"one-size-fits-all" pangenotypic regimens—sofosbuvir-velpatasvir and glecaprevir-pibrentasvir—can be used, for 8–12 weeks, mostly without ribavirin, in almost all treatment-naïve, noncirrhotic and cirrhotic patients, including those with advanced renal failure and HCV-HIV co-infection. Applicability of the triple-drug combination sofosbuvir-velpatasvir-voxilaprevir is quite limited in treatment-naïve patients, reserved primarily for cirrhotic patients with genotype 3. As noted above, protease-inhibitor-containing DAA regimens (elbasvir-grazoprevir, glecaprevir-pibrentasvir, and sofosbuvir-velpatasvir-voxilaprevir) are contraindicated in decompensated cirrhosis.

AUTOIMMUNE HEPATITIS

■ DEFINITION

Autoimmune hepatitis is a chronic disorder characterized by continuing hepatocellular necrosis and inflammation, usually with fibrosis, which can progress to cirrhosis and liver failure. When fulfilling criteria of severity, this type of chronic hepatitis, when untreated, may have a 6-month mortality of as high as 40%. Based on contemporary estimates of the natural history of autoimmune hepatitis, the 10-year survival is 80−98% for treated and 67% for untreated patients. The prominence of extrahepatic features of autoimmunity and seroimmunologic abnormalities in this disorder supports an autoimmune process in its pathogenesis; this concept is reflected in the prior labels *lupoid* and *plasma cell hepatitis*. Autoantibodies and other typical features of autoimmunity, however, do not occur in all cases; among the broader categories of "idiopathic" or cryptogenic chronic hepatitis, many, perhaps the majority, are probably autoimmune in origin. Cases in which hepatotropic viruses, metabolic/genetic derangements (including nonalcoholic fatty liver disease), and hepatotoxic drugs have been excluded represent a spectrum of heterogeneous liver disorders of unknown cause, a proportion of which are most likely autoimmune hepatitis.

■ IMMUNOPATHOGENESIS