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Drug-induced liver injury: from pathogenesis to treatment

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The article presents data on classification, pathogenesis, clinical picture, diagnosis and differentiated treatment tactics, as well as practical algorithm for recognizing and preventing the development of drug-induced liver injury. Pathogenesis of drug-induced liver injury is analyzed, mechanisms of drug metabolism are explained, metabolism phases are described. Four main mechanisms of the pathological effect of drugs on the liver are identified: direct toxic effect on hepatocytes; toxic effect of drug metabolites; immunoallergic liver injury; idiosyncrasy. Peculiar attention is paid to the pathogenesis of drug-induced cholestasis. Direct hepatotoxic reactions develop according to the cytolytic (hepatocellular, parenchymal), cholestatic or mixed option. The most commonly diagnosed clinical variant of drug-induced liver injury is drug-induced hepatitis. Five forms of hepatitis induced by the use of pharmacological agents are distinguished: drug-induced hepatitis with an isolated increase in transaminases (anti-TB drugs, methyldopa, amiodarone, statins); acute hepatitis with jaundice; pseudo-surgical form of acute hepatitis: abdominal pain, fever, jaundice, enlarged gall bladder (cytostatics, antidepressants, antiarrhythmic drugs); severe forms of acute hepatitis with liver failure; chronic drug hepatitis. International diagnostic criteria, basic data on morphological liver changes are presented. Action of ursodeoxycholic acid is explained. It has a litholytic, anticholestatic, cytoprotective, immunomodulating, anti-inflammatory, antitoxic, hypocholesterolemic effect, modulates apoptosis, has a differentiated effect on the regeneration of hepatocytes.