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Clinical and Pathogenetic Aspects of Toxic Hepatitis

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Abstract: In the analytical article, the author reviews modern scientific studies of foreign and domestic authors devoted to the study of the pathogenesis of toxic liver lesions. The author analyzes the immuno-inflammatory mechanisms, oxidative stress, cytolytic breakdown of hepatocytes and neurointoxication processes, which are the basis of the pathogenesis of toxic liver lesions.

Keywords: pathogenesis, liver diseases, intoxication, chronic toxic hepatitis.

The issues of pathogenesis, diagnosis and treatment of diffuse liver diseases remain one of the most relevant in medicine, both due to the complexity of diagnosis and the choice of optimal treatment methods, and due to the tendency to increase the number of patients with these diseases. In this regard, early detection of patients with intoxication with industrial poisons, the main syndrome of which is chronic toxic hepatitis, remains one of the urgent tasks of public health [6-9, 11, 12, 17]. In production conditions as Many chemicals with hepatotoxicity are used as starting, intermediate or final products. The following groups are the most common: 1. Chlorinated hydrocarbons: methyl chloride, dichloromethane, chloroform, carbon tetrachloride, ethyl chloride, dichloroethane, tetrachloroethane, etc. 2. Organic solvents: xylene, toluene and their derivatives (nitrobenzene, trinitro-benzene, aniline, styrene). Alcohols — methyl, ethyl, propyl, esters, glycols. 3. Chlorinated naphthalenes — mono-, di-, orthochlor of naphthalene. 4. Metals and metalloids — lead, gold, manganese, arsenic, phosphorus[12].

Pistils (organochlorine and organophosphate), vinyl chloride, heptyl, etc. have hepatotropic effects. [4, 12]. Toxic substances are not found in the air of the working area in significant concentrations. Their slight increase causes sub-clinical forms of chronic toxic hepatitis (CTG). Chemicals or their metabolites have a direct and indirect toxic effect on hepatocytes, and also cause activation of cytotoxic immune cells.

T-cells. The toxic effect of xenobiotics depends on many factors — the chemical structure of the compound, blood flow in the liver, protein bonds, genetic factors, various cellular factors, age, dietary peculiarities, drug and alcohol abuse [5-8, 15]. Hepatotropic poisons or their metabolites cause various chemical reactions in the cell — depletion of reduced glutathione or oxidative stress with subsequent effects on proteins, lipids and DNA. This leads to apoptotic cell death and increased influence cytokines affect the immune system of the liver, as the cell organelles are involved in the process - mitochondria, cytoskeleton, endoplasmic reticulum, microtubules and nucleus [12, 14, 16]. According to a number of authors, xenobiotics may have an indirect effect on cell organelles through activation or inhibition of signaling kinases, transcription factors, and expression of profile genes. The outcome may be the launch of a necrotic or apoptotic process or an increase in the effect of cytokines on the immune system [12, 17, 10, 13-15].

Toxic liver damage (or TG) can manifest itself in acute and chronic form. Acute liver damage is characterized by necrosis and fatty degeneration of liver cells, hemodynamic disorders, edema

and protein dystrophy of cells. Develops through 2-3 days after exposure to the damaging agent and is accompanied by an increase in body temperature, weakness, loss of appetite, pain in the right hypochondrium, intense urine coloration, jaundice of the sclera and skin. It is possible to develop hemorrhagic and hepatorenal syndromes. Unlike acute TG, the clinical picture of chronic toxic liver lesions is unexpressed, characterized by a relatively benign course without a tendency to progression.

There are practically no clear signs of liver failure and outcomes in cirrhosis of the liver. In accordance with the classification of chronic liver diseases, HTG is closest to the so-called nonspecific reactive hepatitis in terms of clinical and morphological signs and course. Despite the duration of the process, lethal there are no outcomes that occurred directly as a result of liver damage. HTG has a long course with periodic exacerbations, usually occurring due to overwork or an error in the diet. It differs little from viral hepatitis - it is accompanied by weakness, anorexia, pain in the right hypochondrium, a feeling of heaviness, increased fatigue, bitterness in the mouth, especially in the morning.

The status indicates a slight ictericity of the sclera and skin. Signs of activity of the process - vascular asterisks on the chest and back, palmar erythema, usually not it happens. The liver is compacted, the size is moderately enlarged. The spleen is not enlarged. There are no signs of portal hypertension. HTG of professional genesis is characterized by a gradual development of the disease, which begins with dyspeptic complaints, the addition of biliary syndrome, moderate enlargement of the liver and a violation of its functional state.

Examination of the patient for markers of viral hepatitis B, C and D makes it possible to exclude the possible etiology of the disease. Ultrasound examination of the liver and biliary tract plays a great diagnostic value. In in complex cases of diagnosis, it is recommended to use computed tomography (CT) with determination of liver density [1-3, 15, 16]. CT allows you to get an image of the liver in the form of consecutive horizontal sections. To study the liver, 10-12 slices are required. It was found that the absorption coefficient in Hounsfield units (normally from 50-70) varied depending on the degree of severity of HTG - at the initial stages — 70-73, with moderate HCG — 75-80, with severe - 84-88 [1, 3].Special significance of CT it has a suspected cirrhosis of the liver, when blood clotting is disrupted and a liver biopsy is dangerous. The main criteria for the diagnosis of HTG of professional genesis are the professional route (prolonged contact with toxic substances) and data on the sanitary and hygienic characteristics of working conditions, indicating the measurements of toxic substances in the air of the working area.

Diagnosis of HTG includes the establishment of hepatitis based on the level of bilirubin and its fractions, albumin in the blood and the pro- thrombin index (decrease), activity transaminases, alkaline phosphatase and gammaglutamyltranspeptidase and exclusion of the viral etiology of the disease. It is necessary to take into account other symptoms of intoxication, changes in the blood, nervous system [16, 17]. All this makes it possible to properly conduct an examination of etiology with a profession for further professional rehabilitation of the patient [12].

The current direction of professional pathology is genetic research [7, 12], which were widely carried out in persons who were in contact with radiation, beryllium, microwave and suffered from professional allergodermatoses. The data of such studies in combination with clinical and functional characteristics help to identify predisposition to the disease and to identify individuals with an individual predisposition to certain industrial hazards.

The condition for the treatment of toxic liver damage is the cessation of industrial contact with toxic substances, detoxification of the body, replacement therapy and the introduction of hepatoprotectors. In addition to medical treatment, vitamin therapy and diet therapy with a restriction of the amount of fats, carbohydrates and salt are necessary. It is also required to limit physical activity, insolation and alcohol consumption. In the absence of signs of hepatitis activity , sanatorium treatment is indicated. The most commonly used hepatoprotectors: essentiale N, carsil, heptral, hepatofalcplank, flacoside [5, 10, 13, 15].

The drug is convenient to take in capsules of 250 mg, without chewing, in the evening before going to bed. Steroid hormones, cytostatics, immunosuppressors are recommended for high activity of the process in the liver. Indications for their appointment are progressive HCG with pronounced activity and cirrhosis of the liver with continued destruction of hepatocytes, high gamma globulinemia and activity of hepatic transaminases [12].

Conclusion. Prevention of occupational hepatitis consists in compliance with safety regulations, general and individual hygiene, proper storage of toxic substances. In industries where there is contact with hepatotropic poisons, uninterrupted and effective ventilation should be organized. Production processes should be sealed, automated and mechanized as much as possible. It is necessary to provide workwear workers and provide them with the opportunity to take a hygienic shower after work. Of great importance for the prevention of TG are the professional selection of workers for such production, proper nutrition with a sufficient amount of proteins and vitamins in the diet, the exclusion of alcohol abuse.

Periodic medical examinations play an essential role in identifying the earliest, most reversible forms of the disease. Contraindications to admission to such enterprises are organic diseases central nervous system, epilepsy, severe neuroses, mental disorders, nephritis and nephrosclerosis, organic myocardial diseases, some skin diseases. Pregnant women and women breastfeeding a child are not allowed to work associated with contact with hepatotropic poisons.

Preventive medical examinations are carried out 1 time every 12 months with the mandatory participation of specialists. To prevent the development of chronic intoxication when at least initial signs of liver damage are detected it is necessary to immediately stop the contact of the worker with hepatotropic substances. The course of professional HTG is usually benign. After eliminating the harmful factor, a full recovery is possible. However, in some cases, cirrhosis of the liver develops.

If chronic toxic liver damage or its residual effects are diagnosed in the form of persistent liver dysfunction, an increase in its size, dyspepsia and pain, as well as morphological changes in liver cells are established, a transfer to work outside is necessary contact with toxic hepatotropic substances for a long time. In this case , the qualification decreases and the patient is referred to a medical and labor expert commission to determine the appropriate disability group for an occupational disease. It should be taken into account that the residual effects of intoxication with hepatotropic poisons in the form of functional and morphological changes in the liver without a tendency to progression can be determined in a patient after a long time after the cessation of contact with toxic substances. In such cases , in the absence of clinical signs of liver damage, rational employment or retraining is advisable.

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