JOURNAL OF INSURANCE MEDICINE

J Insur Med 2001;33:110–113

GRAPHICS SECTION

Liver Biopsy Interpretation in Chronic Hepatitis

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Liver biopsy reports are very variable in their terminology. In this article, frequently used terms and 2 common scoring systems are described.

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Key words: Liver biopsy, chronic hepatitis.

Received: October 30, 2000

Accepted: November 23, 2000

A 42-year–old male had liver biopsies in 1995 and 2000 for chronic hepatitis C. The 1995 pathology report showed:

> ''Mild portal mononuclear cell inflammation with mild periportal and lobular involvement. There is mild portal fibrosis. No stainable iron was detected. Chronic hepatitis C, Grade 2, Stage 1''

The 2000 biopsy report showed:

> ''Liver parenchyma with moderate portal lymphocytic aggregates. There is moderate piecemeal necrosis with some areas showing bridging. Mild focal intralobular necrosis with balloon degeneration is noted with mild steatosis. The trichrome stain is positive for periportal fibrosis with a few areas of bridging fibrosis. Knodell Score: 12 (5,1,3,3)''

This is typical of the variability in hepatitis pathology reports. One needs to understand the various terms and scoring systems used in pathology reports to compare the biopsies and determine the current status. The first biopsy was likely scored with one of the several systems similar to that developed by Batts and Ludwig in 1995.[1] This system, as well as the Desmet, Scheuer, Ishak, and METAVIR systems, scores the degree and location of inflammation as a *grade,* the location (extent) of the fibrosis as a *stage* and offers an *etiology* based on the biopsy, as well as the clinical information (see Table 1). The second biopsy is scored using an older system, the Knodell Histological Activity Index (HAI) Score.[2] This system assigns a numerical value to 4 different histological characteristics with a maximum total score of 22 (see Table 2). The latter system has been used in research protocols and has been shown to have good interobserver and intraobserver reliability. All of these scoring systems are used to describe viral, autoimmune, drug-induced, and cryptogenic hepatitis, as well as α_1-antitrypsin deficiency and Wilson's disease. There are additional findings that are reported separately from the grade and stage. They are sugges-

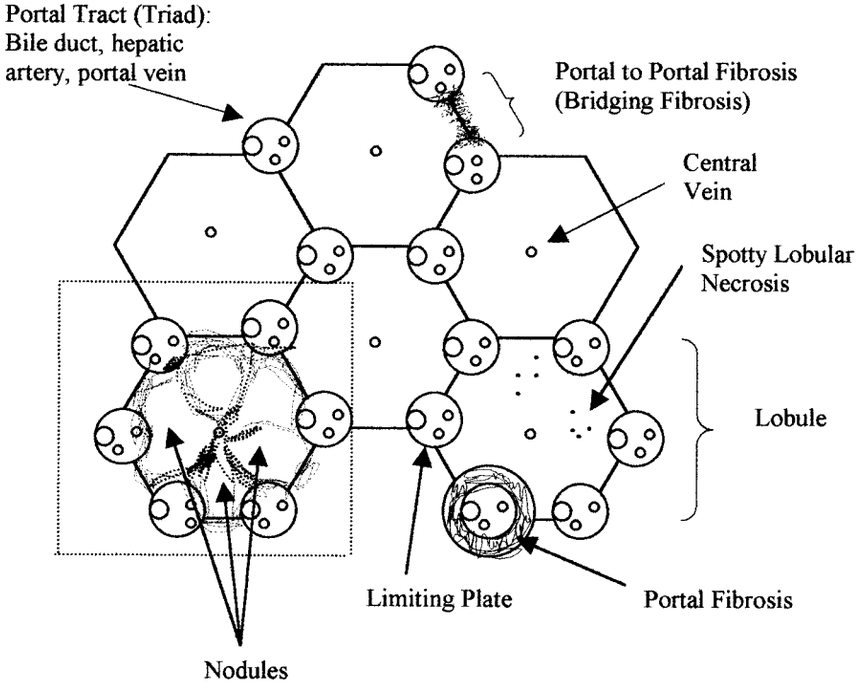
Table 1. Batts and Ludwig Staging and Grading of Chronic Hepatitis[1]

Stage

Score	Description	Criteria
0	No fibrosis	Normal connective tissue
1	Portal fibrosis	Fibrous portal expansion
2	Periportal fibrosis	Periportal or rare portal-portal septa
3	Septal fibrosis	Fibrous septa with architectural distortion; no obvious cirrhosis
4	Cirrhosis	Cirrhosis

Grade

Score	Description	Piecemeal Necrosis	Lobular Inflammation/Necrosis
0	Portal inflammation only (no lobular or piecemeal necrosis)	None	None
1	Minimal	Minimal, patchy	Minimal/few areas of patchy necrosis
2	Mild	Mild; involving some or all portal tracts	Mild/mild hepatocellular damage
3	Moderate	Moderate; involving all portal tracts	Moderate/noticeable hepatocellular damage
4	Severe	Severe; with bridging necrosis	Severe/prominent, diffuse hepatocellular damage



Schematic representation of liver histology with various pathologic changes. Area within the dotted lines demonstrates cirrhosis. Portal-to-portal and portal-to-central fibrosis results in nodule formation.

Table 2. Knodell Histological Activity Index[2]

Periportal± Bridging Necrosis	Score	Intralobular Degeneration and Focal Necrosis	Score	Portal Inflammation	Score	Fibrosis	Score
None	0	None	0	No portal inflammation	0	No fibrosis	0
Mild piecemeal necrosis	1	Mild (acidophilic bodies, ballooning degeneration and/or scattered foci of hepatocellular necrosis in one third of lobules or nodules)	1	Mild (sprinkling of inflammatory cells in less than one third of portal tracts)	1	Fibrous portal expansion	1
Moderate piecemeal necrosis (involves <50% of the circumference of most portal tracts)	3	Moderate (involvement of one third to two thirds of lobules or nodules)	4	Moderate (increased inflammatory cells in one third to two thirds of portal tracts)	3	Bridging Fibrosis (portal-portal or portal-central linkage)	3
Marked piecemeal necrosis (involves >50% of circumference of most portal tracts)	4	Marked (involvement of greater than two thirds of lobules or nodules)	4	Marked (dense packing of inflammatory cells in greater than two thirds of portal tracts)	4	Cirrhosis	4
Moderate piecemeal necrosis plus bridging necrosis	5						
Marked piecemeal necrosis plus bridging necrosis	6						
Multilobular necrosis	10						

tive, though not diagnostic, of the specific etiologies indicated. These include steatosis (hepatitis C), Mallory bodies (alcohol, Wilson's disease), copper deposits (Wilson's disease), ground glass hepatocytes (hepatitis B), and hepatocellular carcinoma.

The terms used in liver biopsies are defined below and illustrated in the Figure:

Inflammation and Necrosis

Portal inflammation: lymphocytes confined to the portal tract (triad).

Piecemeal (periportal) necrosis: the extension of lymphocytes or monocytes from the portal tract into the lobule (crossing the limiting plate) with the destruction of the periportal hepatocytes.

Interface hepatitis: same as piecemeal necrosis but introduced to reflect that apoptosis rather than necrosis is the predominant process at the limiting plate.

Bridging necrosis: the extension of inflammation and necrosis from one portal tract to another.

Lobular inflammation: presence of increased number of lymphocytes in the lobule.

Lobular necrosis: hepatocyte damage separate from the portal tract.

Fibrosis and Cirrhosis

Portal fibrosis: expansion of the portal tract by fibrosis without extension outside the tract.

Periportal fibrosis: extension of fibrosis outside the portal tract.

Bridging fibrosis: extension of fibrosis from one portal tract to another.

Cirrhosis: diffuse fibrosis with nodule formation. Nodules are groups of cells ringed by fibrosis as a result of portal-to-portal and portal-to-central vein fibrosis. They have no organized circulation or biliary drainage.

The terms, chronic persistent and chronic active hepatitis, are obsolete. These were inconsistently used to describe the morphology and disease etiology. Importantly, prognosis cannot be predicted by these terms.

The first biopsy is reported as Grade 2 based on the finding of mild periportal necrosis and mild lobular activity, and Stage 1 based on the presence of fibrosis limited to the portal tract. The diagnosis of chronic hepatitis C is made using both the histologic appearance and the clinical information. The Knodell score for this biopsy would be 1,1,1,1.

The second biopsy is scored according to the Knodell system: 5 for moderate piecemeal necrosis with bridging necrosis; 1 for mild intralobular degeneration and necrosis; 3 for moderate portal inflammation; and the last 3 for bridging fibrosis. This would be scored by the Batts-type scale as Grade 4, Stage 2. When there is a disparity between the criteria, the higher score is used. Using either system, there clearly has been progression of disease from 1995 to 2000.

REFERENCES

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2. Batts KP, Ludwig J. Chronic hepatitis an update on terminology and reporting. *Am J Surg Pathol.* 1995; 19:1409–1417.