

**THE IMPORTANCE OF IMMUNOHISTOCHEMICAL LIVER EXAMINATION
IN FORENSIC MEDICINE FOR CASES OF DEATH FROM SURROGATE
ALCOHOL POISONING**

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ANNOTATION

Histological changes in the liver in surrogate alcohol poisoning are reflected. Characteristics of "toxic hepatitis" are given in dynamics, taking into account tissue reactions and cell cooperation. The distribution of Kupffer cells was studied, assessing their role in the course and outcome of liver damage. Sectional materials, histological and immunohistochemical research methods, clinical and additional data were used.

Keywords: surrogate alcohol, toxic hepatitis, fatty dystrophy, hepatonecrosis, immunohistochemical examination, Kupffer cells.

Various aspects of alcohol intoxication have been studied by domestic and foreign researchers from the late 19th century to the present day. In recent decades, forensic medical examination has increasingly focused on cases of poisoning by alcohol surrogates, homemade and counterfeit alcoholic beverages, and liquids not intended for oral use.

By helping to determine the damaging factor, the results of histological examination are taken into account in the diagnosis and expert assessment of injuries to the tissues and organs of victims, in establishing the main mechanisms of thanatogenesis. Simple and accessible, but at the same time objective and reliable histological methods for staining autopsy material are successfully used in the practice of forensic medical bureaus. Along with these, it is possible to use relatively new immunohistochemical analysis methods in expertise and toxicology.

At the same time, it should be noted that at the present stage, the range of toxic substances is expanding. The latest chemical and pharmaceutical industry agents can act as toxicants, the effects of which have been studied on experimental animals, while the mechanisms of their toxicodynamics and toxicokinetics in the human body have not been established. This determines the relevance of forensic medical examination

of lethal poisoning by unknown poisons, based on the results of morphological analysis.

Among the victims were 32 (64%) men aged 31 to 67 years and 18 (36%) women aged 20 to 69 years. 49 (98%) people, including those of retirement age, did not work anywhere, while in 21 (42%) cases, the victims did not have a fixed place of residence. Based on anamnestic and additional data, changes in internal organs that correspond to the principles of postmortem identification of chronic alcohol intoxication, they were classified as individuals who had long-term alcohol abuse.

Some of the victims (22 people) linked their acute poisoning to the use of "Crystal," "Arch" alcohols. In one of the fatal cases of poisoning, the victim drank the detergent "Dolyarishnik." The rest consumed alcohol-containing liquids, which were not detected. In addition to 96% ethyl alcohol, diethyl phthalate and polyhex-samethylene guanidine hydrochloride (antiseptic polymer) were included in these non-drinking alcohols and household chemicals. The known mechanisms of action of each of these chemical substances did not explain the peculiarities of this poisoning.

Changes in microcirculation were characterized by leukostasis, small diapedetic hemorrhages in the centers of the lobules against a background of paretic or uneven fullness of the sinusoids and venules.

In 33 (66%) cases of acute poisoning in September-October 2006, toxic dystrophy with impaired pigment metabolism and hemomicrocirculation in the liver was recorded. This group included all those who died outside the hospital, who did not receive therapy, as well as those who were treated for poisoning for no more than 10 days. In 14 people (28%) in addition to alteration, exudative processes with neutrophil accumulation in the center of the lobules developed on average from the 10th day of treatment, and by the 4th week, proliferative-productive processes joined the exudative reaction. Signs of organization, the appearance of protein dystrophy of hepatocytes instead of their obesity, were also noted in the liver of the treated patients.

Thus, when analyzing surrogate alcohol poisoning in residents of the Chelyabinsk region, we established characteristic microscopic changes in the liver. In cases of fatal poisoning (in September-October 2006), intra-lobular alterative processes with impaired liver pigment metabolism were recorded in the victims. Experiencing poisoning episodes by victims against the background of ongoing therapy led to the development of reactive and reparative processes.

Hepatonecrosis was combined with stromal reactions characteristic of chronic active hepatitis and fatty liver disease. Clinical and additional data allowed for the identification of a leading risk factor for poisoning from prolonged alcohol

consumption and its surrogates. However, the morphological picture of the liver did not correspond to its alcohol, drug, or metabolic damage. Histological data also reflected the absence of positive liver pathomorphosis against the background of therapy for this poisoning, unlike the treatment of acute toxic alcoholic hepatitis.

Immunohistochemical examination revealed the presence of proliferation and hyperplasia of mononuclear-macrophage cells in the zone of centrolobular hepatonecrosis. This indicator, like the combination of available clinical and additional data, indicates a primary chronic course of liver pathology and suggests sclerosis in its outcome.

1. The process of mononuclear infiltration most often develops when difficultly metabolized, coarsely dispersed, but weakly immunogenic material enters the internal environment of the body, causing persistent irritation of the mononuclear phagocyte system. In the cases of poisoning we studied, such a material could be PGMG-GC, products of impaired fat and pigment metabolism. It is known that postcapillary venules with a second type of endothelium are present in the vascular bed. Here, the main processes occur, culminating in the release of mononuclear phagocytes from the vessel into the tissue, from which inflammatory infiltrates (granulomas) are formed. In chronic inflammation, direct tissue damage cannot be observed. Destruction develops only after a sufficient number of activated macrophages accumulate in the tissue. The entire process of mononuclear infiltrate formation over time begins with the stimulation of monocytes released from the blood and their transformation into activated macrophages. The latter secrete chemoattractors and become centers for attracting new mononuclear phagocytes, granulocytes, and other inflammatory participants. D.N. Mayansky (1991) believes that situations where the infiltration process begins with the appearance and accumulation of activated macrophages in the tissue are possible. Our research allows us to agree with this opinion when interpreting the morphological picture of surrogate alcohol poisoning.

"Toxic hepatitis" reflects the collective concept of a certain suffering that transitions from acute poisoning by surrogate alcohol to liver disease.

In the morphological picture of poligexamethylene guanidine hydrochloride alcohol poisoning in the liver, alteration (damage) and proliferative reactions of the mononuclear-macrophage cellular link predominate. Changes in the liver during surgical alcohol poisoning differ from those in acute and chronic alcoholic hepatitis. Against the background of poisoning therapy, liver pathomorphosis develops.

Immunohistochemical examination allowed for the identification of the primary-chronic course of alterative processes in the liver in surrogate alcohol poisoning.

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