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Pathomorphological Signs of Acute Alcohol Poisoning

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Annotation. The presented article contains structured information on the issues of forensic medical examination of alcohol poisoning: the stages of ethanol biotransformation and variants of thanatogenesis, factors influencing the acceleration or deceleration of ethanol resorption and elimination are given; the principles of diagnosis and differential diagnosis of alcohol poisoning are outlined. The practical application of approaches to differential diagnosis of causes of death makes it possible to increase the comprehensiveness, completeness and validity of the forensic medical examination of a corpse in the event of death from alcohol poisoning.

Key words: poisoning, alcohol, ethanol, forensic examination, death.

Relevance. Pathomorphological (macro- and microscopic) signs of acute alcohol poisoning, detected during a forensic medical examination of a corpse, are nonspecific. They can only be considered in conjunction with the results of a forensic chemical examination of blood and urine and taking into account the circumstances of the preliminary investigation.

Macroscopic signs during external examination of a corpse: - cadaveric spots of an intense blue-purple color; - puffiness and bluishness of the facial skin; - swelling of the eyelids; - exophthalmos; - congestion of the vessels of the conjunctiva of the eyelids; - ecchymosis on the conjunctivae of the eyelids, in the skin of the face, upper chest, shoulder girdle; - accumulation of mucus in the nasal passages. Macroscopic signs during internal examination of the corpse: - plethora and swelling of the soft membrane of the brain; - increased amount of fluid in the lateral ventricles of the brain; - swelling of the choroid plexuses of the lateral ventricles of the brain (in the form of “bunches of grapes”); - swelling of the mucous membrane of the pharyngeal ring; - sharp hyperemia and swelling of the mucous membrane of the trachea and large bronchi; - pulmonary edema, hemorrhages under the visceral pleura; - hemorrhages under the epicardium; - hyperemia of the gastric mucosa with numerous hemorrhages, sometimes minor erosions; - swelling of Vater's nipple; - overflow of the gallbladder, gelatinous swelling of its bed and wall; - discolored contents of the duodenum; - acute venous congestion of internal organs; - focal hemorrhages in the pancreas; - pinpoint hemorrhages in the kidneys, adrenal glands (usually unilateral), and the abdominal surface of the diaphragm; - bladder fullness.

Microscopic signs: - severe venous congestion of internal organs; - violation of the permeability of the walls of vessels of all calibers (loosening of the vascular wall, swelling, desquamation of endothelial cells, plasma saturation of arterial walls; perivascular hemorrhages); - in the lungs: intraalveolar hemorrhages against the background of congestion, acute emphysema of the alveoli with rupture of their walls; - in the heart: myolysis of individual mosaic cardiomyocytes, sharp aggregation of erythrocytes in microvessels, numerous swellings of myocardial microvessels filled with plasma, sludge in venules, mainly in the myocardium, edema

of the myocardial stroma; - in the brain: swelling of brain tissue, diapedetic and perivascular hemorrhages in brain tissue; - in the liver: pronounced congestion of the central and interlobular veins, sinusoids; weak manifestations of microcirculatory disorders in the form of erythro-, leuko- and plasmastases, microthrombi in the lumen of blood vessels, expansion of the perisinusoidal spaces [4, 8, 14, 15, 18, 21, 22].

Forensic medical assessment of the results of quantitative determination of ethyl alcohol in cadaveric blood:

Ethanol content, ‰ 0.001–0.1 Endogenous ethanol

0.2–0.3 Ethanol consumption occurred

0.31–0.5 Minor influence of ethanol

0.51–1.5 Mild intoxication

1.51–2.5 Moderate intoxication

2.51–3.0 Severe intoxication

3.1–5.0 Severe poisoning. Possible death

5.1–7.0 Fatal poisoning of alcohol intolerant people

7.1–14.6 Fatal poisoning of alcohol-tolerant people

14.7–15.0 Maximum possible lethal level

More than 15.0 Accidental ethanol contamination of the blood sample being tested.

Differential diagnosis (DD) of death from acute alcohol poisoning and death from other causes that occurred while intoxicated is based on a qualitative analysis of the entire complex of data available to the forensic expert, namely: - the circumstances of death (time of drinking , general condition of the victim before death); - information about medical measures, if they were carried out before death; - information about alcohol tolerance; - information about addiction to alcohol; - information about the presence of diseases and medical treatment. An affirmative conclusion about the cause of death due to ethanol poisoning is based on the exclusion of somatic diseases and injuries that, by themselves or through their complications, can lead to death. In this case, diseases and conditions the course of which is complicated by acute alcohol intoxication should be excluded. The data on changes in the myocardium given below are of great importance; they can serve as evidence for reliably establishing the cause of death and facilitate the correct forensic diagnosis.

Morphological characteristics of microscopic changes in the myocardium (according to A.V. Kapustin, 2006):

Sudden death from ischemic heart disease as a result of acute coronary insufficiency

A. Chronic changes: Atherosclerosis of the coronary arteries and their branches of varying degrees: from lipoidosis to calcification. Organized thrombi in the coronary arteries. Cardiosclerosis: myofibrosis, small focal, large focal. Hypertrophy of cardiac muscle fibers.

B. Acute changes: Complicated atherosclerotic lesions of the coronary arteries: ulceration of plaques, hemorrhages into plaques, thrombotic deposits on damaged plaques. Fresh (disorganized) thrombi in the coronary arteries. Foci of clumpy decay of cardiomyocytes. Segmental contractures of the third degree in a small number of cardiomyocytes. Subsegmental contractures in a small number of cardiomyocytes. Most plasmatic microvessels. Sludge in the venules. Plasmorrhages in the walls of arterioles (in a small number of arterioles). Discoid decay in a small number of cardiomyocytes. Focal fragmentation of cardiac muscle fibers, their cracking. The unequal state of various cardiomyocytes: some of them are in a state of contraction, up to contracture (segmental), others are in a state of relaxation, up to hyperrelaxation.

Chronic alcohol intoxication (chronic changes) Atrophy of individual cardiac muscle fibers or their groups. Focal hypertrophy of individual cardiac muscle fibers or their groups. Increased deposition of lipofuscin grains, including large ones, in cardiomyocytes. Increased number of cardiomyocytes with nuclear amitoses. Absence of atherosclerotic changes in the coronary arteries and their branches or minor phenomena of atherosclerosis in the form of small flat lipid spots. Mild fibrosis or small-focal cardiosclerosis, perivascular atherosclerosis. Uneven thickening of the ventricular endocardium. Lymphoid infiltrates in the epicardium and myocardial stroma; lymphohistiocytic infiltrates may also occur. Deposition of small droplets of acidic lipids in the sarcoplasm of cardiomyocytes.

Acute fatal alcohol poisoning (acute changes)

Foci of cardiomyocyte lysis located in a mosaic pattern. Increased acid phosphatase activity. Sharp aggregation of erythrocytes in the form of "trains". Coagulation of plasma in microvessels. Swelling of endothelial cells in myocardial microvessels. Constriction and sharp expansion of microvessels. Sludge in the venules. Pronounced adhesion of erythrocytes to the walls of microvessels. Swelling of the myocardial interstitium, perivascular edema may occur. The arrangement of endothelial cells in arterioles in the form of a "picket fence".

Sudden death from alcoholic cardiomyopathy (acute changes, chronic changes listed above)

Edema of the myocardial interstitium, including often perivascular edema. Intracellular edema of many cardiomyocytes in the form of the appearance of longitudinal slits in their sarcoplasm, expanding towards the intercalary disc. Smearing (weak homogenization) of sarcoplasm in individual cardiomyocytes.

Changes in the myocardium resulting from reflex effects on the heart

Uneven state of cardiomyocytes: one is contracted, the adjacent one is relaxed. Contraction of some and relaxation of other groups of sarcomeres in the same cardiomyocyte. Expansion (opening) of the intercellular gap of the intercalary disc with the formation of a dissociation band. Cardiomyocyte contractures: segmental and subsegmental (contraction bands). A sharp relaxation of some cardiac muscle fibers, a contracted state of others. Sharp relaxation of cardiac muscle fibers without their wavy bends and deformations; an increase in the length of cardiac muscle fibers as a result of an increase in the height of isotropic discs. Wave-like bends and deformations of bundles of cardiac muscle fibers located surrounded by straight muscle fibers. Overextension of cardiomyocytes: segmental and subsegmental. Reflex cardiac arrest

Relaxation of cardiomyocytes: an increase in the height of anisotropic discs, exceeding that during normal relaxation of cardiomyocytes, a significant decrease in the height of isotropic discs. Another possible option: an increase in the height of the isotropic disks, a slight increase in the height of the anisotropic disks, which, however, is significantly less than the height of the isotropic disks. Multiple dissociation bands. Other reflex-induced changes. Focal fragmentation of cardiac muscle fibers. A variant of reflex cardiac arrest of the "vagal death" type (blows to the neck, compression of the neurovascular bundle, traumatic brain injury) - characteristic changes in cardiomyocytes: their sharp relaxation due to a sharp increase in the height of the isotropic discs while simultaneously decreasing the height of the anisotropic discs; absence of fragmentation of cardiac muscle fibers as a result of death from asystole.

In cases of death of persons with pathology of the cardiovascular system, in whom, according to the results of a forensic chemical study, ethyl alcohol was found in the blood and urine in a concentration of less than 3.0 ‰, it should be assumed that death occurred from acute cardiovascular failure against the background of toxic effects of alcohol. The possibility of

concluding fatal ethanol poisoning when its content in the blood is below 3.0 ‰ is allowed only in cases of reliable exclusion of other possible causes of death. A necessary condition for this is the detection of ethyl alcohol in the urine in a concentration exceeding 3.0 ‰ and indicating that shortly before death the ethanol content in the blood corresponded to the toxic level of death and that the death of the victim occurred in the elimination phase [8, 14].

The substantiation of the cause of death from acute alcohol poisoning consists of qualified preliminary information about the circumstances of death, the quantitative determination of ethyl alcohol in the blood and urine, and a complex of macro- and microscopic changes in internal organs.

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