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Microscopic Alterations in Hepatitis

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ABSTRACT

Hepatitis refers to inflammation of the liver that can arise from infectious and non-infectious etiologies. Each cause may produce a distinct microscopic signature, although some histological patterns overlap. Examination of hepatitis A, B, and C, autoimmune hepatitis (AIH), drug-induced liver injury (DILI), alcohol-associated hepatitis, and metabolic dysfunction-associated steatohepatitis (MASH) reveals several recurring histological features. Portal and lobular inflammation, interface hepatitis, hepatocyte ballooning, cholestasis, lymphoid aggregates, plasma cell-rich infiltrates, ground-glass hepatocytes, giant-cell transformation, fibrosis, Kupffer cell hyperplasia, hydropic change, Councilman bodies (apoptotic hepatocytes), and steatosis may occur in different combinations across acute and chronic hepatitis and related hepatitis conditions. Giant-cell transformation is uncommon and non-specific, while sinusoidal dilatation may occur in selected vascular injury patterns. Persistent interface activity accompanied by lobular necrosis tends to be associated with higher activity grades, whereas fibrosis staging reflects the extent of structural remodeling. Standard systems, including Batts–Ludwig, Ishak, Knodell, and METAVIR, remain established approaches for describing histological activity and fibrosis. In routine histopathological practice, biopsy interpretation is most informative when the necroinflammatory pattern, activity grade, and fibrosis stage are considered together. This review discusses microscopic alterations in viral hepatitis, AIH, and DILI, together with the assessment of disease severity using the Batts–Ludwig, Ishak, Knodell, and METAVIR systems.

Keywords: liver disease; hepatitis; microscopic change; hepatitis grading; hepatitis staging

INTRODUCTION

Hepatitis is a tissue response in which hepatocytes and the surrounding hepatic compartments become inflamed or injured. The initiating cause may be viral infection, an autoimmune process, medication exposure, alcohol-related injury, or metabolic dysfunction [1].

Although these conditions have different causes, their biopsy appearances may overlap. A systematic examination therefore considers the distribution of injury and the dominant microscopic pattern, with attention to hepatocytes, portal tracts, lobules, sinusoids, bile ducts, and fibrosis [2,3,4]. The findings must be interpreted alongside the clinical history and laboratory results rather than as isolated diagnostic markers [3].

The distinction between acute and chronic disease is important when describing the tissue response. Hepatitis A typically resolves on its own after an acute illness and does not progress to chronic infection [5]. Hepatitis B may persist and produce chronic inflammatory activity with variable fibrosis [6], while chronic hepatitis C may show portal and lobular inflammation with progressive structural injury [7].

Autoimmune hepatitis (AIH) is a chronic immune-mediated disease, although its initial presentation may be acute and dominated by lobular injury [8,9]. Drug-induced liver injury (DILI) may produce hepatocellular, cholestasis, or mixed microscopic patterns [10]. Alcohol-associated hepatitis has a steatohepatitis pattern that may include neutrophilic inflammation [11]. The hallmark features of metabolic dysfunction-associated steatohepatitis are fatty change, ballooned hepatocytes, and inflammation within the lobules [12,13,14].

This review describes the principal microscopic alterations found in hepatitis and related hepatitis conditions, with emphasis on acute hepatitis A and chronic hepatitis B, hepatitis C, and AIH. Drug-induced liver injury, alcohol-associated hepatitis, and metabolic dysfunction-associated steatohepatitis are also discussed as conditions that may produce overlapping hepatitis patterns. Grading and staging are mentioned only as supporting descriptors of inflammatory activity and fibrosis [1].

1. Acute Hepatitis

Acute hepatitis commonly produces lobular inflammation accompanied by hepatocyte swelling, hydropic change, apoptosis, and variable necrosis. Hydropic change describes cytoplasmic swelling and rarefaction in injured hepatocytes [1,15,16]. Necrosis may be spotty, confluent, bridging, zonal, or extensive, depending on the severity and cause of injury [15,16,17].

Apoptotic hepatocytes may appear as shrunken, eosinophilic bodies within lobules or sinusoids, commonly termed Councilman bodies. Kupffer cells may become activated and more prominent in response to hepatocellular injury [1,5,16].

1.1 *Hepatitis A*

Hepatitis A virus infection is generally an acute, self-limited disease and is not associated with persistent viral infection. When a biopsy is obtained, the tissue may show hepatocyte swelling, lobular disarray, apoptotic bodies, focal necrosis, and inflammatory cells within the lobules and portal tracts [5]. The interpretation of acute hepatitis patterns should also account for the clinical setting and the possibility of overlapping histological appearances [17].

Portal inflammation may be conspicuous, and activated Kupffer cells or macrophages may be present within the parenchyma. Canalicular cholestasis can also occur, occasionally persisting after the peak of hepatocellular injury [5]. Histological patterns in other acute viral infections, including hepatitis E, may vary with immune status and pre-existing liver disease [18].

Routine hematoxylin and eosin (HE) staining demonstrates the hepatocellular and inflammatory changes. Fibrosis is not a usual feature of uncomplicated acute hepatitis A [1,5].

2. Chronic Hepatitis

Chronic hepatitis is characterized by persistent portal inflammation, variable interface activity, and lobular injury. The degree and distribution of fibrosis differ between patients and may increase with continuing inflammatory damage [1,19].

2.1 *Chronic Hepatitis B*

Chronic hepatitis B may show portal lymphocytic inflammation, variable interface activity, and spotty lobular inflammation. The extent of these changes differs among patients and across clinical phases of infection [20]. Current guidance emphasizes that assessment of chronic Hepatitis B should integrate virological and biochemical findings with the evaluation of liver disease severity [6].

Ground-glass hepatocytes are a recognizable feature in some cases and result from cytoplasmic accumulation of hepatitis B surface antigen. Their presence is a useful morphologic clue, although the extent of this finding varies among patients [20]. Histological findings have also been reported to differ according to hepatitis B e antigen status [21].

With ongoing injury, portal fibrosis may extend into periportal or bridging septa and, in advanced cases, produce cirrhotic architectural remodeling [20]. Liver biopsy can provide additional information about histological injury in patients whose clinical or virological profile does not clearly establish disease activity [22].

HE is used to assess the inflammatory pattern. Orcein staining or immunohistochemistry for hepatitis B surface antigen may be used when demonstration of viral antigen within hepatocytes is required [1,23].

2.2 *Chronic Hepatitis C*

Biopsies from chronic hepatitis C may show lymphoid aggregates or follicles in the portal tracts, along with interface activity and lobular inflammation of varying degree. Bile-duct injury, steatosis, and sinusoidal inflammatory infiltrates may also be present, although none of these findings is unique to hepatitis C [7].

With persistent disease, fibrosis may extend from portal expansion to bridging septa and cirrhosis in advanced cases [7]. A pattern-based review of liver biopsies also emphasizes the need to interpret portal and lobular findings together rather than relying on a single microscopic feature [24].

Routine HE sections demonstrate the inflammatory and hepatocellular changes, while a collagen stain such as Masson's trichrome may be used to clarify the extent of accompanying fibrosis [1,23].

2.3 *Autoimmune Hepatitis*

The usual chronic pattern of autoimmune hepatitis includes portal lymphoplasmacytic inflammation, often with plasma-cell clusters, together with interface hepatitis and variable lobular activity [8]. Acute presentations may instead show predominant lobular or centrilobular injury, sometimes with confluent necrosis [9]. Hepatocyte rosettes and emperipolesis may be observed, but they are not sufficiently specific to establish the diagnosis independently [25].

Histological findings should be interpreted together with autoantibodies, serum immunoglobulin levels, and the exclusion of alternative causes [26]. Current guidance also recognizes that the distribution and severity of inflammation may vary according to the timing and clinical presentation of the biopsy [27].

Recent histopathological reviews further emphasize that AIH may show different combinations of portal, interface, and lobular injury, and that these features can overlap with DILI or other chronic liver diseases [28,29]. Emerging diagnostic work continues to examine how histological findings can be integrated with clinical and laboratory information [30]. In follow-up biopsies, some inflammatory or hepatocellular changes may remain detectable despite treatment, so the tissue findings should be interpreted in the context of therapy and timing [31].

Giant-cell transformation is an uncommon histological pattern characterized by enlarged, multinucleated hepatocytes. It is not specific to a single cause and may occur in selected forms of hepatitis, including giant-cell hepatitis [1,17].

HE is used to evaluate the inflammatory pattern, and a connective-tissue stain can assist in the assessment of fibrosis when required [1,23]. Recent pathology reviews describe the broad histological spectrum of autoimmune hepatitis and the need to distinguish it from viral hepatitis, DILI, and coexisting metabolic liver disease [32].

3. Other Hepatitis Injury Patterns

3.1 *Drug-Induced Liver Injury*

Drug-induced liver injury (DILI) has a broad histological spectrum and may appear as hepatocellular, cholestasis, or mixed injury. Biopsies may show lobular inflammation, hepatocyte apoptosis or necrosis, interface activity, canalicular cholestasis, bile-duct injury, eosinophils, granulomas, or centrilobular damage, depending on the implicated agent [10]. The range of patterns may extend from acute extensive necrosis to resolving injury with pigment-laden macrophages [33].

Selected drug-related vascular injury patterns may also involve the hepatic sinusoids. Sinusoidal dilatation, sometimes accompanied by congestion, may occur in vascular-pattern liver injury and has also been reported in biopsies showing biliary-pattern injury; it is not a defining feature of routine hepatocellular hepatitis [1,34,35].

Some cases resemble viral or autoimmune hepatitis, whereas others are dominated by cholestasis or extensive hepatocyte necrosis [33]. Biopsy may help characterize the pattern of injury and distinguish DILI from competing diagnoses, including autoimmune hepatitis [34]. Because no single microscopic feature confirms drug causation, the findings must be considered with medication exposure, timing, and exclusion of alternative causes [10].

HE provides the principal pattern assessment, while additional stains are selected to investigate specific alternatives or clarify fibrosis rather than to identify a unique DILI marker [1,23]. The timing of biopsy may also influence the observed pattern, because acute hepatocellular or cholestasis features can become less conspicuous as the injury evolves [36]. When cholestasis injury predominates, bile pigment may accumulate within hepatocytes or canaliculi. Prolonged cholestasis may be accompanied by ductular reaction, portal inflammation, bile-duct injury, and progressive portal fibrosis [37,38]. Comparative histological studies of biliary-pattern injury also identify cholestasis and bile-duct or ductular changes in DILI [35].

3.2 *Alcohol-Associated Hepatitis*

Alcohol-associated hepatitis may show macrovesicular steatosis, swollen or ballooned hepatocytes, neutrophil-rich lobular inflammation, Mallory-Denk bodies, and cholestasis. The injury is often prominent around central veins, where pericellular or perisinusoidal fibrosis may also be present [11]. These findings occur within a broader spectrum of alcohol-associated liver disease that includes steatosis, steatohepatitis, and cirrhosis [39].

HE demonstrates the steatotic and inflammatory changes, while trichrome may help display the distribution of collagen when fibrosis is present [1,23]. When alcohol exposure coexists with cardiometabolic risk factors, the combined phenotype may be classified within the metabolic dysfunction and alcohol-related liver disease (MetALD) framework [40].

3.3 *Metabolic Dysfunction-Associated Steatohepatitis*

Metabolic dysfunction-associated steatohepatitis (MASH) is defined histologically characterized by the combination of steatosis, hepatocyte ballooning, and lobular inflammation. Fibrosis commonly begins in a perisinusoidal or pericellular distribution, particularly in zone 3, and may extend to portal areas, bridging septa, and cirrhosis in advanced disease [12,13,14].

The term MASH is used within the updated metabolic dysfunction-associated steatotic liver disease (MASLD) nomenclature, replacing the former NAFLD/NASH terminology [41]. Current European guidance describes MASLD as a spectrum that includes steatosis, steatohepatitis, fibrosis, and cirrhosis [12]. The 2024 Chinese guideline also identifies liver histology as a means of evaluating steatosis, ballooning, inflammation, and fibrosis [42].

AASLD guidance continues to recognize biopsy as a method for identifying steatohepatitis and evaluating competing causes when non-invasive assessment is insufficient [13]. More recent pathology consensus statements refine the description of steatosis, ballooning, inflammation, and fibrosis [43], while current reviews discuss emerging issues in the interpretation of these lesions [14].

4. Grading and Staging as Complementary Assessments

Grading summarizes the degree of active necroinflammatory injury observed in the biopsy, whereas staging describes the extent of fibrosis and architectural remodeling [1]. More extensive interface activity and lobular necrosis generally contribute to higher activity grades (Figure 1), while the fibrosis stage records the degree of structural remodeling (Figure 2) [1].

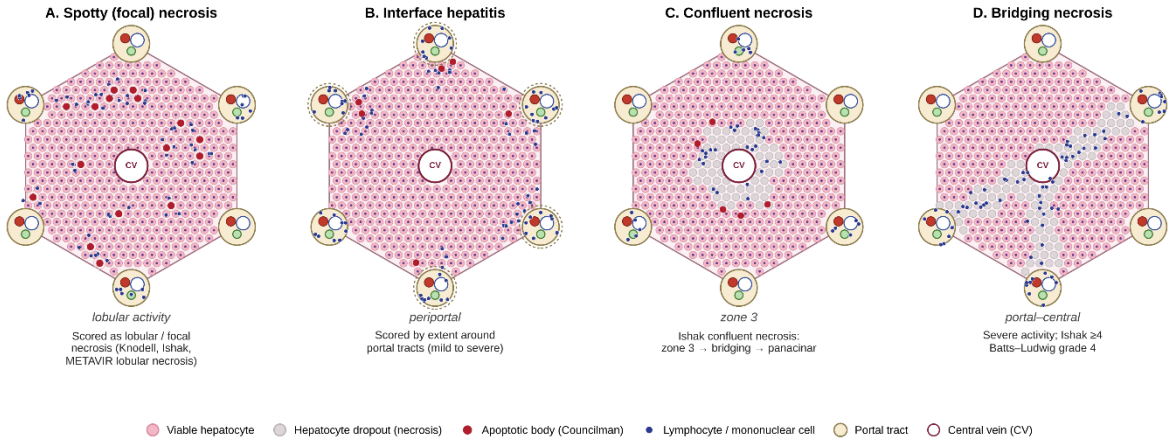


FIGURE 1: Schematic illustration of necroinflammatory injury patterns in a hepatic lobule (portal tracts at the corners, central vein [CV] in the center). (A) Spotty necrosis: scattered apoptotic bodies with small foci of lymphocytes. (B) Interface hepatitis: mononuclear cells cross the limiting plate around portal tracts with periportal hepatocyte injury. (C) Confluent necrosis: dropout of zone 3 hepatocytes around the central vein. (D) Bridging necrosis: confluent necrosis linking the central vein to portal tracts. Original conceptual drawing prepared for this review; it is not a photomicrograph. Source: figure generated with AI assistance (Claude, Anthropic; code-generated with Python/Matplotlib) and reviewed by the authors; conceptual content based on the descriptions of necroinflammatory activity and grading in [1].

Commonly used frameworks include the Batts–Ludwig, Ishak, Knodell, and METAVIR systems. These provide structured approaches to describing necroinflammatory activity and fibrosis, without replacing assessment of the dominant microscopic pattern or establishing the cause of hepatitis by themselves [1,2].

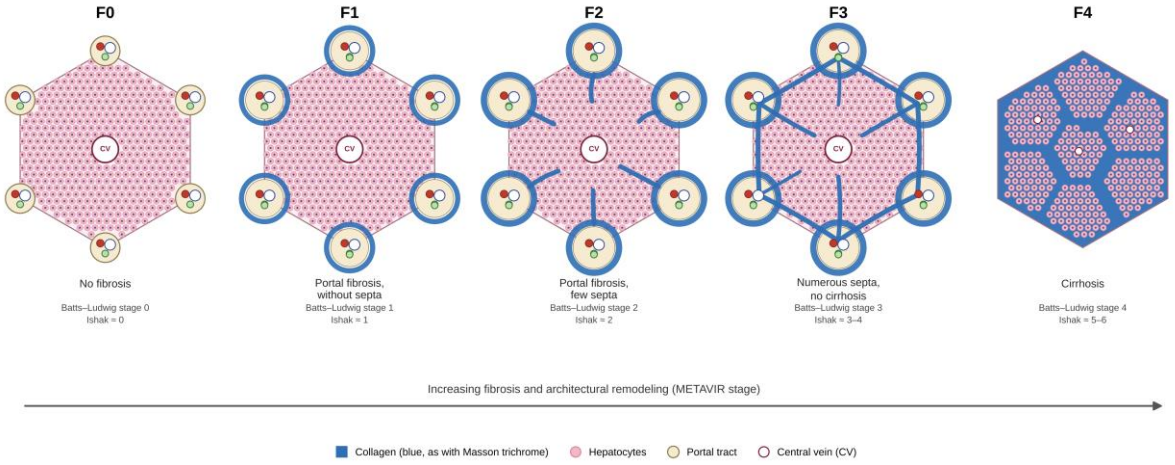


FIGURE 2: Schematic illustration of fibrosis staging. Collagen is shown in blue, as it appears with Masson trichrome. F0, no fibrosis; F1, portal fibrosis without septa; F2, portal fibrosis with few septa; F3, numerous septa (portal–portal and portal–central bridging) without cirrhosis; F4, cirrhosis with regenerative nodules surrounded by fibrous septa. METAVIR (F), Batts–Ludwig, and approximate Ishak stages are indicated. Original conceptual drawing prepared for this review. Source: figure generated with AI assistance (Claude, Anthropic; code-generated with Python/Matplotlib) and reviewed by the authors; staging categories based on the fibrosis staging systems described in [1,2].

CONCLUSION

Hepatitis produces a range of microscopic changes involving hepatocytes, portal tracts, lobules, sinusoids, biliary structures, and the extracellular matrix. Acute hepatitis A is commonly associated with hepatocyte swelling, lobular disarray, apoptosis, variable necrosis, and portal inflammation [5,17]. Chronic hepatitis B may show ground-glass hepatocytes together with portal and interface inflammation [20]. Chronic hepatitis C may be accompanied by portal lymphoid aggregates, bile-duct injury, and steatosis [7]. Autoimmune hepatitis is often marked by lymphoplasmacytic portal inflammation and interface activity, although acute presentations may be lobular-predominant [8,9]. Drug-induced liver injury may produce a range of hepatocellular and cholestasis patterns [10,33,35], while alcohol-associated hepatitis

is often marked by steatosis, neutrophilic inflammation, and Mallory-Denk bodies [11]. MASH is histologically by the combination of steatosis, ballooning, and lobular inflammation [12,13,14]. Careful description of microscopic findings, supported by selective ancillary staining and clinical correlation, remains central to the histopathological assessment of hepatitis [1].

Declaration of AI-Assisted Figures

Figures 1 and 2 are original schematic illustrations generated with the assistance of an AI tool (Claude, Anthropic) and reviewed and approved by the authors. They are conceptual drawings, not photomicrographs of patient specimens, and they were not copied from published images.

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