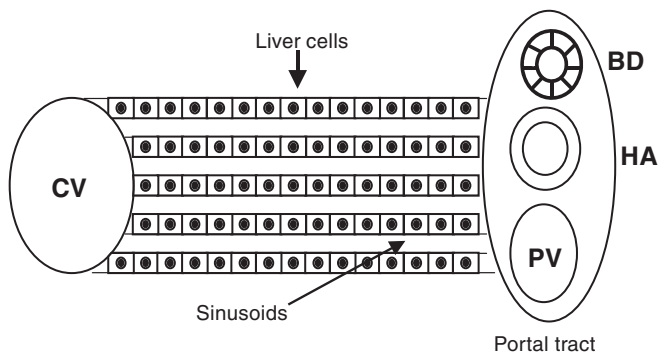


Although clinical and laboratory features yield clues to the extent of inflammatory processes (disease grade), the degree of scarring and architectural distortion (disease stage), and the nature of the disease process, the liver biopsy is felt to represent the gold standard for assessing the degree of liver injury and fibrosis. Examination of liver histology provides not only a basis for quantitative scoring of disease activity and progression but also a wealth of qualitative information that can direct and inform diagnosis and management.

A normal liver lobule consists of portal (zone 1), lobular (midzonal or zone 2), and central (zone 3) zones. The portal tract contains the hepatic artery (HA) and portal vein (PV), which represent the dual vascular supply to the liver, as well as the bile duct (BD). The lobular area contains cords of liver cells surrounded by vascular sinusoids, and the central zone consists of the central vein (CV), the terminal branch of the hepatic vein (see figure below).



Included in this atlas of liver biopsies are examples of common morphologic features of acute and chronic liver disorders, some involving the lobular areas (e.g., the lobular inflammatory changes of acute hepatitis, apoptotic hepatocyte degeneration in acute and chronic hepatitis, virus antigen localization in hepatocyte cytoplasm

and/or nuclei, viral inclusion bodies, copper or iron deposition, other inclusion bodies) and others involving the portal tracts (e.g., the portal mononuclear infiltrate that expands and spills over beyond the border of periportal hepatocytes in chronic hepatitis C, autoimmune hepatitis, and liver allograft rejection) or centrilobular areas (e.g., acute acetaminophen hepatotoxicity). Other histologic features of importance include hepatic steatosis (observed in alcoholic liver injury, nonalcoholic fatty liver disorders, and metabolic disorders—including mitochondrial injury—and in patients with chronic viral hepatitis); injury of bile ducts in the portal tract, an important diagnostic hallmark of primary biliary cirrhosis, primary sclerosing cholangitis, and liver allograft rejection; cholestasis in intrahepatic or extrahepatic biliary obstruction or in infiltrative disorders; ductular proliferation in the setting of marked hepatocellular necrosis; plasma cell infiltration common in autoimmune hepatitis; portal inflammation affecting portal veins (“endothelialitis”) in liver allograft rejection; and mild-to-severe fibrosis, in varying distribution and pattern, as a consequence of liver injury common to many disorders. (All magnifications reflect the objective lens used.)

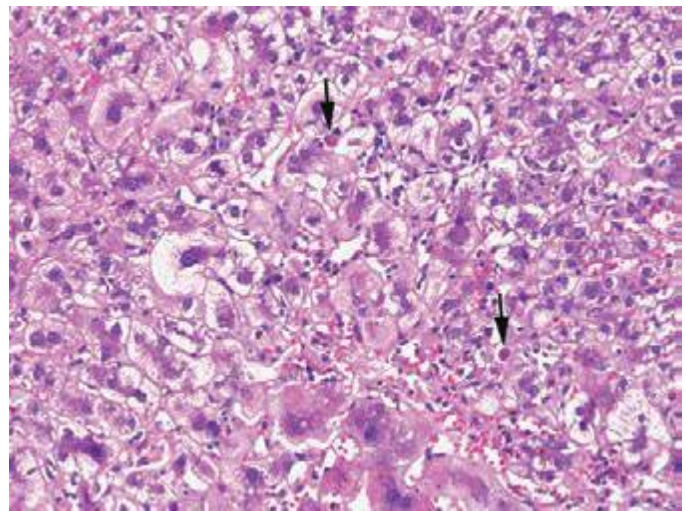


FIGURE 366e-2 Acute hepatitis, higher magnification, showing lobular inflammation, hepatocellular ballooning, and acidophilic bodies (arrows) (H&E, 20 \times).

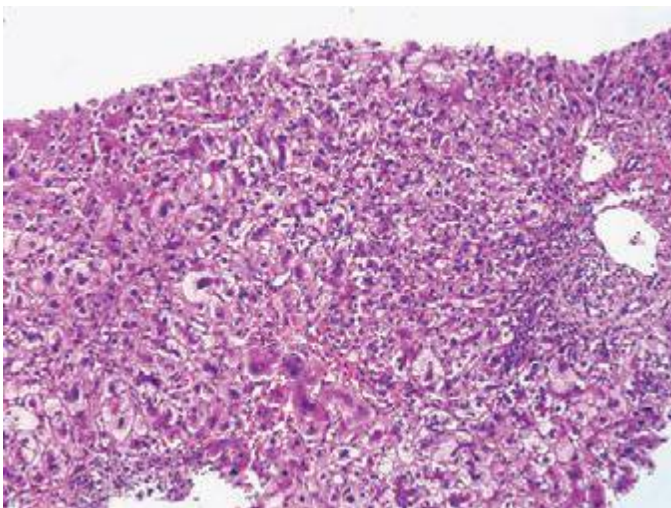


FIGURE 366e-1 Acute hepatitis with lobular inflammation and hepatocellular ballooning (hematoxylin and eosin [H&E], 10 \times).

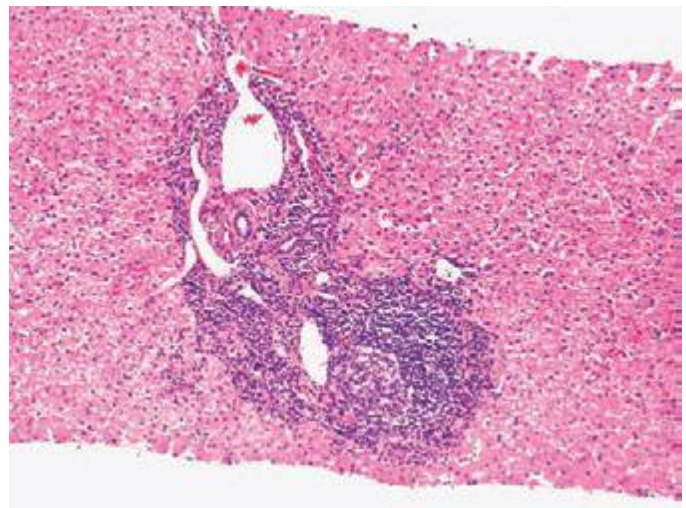


FIGURE 366e-3 Chronic hepatitis C with portal lymphoid infiltrate and lymphoid follicle containing germinal center (H&E, 10 \times).