LIVER CIRRHOSIS: STRUCTURAL AND FUNCTIONAL CHANGES – A SCIENTIFIC REVIEW

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Abstract

Liver cirrhosis is a chronic, progressive disease characterized by extensive fibrosis and the formation of regenerative nodules, which leads to significant alteration of liver structure and function. This article presents a detailed scientific analysis of the histological, vascular, and functional changes that occur in cirrhotic liver tissue. The structural remodeling of hepatic lobules and vascular architecture is strongly associated with impaired metabolic, synthetic, and detoxification functions of the liver. Understanding these changes is crucial for effective diagnosis, management, and research in hepatology.

Keywords: liver cirrhosis, fibrosis, hepatic structure, functional impairment, portal hypertension.

Introduction

Liver cirrhosis represents the late stage of chronic liver disease resulting from long-term liver injury due to factors such as chronic viral hepatitis, alcohol abuse, or non-alcoholic steatohepatitis. In cirrhosis, normal liver tissue is progressively

replaced by fibrotic scar tissue and regenerative nodules, leading to a distorted organ architecture and compromised function. The changes in liver structure have major implications on its metabolic, endocrine, and detoxification roles.

Normal Liver Structure and Function

The liver is the largest internal organ and performs essential physiological functions including metabolism of nutrients, synthesis of plasma proteins, detoxification of harmful substances, and production of bile. Histologically, the liver is organized into functional units called lobules, consisting of hepatocytes arranged around a central vein, with sinusoids permitting blood flow from the portal triads to the central vein. Kupffer cells and stellate cells play roles in immune defense and extracellular matrix homeostasis. Dual blood supply from the hepatic artery and portal vein supports both oxygen delivery and nutrient processing. [1]

Pathogenesis of Cirrhosis: Structural Change

Fibrosis and Nodule Formation

Cirrhosis begins with chronic injury that activates hepatic stellate cells, leading to excessive deposition of extracellular matrix (ECM) proteins, predominantly collagen. The ECM accumulation disrupts the normal lobular architecture, resulting in the formation of fibrous septa and regenerative nodules. These nodules are composed of proliferating hepatocytes surrounded by bands of fibrotic tissue, which effectively replace functional parenchyma, impairing liver function and blood flow. [2]

Vascular Remodeling and Portal Hypertension

Structural remodeling in cirrhosis also affects liver vasculature. Fibrosis compresses intrahepatic blood vessels, increasing resistance to blood flow. This

leads to elevated pressure in the portal vein (portal hypertension), which can cause splenomegaly, varices, and ascites. Advanced imaging techniques such as four-dimensional flow MRI reveal altered portal hemodynamics associated with morphological changes in cirrhosis. [2]

Functional Changes in Cirrhotic Liver

Metabolic Dysfunction

As normal hepatocytes are replaced by fibrotic tissue, metabolic functions deteriorate. Cirrhosis affects carbohydrate, lipid, and protein metabolism. Reduced glycogen storage and impaired gluconeogenesis can lead to fasting intolerance and hypoglycemia. Protein synthesis, including albumin production, becomes insufficient, contributing to edema and ascites due to decreased oncotic pressure.

Detoxification Impairment

The liver's ability to metabolize drugs, hormones, and toxins declines in cirrhosis. Biliary secretion is disrupted due to structural distortion of bile canaliculi and ductules, leading to cholestasis and accumulation of toxic substances. Impaired detoxification elevates the risk of hepatic encephalopathy and drug toxicity.

Synthetic Function Decline

Synthesis of key plasma proteins (e.g., albumin, clotting factors) declines with disease progression, resulting in coagulopathy and hypoalbuminemia. These changes contribute to bleeding tendencies and fluid imbalance.

Extrahepatic Organ Changes

Cirrhosis also influences the function of other organs. Recent studies show that patients with cirrhosis can develop structural and functional changes in the heart, such as myocardial extracellular volume expansion and altered systolic function, a condition often referred to as cirrhotic cardiomyopathy. [3]

Clinical Implications

Structural changes in a cirrhotic liver are correlated with progressive functional impairment, leading to clinical manifestations such as jaundice, coagulopathy, ascites, and hepatic encephalopathy. Early detection and staging of cirrhosis through biopsy or non-invasive imaging are essential in clinical practice for appropriate management and monitoring progression.

Conclusion

Liver cirrhosis involves profound structural and functional transformations. Fibrosis and regenerative nodule formation disrupt normal lobular architecture and compromise hepatic functions, including metabolism, detoxification, and synthesis. These changes contribute to serious clinical complications. Ongoing research aims to deepen our understanding of the pathophysiological mechanisms and improve diagnostic and therapeutic strategies.

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