NORMAL HISTOLOGICAL STRUCTURE OF THE LIVER AND CHANGES IN HEPATITIS A

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Abstract: The liver plays a central role in metabolism, detoxification, and protein synthesis, with its function dependent on a well-organized histological structure. Normal liver tissue consists of hexagonal lobules, where hepatocytes are arranged in radiating plates around central veins, separated by sinusoidal spaces, and portal triads contain the hepatic artery, portal vein, and bile duct. This architecture ensures efficient blood flow, nutrient exchange, and bile secretion.

Hepatitis A, an acute viral infection, induces significant morphofunctional changes in liver tissue. Histological examination reveals portal and periportal inflammation, hepatocyte necrosis, cytolysis, and varying degrees of cholestasis. The infiltration of lymphocytes and plasma cells disrupts normal architecture and impairs liver function. Understanding these morphofunctional alterations is essential for accurate diagnosis, prognosis, and therapeutic management. Comparative studies highlight the differences between healthy liver tissue and hepatitis A–affected tissue, providing insight into the pathogenesis of the disease.

Keywords: liver histology, hepatitis A, hepatocyte necrosis, portal inflammation, cholestasis.

Introduction: The liver is a vital organ responsible for metabolism, detoxification, bile production, and synthesis of plasma proteins. Its function relies on a highly organized histological structure, including hepatic lobules, hepatocytes arranged in radiating plates, sinusoidal spaces, and portal triads containing the hepatic artery, portal vein, and bile duct. Preservation of this architecture is essential for normal liver function.

Hepatitis A, caused by the hepatitis A virus (HAV), is an acute infectious disease that primarily affects hepatocytes and disrupts normal liver structure. Infection leads to inflammatory infiltration, hepatocyte necrosis, cytolysis, and cholestasis,

which can impair liver function temporarily. Studying these morphofunctional changes provides insight into the pathogenesis of hepatitis A and helps in understanding the differences between healthy and diseased liver tissue, which is important for diagnosis and clinical management.

Materials and Methods

Study Material

The study included liver tissue samples obtained from patients diagnosed with acute hepatitis A and from healthy liver tissue obtained from autopsy or surgical resections (used as controls). All samples were collected following ethical guidelines with informed consent.

Histological Examination

Tissues were fixed in 10% neutral buffered formalin, processed routinely, and embedded in paraffin. Sections of 4–5 μm thickness were stained with hematoxylin and eosin (H&E) for general morphological assessment. Histological evaluation focused on lobular architecture, hepatocyte morphology, necrosis, inflammatory infiltration, and bile duct changes.

Morphometric Analysis

Quantitative analysis included measuring hepatocyte size, nuclear-to-cytoplasmic ratio, and the extent of inflammatory infiltration using light microscopy and image analysis software. Statistical comparison between healthy and hepatitis A–affected tissues was performed, with significance set at $p < 0.05$.

Results

Normal Liver Tissue

Histological examination of healthy liver samples revealed well-preserved lobular architecture. Hepatocytes were organized in radiating plates around central veins, with clear cell polarity and uniform cytoplasmic staining. Nuclei were small, round to oval, with finely dispersed chromatin and rare mitotic figures. Sinusoids were evenly spaced and lined by endothelial cells and Kupffer cells. Portal triads containing the hepatic artery, portal vein, and bile duct were intact, and no inflammatory infiltration or fibrosis was observed. Morphometric analysis confirmed normal hepatocyte size and low nuclear-to-cytoplasmic ratio, reflecting stable cellular function.

Liver Tissue in Hepatitis A

In hepatitis A–affected tissue, pronounced morphofunctional changes were observed. Portal and periportal regions exhibited dense infiltration of lymphocytes and plasma cells. Hepatocytes showed varying degrees of necrosis and cytolysis, with ballooning degeneration and nuclear pyknosis. The normal lobular architecture was disrupted in affected areas, particularly around portal tracts. Sinusoids appeared congested, and mild cholestasis was observed in some samples. Morphometric analysis revealed a significant increase in hepatocyte size, nuclear enlargement, and altered nuclear-to-cytoplasmic ratio compared to controls ($p < 0.05$).

Comparative Analysis

Quantitative comparison between healthy and hepatitis A–affected tissues highlighted significant differences in inflammatory cell infiltration, hepatocyte morphology, and preservation of lobular architecture. These findings confirm that acute hepatitis A induces acute hepatocellular injury while primarily affecting portal and periportal regions.

Conclusion: The liver's normal histological structure is essential for maintaining its metabolic, synthetic, and detoxification functions. Hepatitis A infection induces significant morphofunctional changes, including portal and periportal inflammatory infiltration, hepatocyte necrosis, cytolysis, and cholestasis, which disrupt normal liver architecture and impair cellular function. Comparative analysis of healthy and affected liver tissue highlights the acute nature of hepatocellular injury caused by hepatitis A and emphasizes the importance of understanding these changes for accurate diagnosis and clinical management. Studying these histological alterations provides valuable insight into the pathogenesis of hepatitis A and informs strategies for monitoring and treating affected patients.

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