral hepatitis (B, C, and D), and metabolic disorders, conclude with it.

Diffuse fibrosis around regenerating nodules is a histological characteristic of cirrhosis that alters the normal lobular architecture and compromises liver function. Clinically, it shows up as portal hypertension and hepatic insufficiency. Developing successful diagnostic and treatment strategies requires an understanding of its origin and progression processes.

Antiviral medications are used at specialist clinics to treat viral liver cirrhosis (LC). The risk of bacterial infections and LC decompensation is greatly increased when interferon-alpha, particularly pegylated versions, is administered. Only compensated viral cirrhosis C and D are treated with standard interferon treatment and ribavirin under close hematologic supervision.

Nucleotide/nucleoside analogs as entecavir and tenofovir are thought to be the safest and most effective treatments for viral LC. Under careful clinical and laboratory observation, treatment is continued until the hepatitis B virus DNA is eliminated and HBsAg seroconverts to anti-HBs.

It has been demonstrated that persistent viral suppression encourages the reversal of cirrhosis and the regression of hepatocyte necrosis and fibrosis. As a result, the disease progresses much more slowly when the etiologic factor is eliminated or controlled. Since the etiologic factor is unknown in many individuals, therapy focuses on blocking pathogenic pathways, such as lowering iron in hemochromatosis, copper in Wilson's disease, or employing immunosuppressants in cirrhosis linked to autoimmune hepatitis.

Inhibiting fibrogenesis and encouraging the resorption of fibrotic tissue are the primary objectives of pathogenetic treatment. Stellate cells are important extracellular matrix makers that are triggered by oxidative stress, inflammatory cytokines, and many growth factors.

Hepatoprotectors are utilized extensively for their antifibrotic, cytoprotective, and antioxidant properties because direct antifibrotic medications are still being studied. UDCA (Ursosan, Ursofalk) and essential phospholipids (Essentiale Forte N, Essliver, Esledin) are

the most clinically proved.

Adetionine (Heptral) with silymarin (Legalon)

These substances stabilize hepatocyte membranes, enhance detoxification, and subtly prevent fibrosis. Silymarin and essential phospholipids also directly inhibit fibrosis. Intrahepatic cholestasis is treated with UDCA and ademetionine.

Essentiale, Esledin, and Legalon are medications with direct antifibrotic effect that are recommended for compensated LC without problems. UDCA's anti-inflammatory and antiapoptotic qualities are other reasons for its usage.

Antifibrotic treatment lasts around three months if the causative cause is eliminated; in LC, it should be ongoing. An alternation plan that is suggested: For three months, take two capsules of Essentiale Forte N every day. For three months, take 140 mg ×2 of Legalon every day. Esledin: two caps × two every day for three months. For three months, take 10–15 mg/kg of ursosan daily.

UDCA (Ursosan/Ursofalk) 15 mg/kg/day is the recommended medication for cholestasis.

Hepatoprotectors, B vitamins (particularly B_{12}), and pulse corticosteroid treatment with prednisolone or methylprednisolone are utilized in decompensation. Propranolol (10 mg ×3–4/day) or comparable β-blockers are recommended to lower portal hypertension.

Ascites is treated with albumin infusions and diuretics (furosemide, spirolactone).

Octreotide and midodrine can enhance sodium excretion and hemodynamics.

Lactulose (30–120 mL/day), Rifaximin (600–1200 mg/day), L-ornithine L-aspartate (Hepa-Merz), and a low-protein diet are among the treatments for hepatic encephalopathy.

Laparocentesis and albumin replacement are used when there are problems or refractory ascites.

Terlipressin, norepinephrine, or octreotide + midodrine with albumin are used to treat hepatorenal syndrome. Liver transplantation is still the sole viable option if treatment is unsuccessful or LC is decompensated.

1. Table – Treatment of Liver Cirrhosis

Therapy Type	Goal	Main Drugs	Duration / Notes
Etiologic	Eliminate viral or metabolic cause	Interferon + Ribavirin (HCV, HDV); Entecavir, Tenofovir (HBV)	Until viral DNA elimination & seroconversion
Pathogenetic	Suppress fibrogenesis, stabilize hepatocytes	UDCA, Essentiale, Legalon, Heptral, Esledin	3-month rotation, long-term
Anti-inflammatory / Immunosuppressive	Decrease inflammation and autoimmune damage	Prednisolone, Methylprednisolone	Pulse therapy, taper gradually
Symptomatic	Relieve portal hypertension, ascites, HE	Propranolol, Spironolactone, Furosemide, Lactulose, Rifaximin	Continuous / supportive
Ascites Therapy	Fluid removal & prevent recurrence	Spironolactone + Furosemide, Albumin, Midodrine	Maintain weight loss ≤500 g/day
Hepatic Encephalopathy	Reduce ammonia levels	Lactulose, Rifaximin, Hepa-Merz	Long-term, diet control
Hepatorenal Syndrome	Restore renal perfusion	Terlipressin, Norepinephrine, Albumin	Until improvement; consider transplant
Advanced Stage	Definitive treatment	Liver Transplantation	Curative option

Hepatocellular damage, inflammation, and fibrogenesis interact intricately in the pathophysiology of cirrhosis. Normally quiescent hepatic stellate cells (HSCs) are activated in response to liver damage and change into cells that resemble myofibroblasts and generate extracellular matrix (ECM) components, primarily collagen types I and III [4].

Fibrous septa and nodular regeneration are the results of ongoing hepatocyte necrosis and regeneration. Portal hypertension is brought on by the sinusoidal architecture's deformation, which raises intrahepatic vascular resistance.

Hepatocellular hypoxia and dysfunction are further aggravated by impaired sinusoidal capillarization and hepatic microcirculation.

Tumor necrosis factor-alpha (TNF-α), platelet-derived growth factor (PDGF), and transforming growth factor-beta (TGF-β) are cytokines that are essential for activating HSCs and sustaining fibrosis [5]. The liver's capacity to detoxify, metabolize, and synthesize is diminished when fibrous tissue gradually replaces functioning hepatocytes.

The stage and underlying etiology of cirrhosis affect how the disease manifests clinically. Typically, it is divided into stages that are compensated and those that are not.

Frequently asymptomatic or exhibiting vague symptoms including weakness, anorexia, weight loss, and pain in the right upper quadrant.

Hepatomegaly and moderate splenomegaly are possible physical findings. Complications include jaundice, ascites, variceal hemorrhage, and hepatic encephalopathy are hallmarks.

Testicular shrinkage, gynecomastia, palmar erythema, and spider angiomas are indications of hormonal imbalance brought on by a reduction in the liver's ability to metabolize estrogens.

Portal hypertension and hypoalbuminemia cause edema and ascites.

As a result of ammonia and other neurotoxins building up, encephalopathy presents as asterixis, confusion, and disorientation.

A mix of clinical, laboratory, imaging, and histological results are used to make the diagnosis. Increased levels of gamma-glutamyl transferase (GGT), alkaline phosphatase, aminotransferases (ALT, AST), and serum bilirubin.

Prolonged prothrombin time (PT) and decreased serum albumin levels. The main diagnostic method is ultrasound, which might reveal splenomegaly, nodularity, and an uneven liver surface. Hypersplenism-induced thrombocytopenia and leukopenia. MRI or CT scans can identify hepatocellular carcinoma (HCC) and offer a thorough structural evaluation. Liver stiffness can be noninvasively assessed using transient elastography (FibroScan). Liver biopsy, which shows widespread fibrosis, regenerating nodules, and architectural deformation, is still the gold standard for cirrhosis confirmation. APRI (AST to Platelet Ratio Index) and FIB-4 score are two noninvasive fibrosis indicators that are helpful in determining the severity of fibrosis.

CONCLUSION

Liver cirrhosis continues to be a major worldwide health problem due to its high death rate and financial impact. To avoid decompensation and increase survival, early risk factor identification, precise staging, and all-encompassing management techniques are crucial. Patient outcomes have been significantly enhanced by developments in liver transplantation, noninvasive diagnostics, and antiviral medication. There is optimism for future curative treatments due to ongoing research into antifibrotic medicines.

REFERENCES

1. World Health Organization. Global Health Estimates 2023: Disease Burden by Cause, Age, Sex, by Country and by Region, 2000–2022. Geneva: WHO; 2023.
2. European Association for the Study of the Liver (EASL). EASL Clinical Practice Guidelines: Management of Chronic Hepatitis B and C. J Hepatol. 2020;73(1):117–144.
3. Gao B, Bataller R. Alcoholic liver disease: Pathogenesis and new therapeutic targets. Gastroenterology. 2011;141(5):1572–1585.
4. Friedman SL. Mechanisms of hepatic fibrogenesis. Gastroenterology. 2008;134(6):1655–1669.
5. Kisseleva T, Brenner DA. Mechanisms of fibrogenesis. Exp Biol Med. 2008;233(2):109–122.
6. D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: A systematic review. J Hepatol. 2006;44(1):217–231.