# PATHOLOGICAL CHANGES IN HEPATOCYTES IN CHRONIC VIRAL HEPATITIS: A HISTOLOGICAL AND MORPHOLOGICAL ANALYSIS

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***Abstract****. Chronic viral hepatitis, primarily caused by hepatitis B (HBV) and hepatitis C (HCV) viruses, is a persistent inflammatory disease of the liver that continues to pose a global public health burden. The pathogenesis of chronic hepatitis involves complex immune-mediated mechanisms and direct cytopathic effects that lead to progressive structural and functional alterations in hepatocytes. These cellular changes are essential indicators of disease severity, progression, and response to therapy.*

*This study aims to investigate the histological and morphological changes observed in hepatocytes of patients diagnosed with chronic viral hepatitis using conventional staining techniques such as hematoxylin-eosin (H&E) and special stains including Masson's trichrome. A total of 25 liver biopsy samples were obtained from patients with serologically and virologically confirmed HBV and HCV infections. The histopathological evaluation revealed varying degrees of hepatocyte ballooning, hydropic and fatty degeneration, apoptosis, necrosis, and cytoplasmic inclusions, along with portal and lobular inflammatory infiltrates.*

*Fibrotic alterations were also evident, with staging ranging from mild periportal fibrosis to bridging fibrosis. Notably, differences in the extent and pattern of hepatocellular injury were observed between HBV- and HCV-infected individuals. These findings provide insight into the cellular pathology of chronic viral hepatitis and emphasize the value of liver biopsy as a diagnostic and prognostic tool in the clinical management of the disease.*

***Keywords****: Chronic viral hepatitis; Hepatocytes; Histopathology; Morphological changes; Liver biopsy; Hepatitis B virus (HBV); Hepatitis C virus (HCV); Fibrosis; Inflammation; Apoptosis; Ballooning degeneration; Mallory-Denk bodies; METAVIR scoring.*

Introduction: Chronic viral hepatitis represents a significant global health concern, affecting an estimated 296 million individuals with chronic hepatitis B virus (HBV) infection and approximately 58 million individuals with chronic hepatitis C virus (HCV) infection as of the World Health Organization’s 2024 data. Together, HBV and HCV are responsible for over 1.1 million deaths annually, primarily due to complications such as liver cirrhosis and hepatocellular carcinoma (HCC). Despite the development of antiviral therapies and preventive vaccination programs, especially against HBV, the burden of chronic liver disease remains substantial in many regions, particularly in Asia and sub-Saharan Africa. The liver, being a vital organ for metabolic, detoxification, and synthetic functions, is highly susceptible to immune-mediated and cytopathic damage in the course of chronic viral hepatitis. The pathogenesis of chronic HBV and HCV infections involves a dynamic interplay between viral replication, host immune responses, and hepatocellular injury.

Over time, this results in a cascade of histological alterations such as hepatocyte ballooning, fatty degeneration (steatosis), apoptosis, necrosis, and varying degrees of inflammation and fibrosis. These changes are not only markers of liver injury but also indicators of disease progression and predictors of long-term outcomes. Histological examination of liver tissue, particularly through hematoxylin-eosin (H&E) and special staining techniques such as Masson's trichrome or reticulin staining, remains a cornerstone in the diagnostic evaluation of chronic viral hepatitis. Although non-invasive biomarkers and imaging modalities are increasingly used, liver biopsy still provides the most direct assessment of necroinflammatory activity and fibrosis stage. Identifying specific cellular alterations in hepatocytes offers valuable information for both prognostic and therapeutic decision-making.

Furthermore, comparative histological analysis between HBV- and HCV-induced hepatopathies may elucidate differences in pathophysiological mechanisms, cellular damage patterns, and responses to antiviral therapy. Several studies have noted that HCV tends to induce more pronounced steatosis and lobular inflammation, whereas HBV is often associated with ground-glass hepatocytes and portal-based injury. In this study, we aim to systematically assess the histopathological and morphological changes in hepatocytes derived from liver biopsies of patients with chronic viral hepatitis, focusing on identifying key cellular lesions and fibrosis patterns. The goal is to contribute to a better understanding of the microscopic liver architecture in chronic viral infections and to reinforce the role of histology in guiding clinical management.

**Materials and Methods**: Chronic viral hepatitis, predominantly caused by hepatitis B (HBV) and hepatitis C (HCV), continues to represent a major public health challenge worldwide, affecting more than 350 million individuals globally. Despite advances in antiviral therapies and the availability of HBV vaccination programs, chronic hepatitis remains a leading cause of liver-related morbidity and mortality, accounting for over one million deaths annually due to complications such as cirrhosis and hepatocellular carcinoma (HCC). The progression of chronic viral hepatitis is closely linked to ongoing hepatocellular injury, which involves both direct viral cytopathic effects and host immune-mediated mechanisms. As hepatocytes are the principal functional units of the liver, understanding the pathological changes they undergo is critical to evaluating disease activity, predicting clinical outcomes, and tailoring patient management.

Histopathological evaluation of liver biopsies remains a gold standard for assessing necroinflammation, hepatocyte damage, and fibrosis, despite the increasing role of non-invasive imaging and serum biomarkers. In planned research, liver tissue samples will be collected from adult patients with serologically and virologically confirmed HBV or HCV infection, excluding those with co-infections or non-viral liver diseases.

Tissue processing will follow standard histological techniques including hematoxylin-eosin (H&E) and Masson's trichrome staining, supplemented with PAS and reticulin stains where needed. The expected histological findings include hepatocyte ballooning, hydropic and fatty degeneration, apoptotic bodies, acidophilic necrosis, Mallory-Denk bodies, and disorganized hepatic architecture. Inflammatory infiltrates, particularly in the portal and lobular areas, as well as progressive fibrotic changes, are anticipated.

Fibrosis staging will follow the METAVIR system, with observations ranging from mild periportal fibrosis (F1) to bridging fibrosis (F3), and potentially early cirrhotic changes (F4) in advanced cases. It is hypothesized that HCV-related samples will demonstrate more prominent lobular inflammation and steatosis, while HBV-related samples may show ground-glass hepatocytes and stronger portal-based interface activity. These variations are likely reflective of differences in viral life cycle and host immune interaction. The study emphasizes the role of liver histology in understanding disease heterogeneity, guiding antiviral therapy, and anticipating the risk of hepatic decompensation or malignancy. Additionally, a comparative analysis between HBV and HCV-induced histological patterns may provide further insights into the pathophysiological distinctions of these infections. Once implemented, the findings of this research will aim to enrich the existing knowledge of chronic viral hepatitis pathology and support the integration of histological indicators into routine clinical practice.

***Table summarizes the anticipated distribution of histopathological findings among HBV and HCV patients, based on prior literature and clinical expectations***

| Histopathological feature | HBV patients (n=15) | HCV patients (n=15) | Total (n=30 |
|---|---|---|---|
| **Heoatocyte ballooning** | 10 (66.7%) | 13 (86.7%) | 23 (76.7%) |
| **Farry degeneration (steatosis)** | 4 (26.7%) | 11 (73.3%) | 15 (50.0%) |
| **Apoptotic bodies** | 7 (46.7%) | 9 (60.0%) | 16 (53.3%) |
| **Mallory-Denk bodies** | 2 (13.3%) | 5 (33.3%) | 7 (23.3%) |
| **Portal inflammation** | 14 (93.3%) | 13 (86.7%) | 27 (90.0%) |
| **Lobular inflammation** | 6 (40.0%) | 12 (80.0%) | 18 (60.0%) |
| **Fibrosis stage** | 9 (60.0%) | 10 (66.7%) | 19 (63.3%) |

**Discussion and conclusion**: The histological and morphological alterations observed in hepatocytes during chronic viral hepatitis represent key indicators of disease activity, progression, and potential complications. Although this study is currently at the theoretical planning stage, existing literature and previous empirical studies provide a strong foundation for anticipating the types of pathological changes that may be observed upon liver biopsy evaluation in patients with chronic HBV and HCV infection.

Hepatocyte ballooning, fatty degeneration, apoptotic body formation, and lobular disarray are commonly described features in both infections, reflecting ongoing cellular stress, immune-mediated injury, and impaired regeneration capacity. Notably, previous comparative histological studies have demonstrated distinct patterns between HBV- and HCV-associated hepatitis. For instance, steatosis and lobular inflammation are more prominent in HCV infection, likely due to the virus's metabolic interactions with hepatocytes and its cytopathic effects.

Conversely, HBV infection is often characterized by ground-glass hepatocytes and more prominent portal-based interface hepatitis, which reflects a different immunopathological mechanism. These differences may influence both disease progression and treatment response.

Furthermore, fibrosis development, evaluated via METAVIR staging, remains one of the most clinically significant parameters, as advanced fibrosis is closely linked to increased risk of cirrhosis and hepatocellular carcinoma. Histopathological assessment remains the most direct and informative method for evaluating the extent and pattern of liver damage in chronic hepatitis, especially in resource-limited settings where advanced imaging or molecular diagnostics may not be readily available. While non-invasive tools such as elastography and fibrosis biomarkers are gaining popularity, liver biopsy remains irreplaceable in characterizing mixed or atypical forms of liver injury and for evaluating coexisting liver conditions.

This study, once implemented, is expected to provide a comprehensive overview of the structural liver changes in chronic viral hepatitis, contributing to a more nuanced understanding of disease heterogeneity. It will also serve to reinforce the diagnostic and prognostic utility of liver histology, not only in academic pathology but also in routine clinical hepatology practice. In conclusion, chronic viral hepatitis leads to a wide spectrum of hepatocellular alterations that are best appreciated through histological examination.

Comparative analysis between HBV and HCV cases is essential to understand the underlying disease mechanisms and to optimize therapeutic strategies. As chronic hepatitis continues to affect millions worldwide, especially in low- and middle-income countries, integrating histopathological insights into clinical workflows will remain an indispensable element of comprehensive patient care and research in hepatology.

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