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Review Article

A Comprehensive Review on The Complications of Decompensated Liver Disease

Shaik Irshad Nufshrun*, Dr. M. Sri Ramachandra, G. Kavya, Shaik Khalid

Dr. K. V. Subba Reddy Institute of Pharmacy, Dupadu, Pharm D VI Year.

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DCLD: Decompensated Chronic Liver Disease, CLD: Chronic Liver Disease, HE: Hepatic Encephalopathy, PH: Portal Hypertension, HRS-AKI: Hepatorenal Syndrome-Acute Kidney Injury, EVL: Esophageal Variceal Ligation, SBP: Spontaneous Bacterial Peritonitis, MELD: Model for End-Stage Liver Disease, CP: Child-Pugh Score, NSBB: Non-Selective Beta Blockers, PT/INR: Prothrombin Time / International Normalized Ratio
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ABSTRACT

Decompensated Chronic Liver Disease (DCLD) signifies the advanced stage of cirrhosis, characterized by the emergence of critical complications such as ascites, hepatic encephalopathy, variceal bleeding, and hepatorenal syndrome (HRS). This review aims to provide an in-depth overview of DCLD, including its epidemiology, underlying pathophysiology, diagnostic approaches, risk factors, and current management strategies. Cirrhosis results from persistent liver injury that leads to fibrosis and irreversible architectural damage. It is a global health concern, with increasing prevalence in developing countries like India, largely due to rising rates of alcohol use, obesity, and viral hepatitis. The transition from compensated to decompensated cirrhosis marks a significant decline in survival outcomes. The main causes include alcohol-related liver disease, viral hepatitis (B and C), NAFLD, and various autoimmune and genetic disorders. Diagnosis is based on clinical features, lab investigations, imaging studies, and prognostic scoring systems such as Child-Pugh and MELD. Management is multidisciplinary and includes interventions for complications such as portal hypertension, ascites, encephalopathy, and renal failure. Liver transplantation remains the definitive treatment. Early detection and targeted therapy can substantially improve clinical outcomes and reduce disease burden.

INTRODUCTION

Cirrhosis is a progressive liver condition where healthy hepatic tissue is replaced by fibrotic scar tissue, ultimately impairing liver function. The

***Corresponding Author:** Shaik Irshad Nufshrun
Address: *Dr. K. V. Subba Reddy Institute of Pharmacy, Dupadu, Pharm D VI Year.*
Email ✉: irshaddadsgirl@gmail.com
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liver loses its ability to perform vital roles such as metabolism, detoxification, and protein synthesis. Persistent liver injury from alcohol, viruses, or metabolic disorders leads to structural remodeling and irreversible damage.

Key Features of Cirrhosis

Fibrosis and Scarring: Chronic damage results in fibrous tissue accumulation.

Loss of Function: Normal liver processes are disrupted, including bile production and ammonia clearance.

Progressive Nature: Complications arise as liver function declines.

Stages if Cirrhosis

Compensated Stage: Patients remain asymptomatic without signs such as ascites, encephalopathy, or jaundice.

Decompensated Stage: Characterized by clinical manifestations including ascites, variceal bleeding, and encephalopathy. The onset of decompensation is associated with a significant decrease in survival.

Epidemiology

Cirrhosis represents a major global health challenge. Its prevalence ranges from 4.5% to 9.5% in the general population. In 2017, it accounted for over 1.3 million deaths worldwide. In countries like India, increasing alcohol use, viral hepatitis, and obesity have contributed to the rising incidence of cirrhosis.

Etiologies Of Cirrhosis

The primary causes of cirrhosis include:

Alcoholic Liver Disease

Non-Alcoholic Fatty Liver Disease (NAFLD)

Hepatitis B and C Infections

Autoimmune Liver Disorders

Metabolic and Genetic Disorders (e.g., Wilson's disease, hemochromatosis)

Iatrogenic and Toxic Injuries (e.g., certain medications, arsenic)

Other categories:

Viral: HBV, HCV, HDV

Autoimmune: Autoimmune hepatitis, primary biliary cholangitis

Metabolic: NAFLD, NASH, alpha-1 antitrypsin deficiency

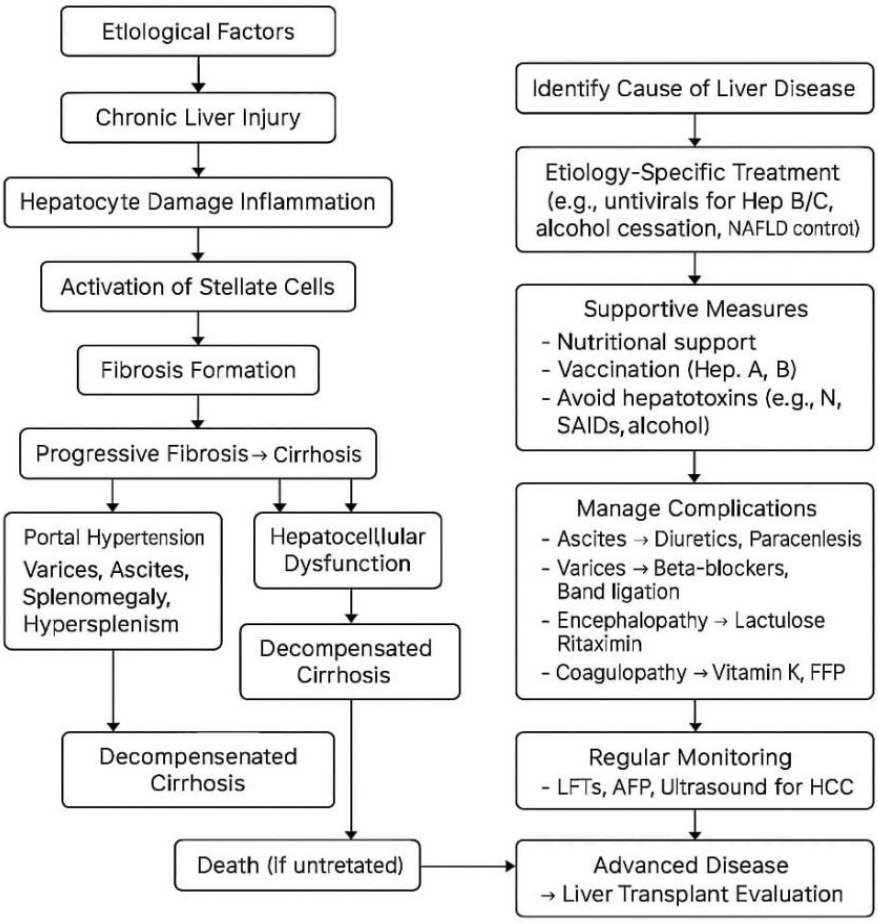
Vascular: Budd-Chiari syndrome

Genetic: Cystic fibrosis

Toxic: Alcohol, drugs

Pathophysiology

The development of cirrhosis involves persistent hepatic inflammation, regeneration of hepatocytes, and fibrotic remodeling. As intrahepatic resistance rises, portal hypertension develops. This leads to splanchnic vasodilation, systemic circulatory changes, and multi-organ dysfunction in advanced stages.



Risk Factors

- Chronic alcohol use
- Chronic hepatitis B/C infections
- NAFLD/NASH
- Autoimmune liver diseases
- Metabolic syndrome and obesity
- Genetic liver disorders
- Exposure to hepatotoxic drugs or toxins
- Age and male sex
- Sedentary lifestyle

DIAGNOSIS OF DCLD

Clinical Evaluation

History: Focuses on alcohol use, hepatitis exposure, comorbidities

Physical Examination: Includes assessment for jaundice, ascites, mental status changes

Laboratory Tests

LFTs: Bilirubin, albumin, PT/INR

CBC: To detect thrombocytopenia

Electrolytes: Monitoring for hyponatremia

Imaging

Ultrasound/CT/MRI: Evaluate liver morphology and ascites

Elastography (FibroScan): Assesses liver stiffness

Scoring Systems

Child-Pugh Score: Classifies severity based on labs and clinical features

MELD/MELD-Na Score: Predicts mortality using bilirubin, INR, creatinine, and sodium

Management Strategies

Portal Hypertension

Assessed via HVPG; treatment includes NSBBs (e.g., propranolol, carvedilol), somatostatin analogues, and vasopressin to reduce portal flow.

Esophageal Varices

- Surveillance endoscopy based on risk.
- Primary prophylaxis with NSBBs or EVL.
- Acute bleeding managed with vasoactive drugs (terlipressin), endoscopic band ligation, blood transfusion, and antibiotics (ceftriaxone).

Ascites

- Sodium restriction and diuretics (spironolactone ± furosemide).
- Large-volume paracentesis with albumin replacement if >5L removed.
- Refractory cases may require TIPS.

Hepatic Encephalopathy

- Caused by hyperammonemia.
- Treated with lactulose (acidifies colon, traps ammonia), rifaximin (reduces ammonia-producing bacteria), and adjuncts like LOLA or sodium benzoate.

Hepatorenal Syndrome

- Result of splanchnic vasodilation and renal hypoperfusion.
- Managed with vasoconstrictors (terlipressin, norepinephrine) and volume expansion.
- Liver transplantation is the only definitive cure.

CONCLUSION

Decompensated Chronic Liver Disease represents a critical stage in the progression of cirrhosis, marked by life-threatening complications. Its onset significantly worsens prognosis and increases healthcare burden globally. Early identification and prompt management of complications like ascites, variceal bleeding, encephalopathy, and hepatorenal syndrome are crucial for improving patient outcomes. A multidisciplinary approach combining medical therapy, nutritional support, and consideration for liver transplantation is essential. Preventive strategies targeting alcohol use, obesity, and hepatitis can reduce disease incidence. Prognostic scoring systems such as MELD and Child-Pugh aid in risk stratification and treatment planning. Despite advancements, DCLD remains a major cause of mortality, especially in developing countries. Public health initiatives and patient education are vital for early intervention. Continued research is needed to refine therapeutic strategies and optimize transplant allocation. Ultimately, improving awareness, access, and early care can significantly reduce the burden of advanced liver disease.

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