

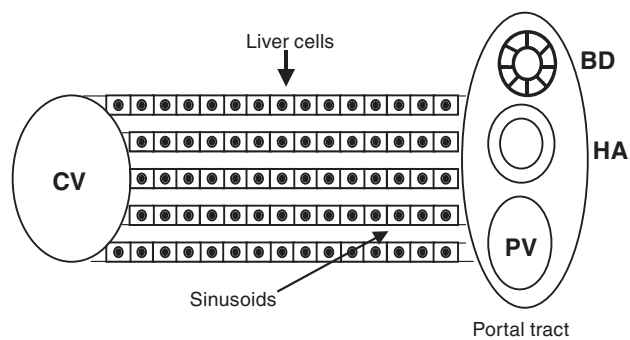
CHAPTER e38

Atlas of Liver Biopsies

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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, in nonalcoholic fatty liver disorders, in 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, as well as of 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.)

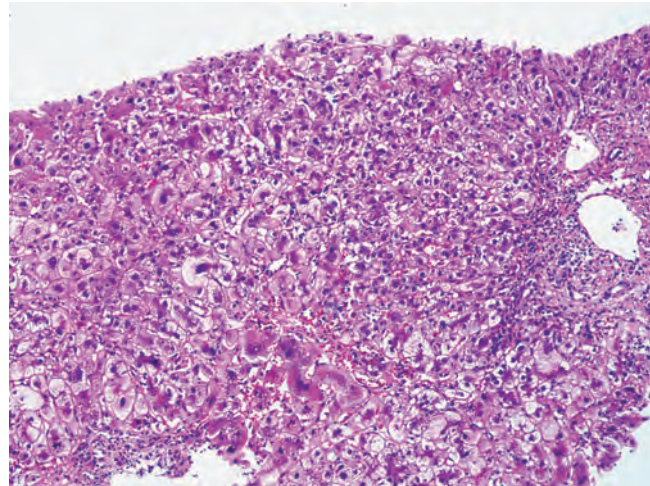


Figure e38-1 Acute hepatitis with lobular inflammation and hepatocellular ballooning (H&E, 10 \times).

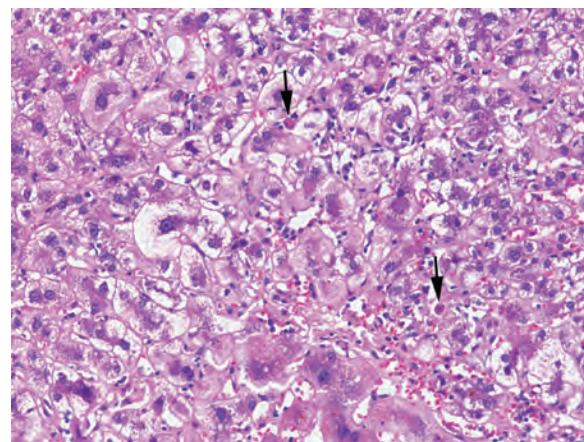


Figure e38-2 Acute hepatitis, higher magnification, showing lobular inflammation, hepatocellular ballooning, and acidophilic bodies (arrows) (H&E, 20 \times).

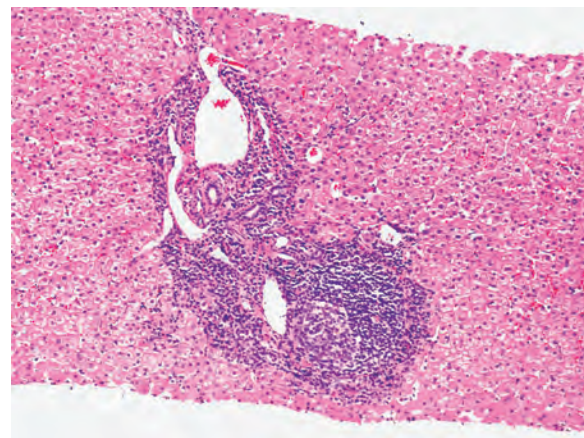


Figure e38-3 Chronic hepatitis C with portal lymphoid infiltrate and lymphoid follicle containing germinal center (H&E, 10 \times).

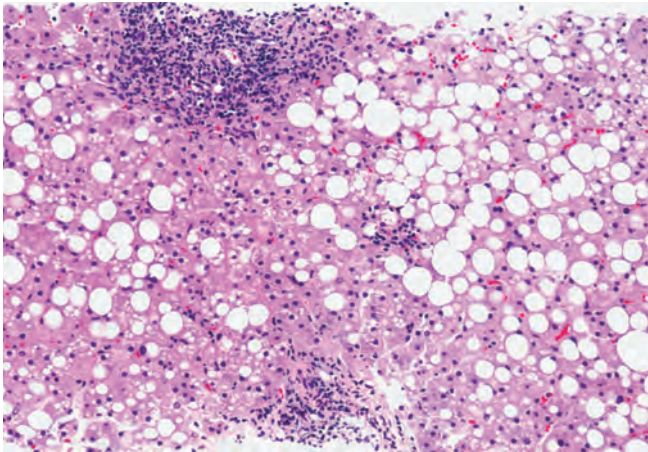


Figure e38-4 Chronic hepatitis C with portal and lobular inflammation and steatosis (H&E, 10 \times).

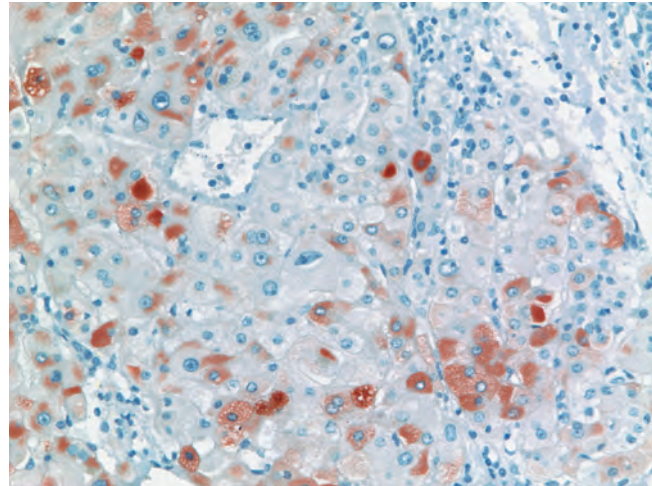


Figure e38-7 Chronic hepatitis B with hepatocellular cytoplasmic staining for hepatitis B surface antigen (immunoperoxidase, 20 \times).

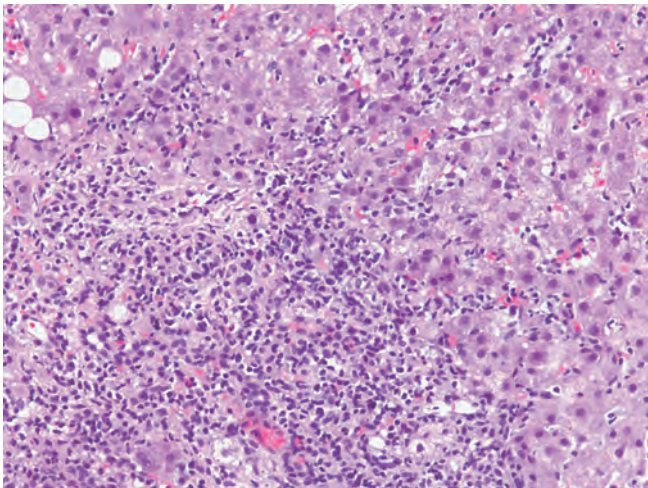


Figure e38-5 Chronic hepatitis C with portal inflammation and interface hepatitis (erosion of the limiting plate of periportal hepatocytes by infiltrating mononuclear cells) (H&E, 20 \times).

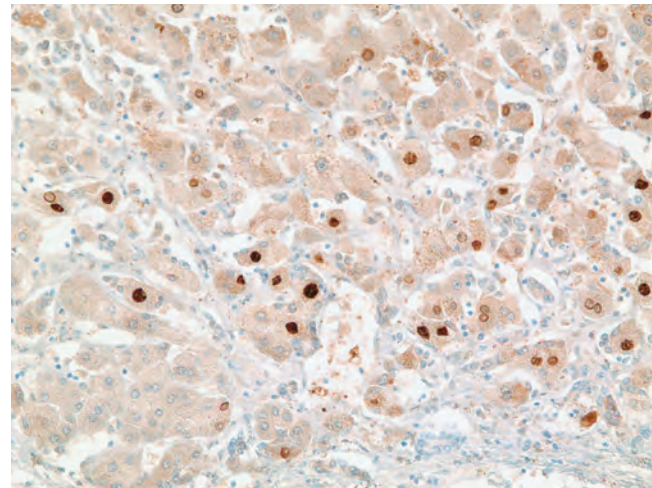


Figure e38-8 Chronic hepatitis B with hepatocellular nuclear staining for hepatitis B core antigen (immunoperoxidase, 20 \times).

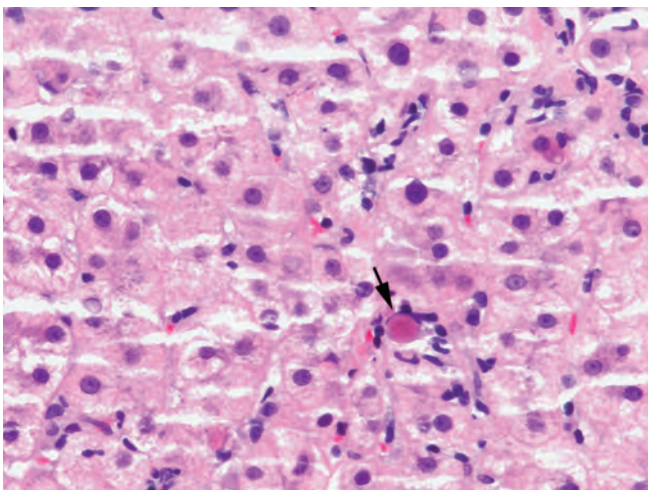


Figure e38-6 Lobular inflammation with acidophilic body (apoptotic body) surrounded by lymphoid cells (H&E, 40 \times).

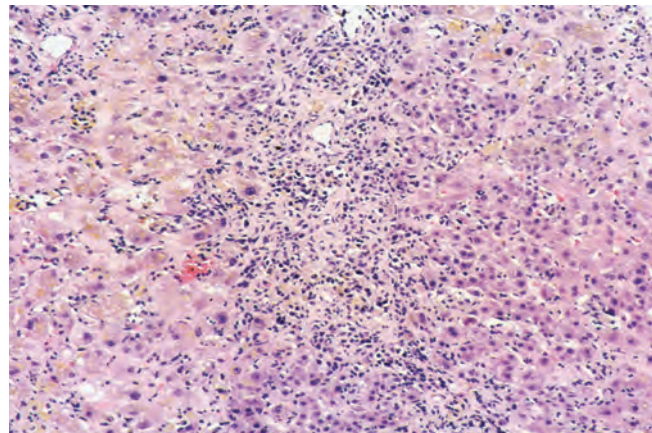


Figure e38-9 Autoimmune hepatitis with portal and lobular inflammation, interface hepatitis, and cholestasis (H&E, 10 \times).

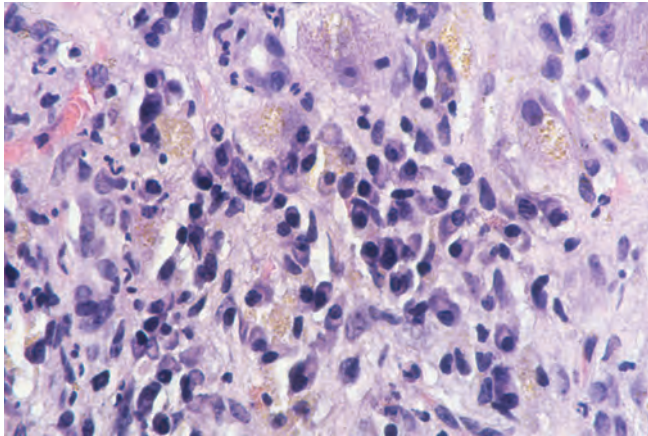


Figure e38-10 Autoimmune hepatitis, higher magnification, showing dense plasma cell infiltrate in the portal and periportal regions (H&E, 40 \times).

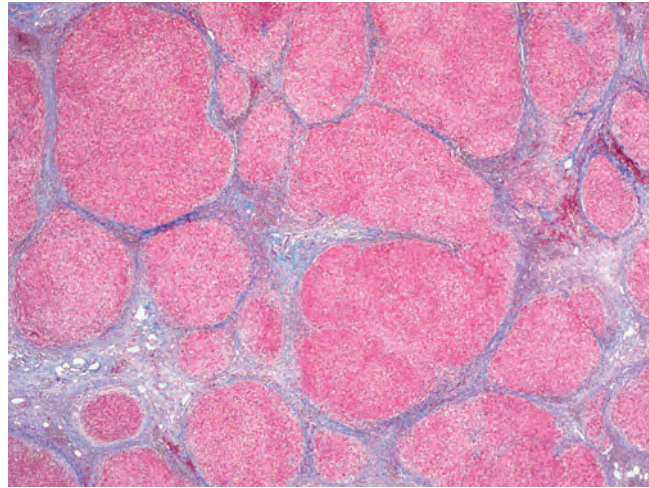


Figure e38-13 Cirrhosis with architectural alteration resulting from fibrosis and nodular hepatocellular regeneration (Masson trichrome, 2 \times).

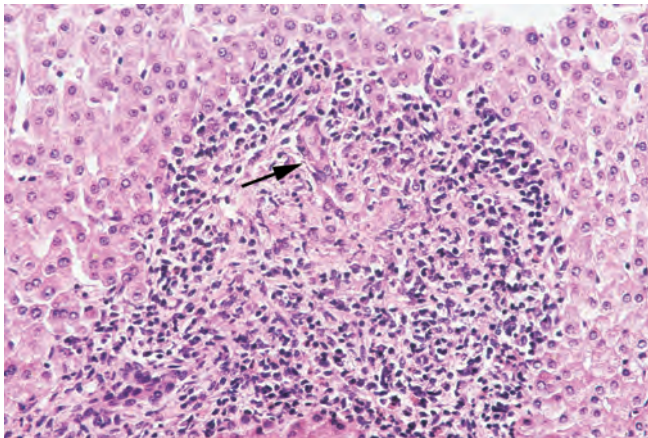


Figure e38-11 Primary biliary cirrhosis with degenerating bile duct epithelium (“florid ductular lesion”) (*arrow*) surrounded by epithelioid granulomatous reaction and lymphoplasmacytic infiltrate (H&E, 40 \times).

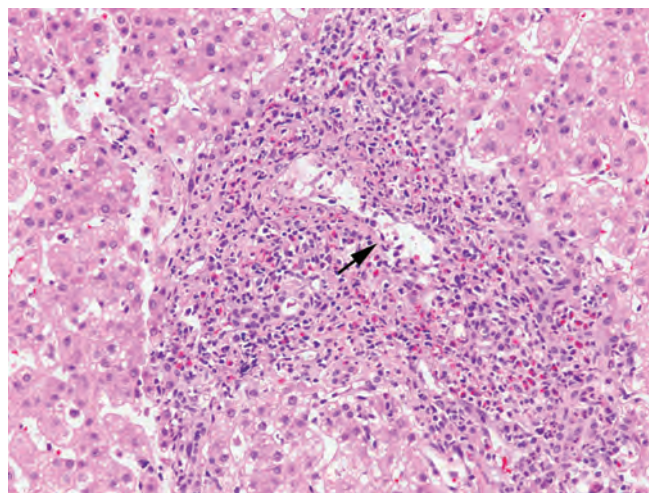


Figure e38-14 Acute cellular rejection of orthotopic liver allograft demonstrating a mixed inflammatory cell infiltrate (lymphoid cells, eosinophils, neutrophils) of the portal tract as well as endothelialitis of the portal vein (*arrow*) and bile duct injury (H&E, 10 \times).

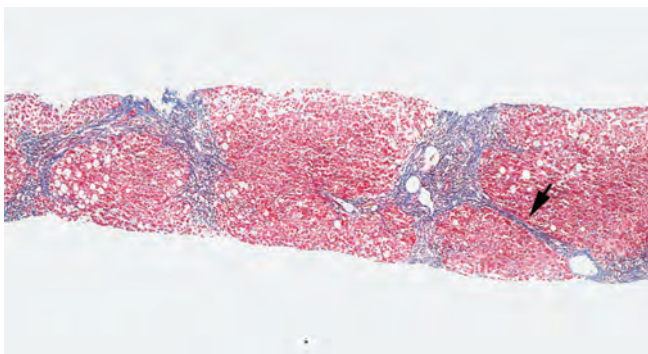


Figure e38-12 Chronic hepatitis C with bridging fibrosis (*arrow*) (Masson trichrome, 10 \times).

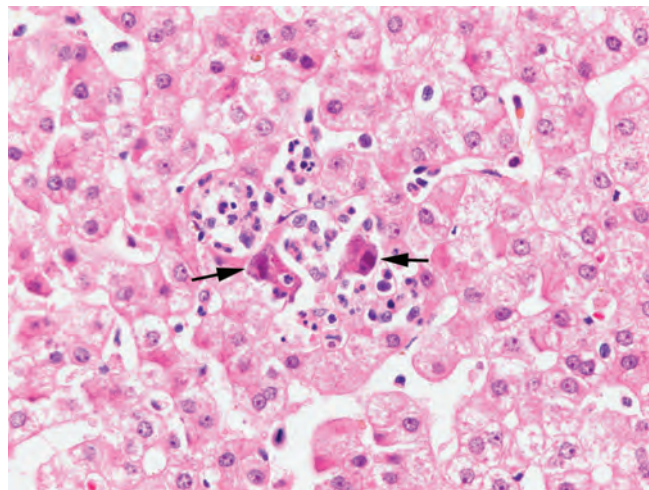


Figure e38-15 Liver allograft with cytomegalovirus infection showing hepatocytes with nuclear inclusions (*arrows*) surrounded by a neutrophilic and lymphoid infiltrate (H&E, 10 \times).

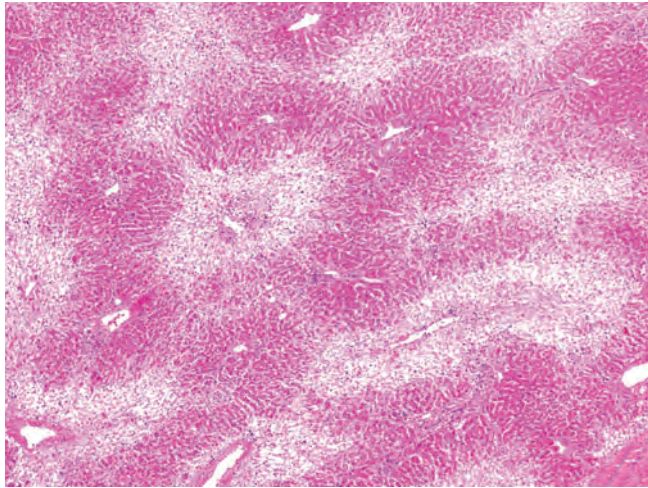


Figure e38-16 Combined acetaminophen hepatotoxicity and alcoholic liver injury with extensive centrilobular areas of necrosis (H&E, 4 \times).

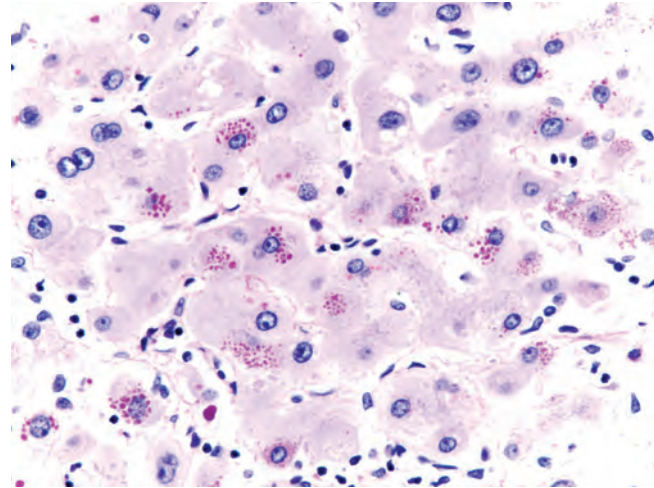


Figure e38-19 α_1 antitrypsin deficiency with higher magnification of PAS-positive, diastase-resistant globules (PAS, 40 \times).

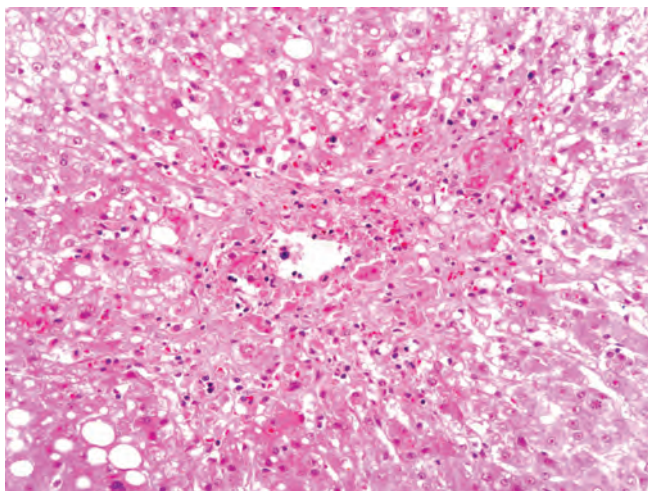


Figure e38-17 Combined acetaminophen hepatotoxicity and alcoholic liver injury at higher magnification showing necrotic centrilobular area with Mallory bodies (H&E 20 \times).

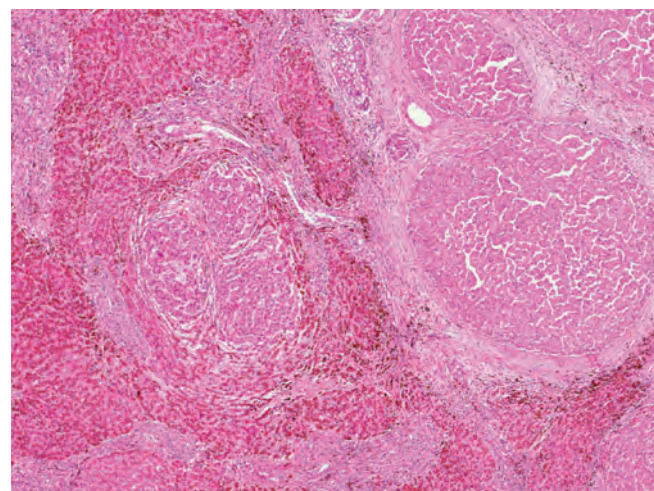


Figure e38-20 Cirrhosis secondary to hemochromatosis with hepatocellular carcinoma; brown hemosiderin pigment (iron) is present in the cirrhotic liver, while the hepatocellular carcinoma nodules are hemosiderin-free (H&E, 4 \times).

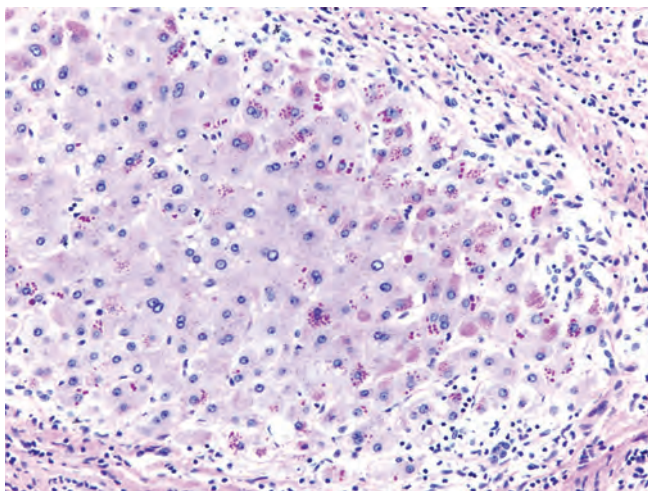


Figure e38-18 α_1 antitrypsin deficiency with cytoplasmic periodic acid-Schiff (PAS)-positive, diastase-resistant globules in many hepatocytes, predominantly at the periphery of a cirrhotic nodule (PAS, 20 \times).

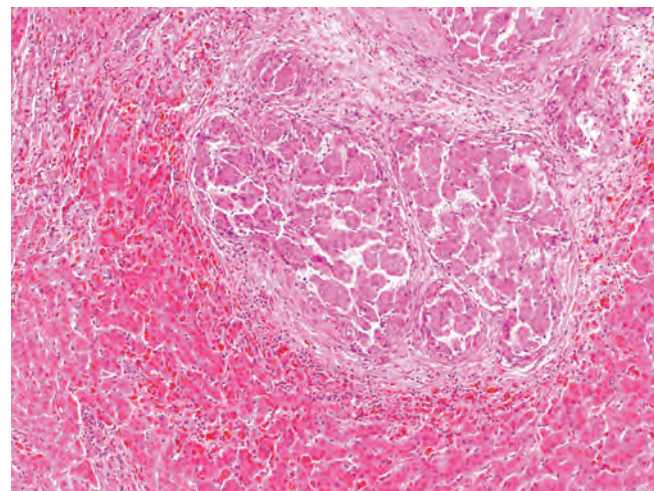


Figure e38-21 Cirrhosis secondary to hemochromatosis with hepatocellular carcinoma at higher magnification, demonstrating nodules of large malignant cells with highly disorganized architecture (H&E, 10 \times).

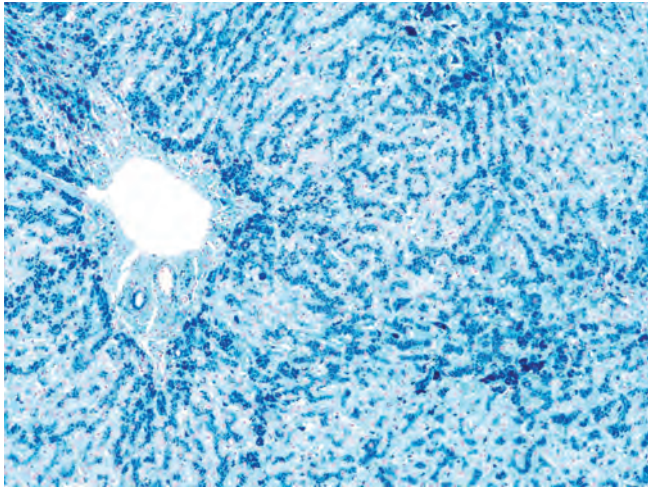


Figure e38-22 Hemochromatosis with iron stain demonstrating extensive iron deposition and characteristic pattern of pericanalicular distribution of iron (iron stain, 10 \times).

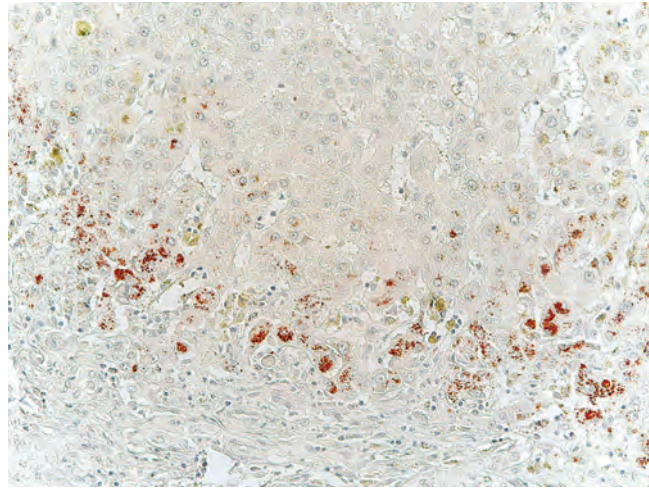


Figure e38-25 Primary sclerosing cholangitis showing peripheral cholestasis (*green*) and cytoplasmic red granular staining of hepatocytes for copper (rhodanine copper stain, 20 \times).

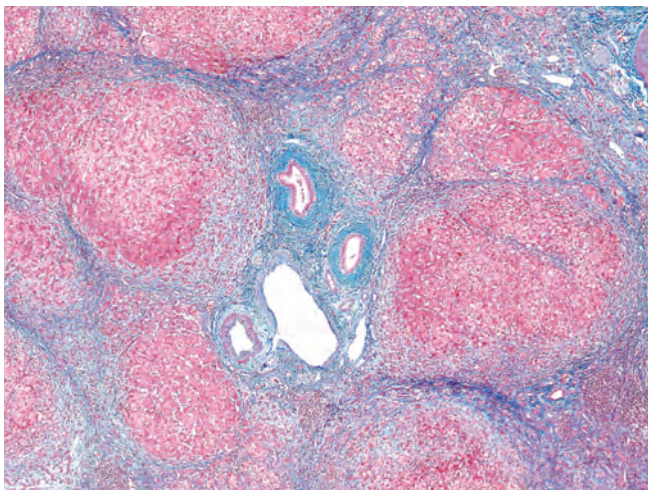


Figure e38-23 Primary sclerosing cholangitis showing cirrhosis and periductular fibrosis (Masson trichrome, 4 \times).

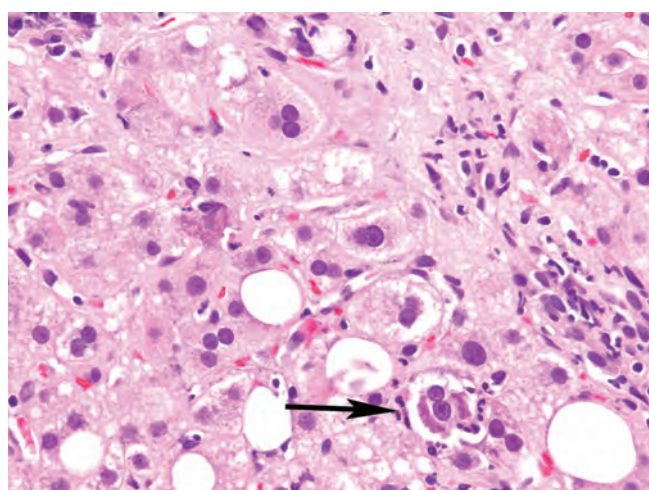


Figure e38-26 Nonalcoholic steatohepatitis (NASH) showing steatosis, ballooned hepatocytes, and Mallory bodies with surrounding polymorphonuclear leukocytes (*arrow*) (H&E, 20 \times).

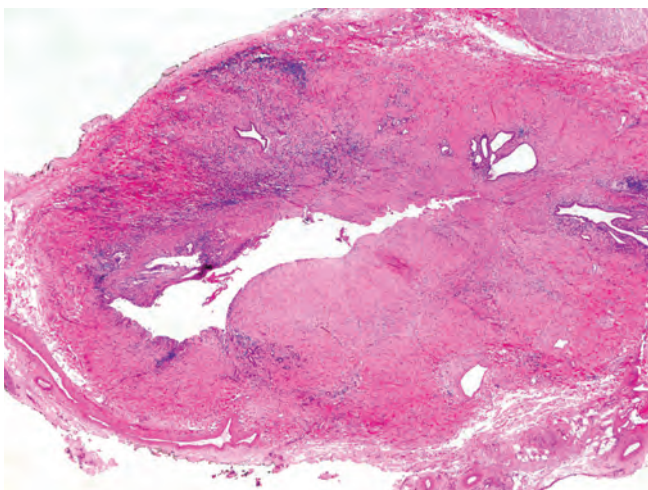


Figure e38-24 Primary sclerosing cholangitis showing the extrahepatic bile duct (in a liver explant obtained at the time of hepatectomy for orthotopic liver transplantation) with marked chronic inflammation and fibrosis as well as peribiliary glands (H&E, 2 \times).

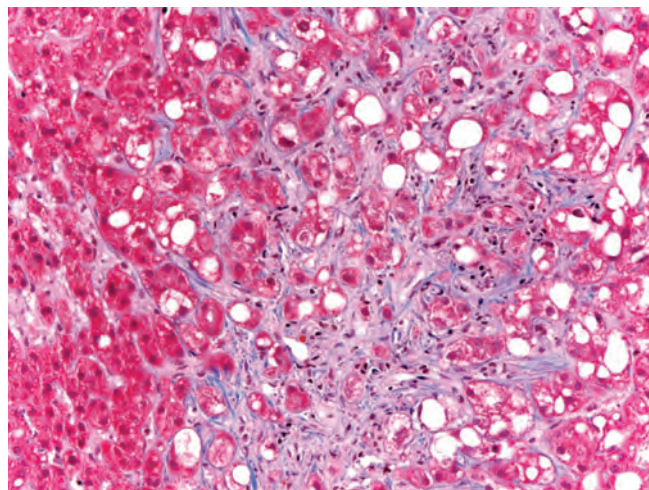


Figure e38-27 Nonalcoholic steatohepatitis (NASH) showing steatosis with perisinusoidal and pericellular fibrosis (H&E, 20 \times).

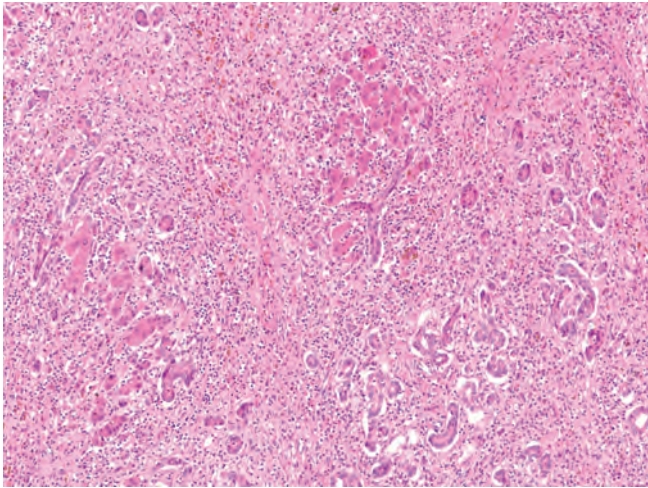


Figure e38-28 Acute hepatitis with submassive hepatic necrosis with marked parenchymal collapse, remnant islands of surviving hepatocytes, and a marked ductular reaction (H&E, 10 \times).

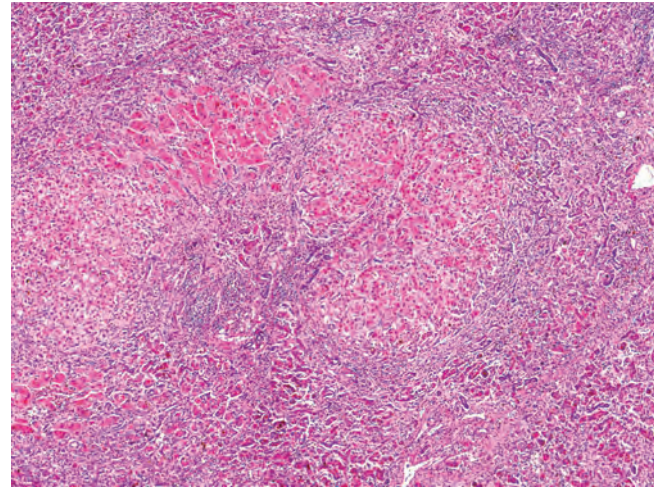


Figure e38-29 Wilson's disease showing cirrhosis, extensive collapse, and ductular reaction in a teenager with an acute presentation (H&E, 4 \times).

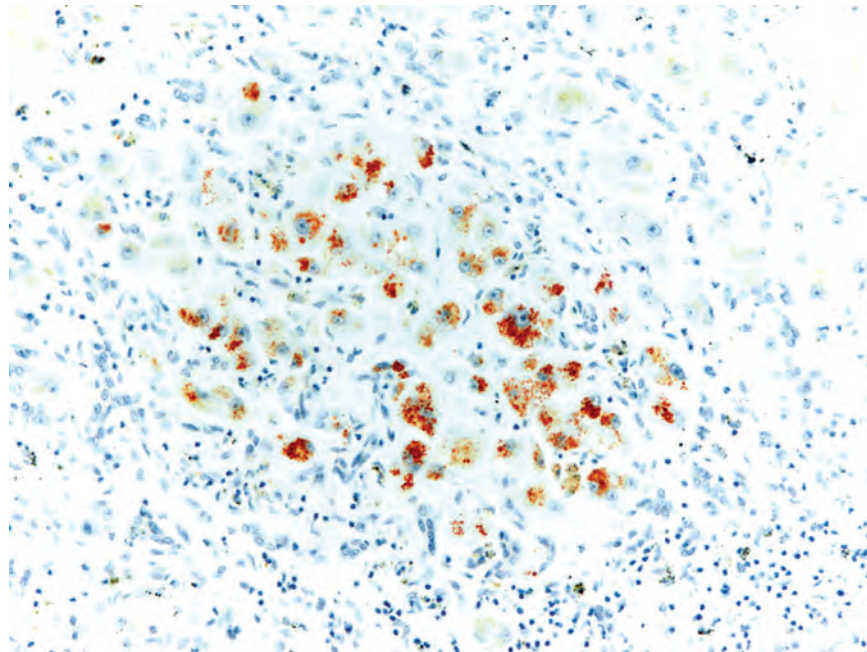


Figure e38-30 Wilson's disease showing extensive hepatocyte cytoplasmic red granular staining for copper in a cirrhotic nodule (rhodanine copper stain, 20 \times).