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FAT EMBOLISM OF THE LUNGS AS A VARIANT OF THANATOGENESIS IN TOXIC LIVER INJURIES

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Annotation. Based on the morphological analysis of cases of "toxic hepatitis" with jaundice, the development of massive toxic liver dystrophy, complicated by adipose embolism of the lungs and other organs, was identified. Features of the macroscopic and histological patterns of embolic and hemorrhagic syndromes, infarcts, and infarct-pneumonias were revealed.

Key words: toxic dystrophy, liver, adipose embolism, morphological features.

Forensic medical examination of acute poisonings is based on chemical and morphological studies. Forensic chemical analysis aims to detect, identify, and quantify the toxic substance. The main objectives of autopsy examination are to establish a causal relationship between poisoning and death, determine the mechanisms of dying, their dependence on the type and concentration of detected toxins, and exclude other possible causes of death.

Modern forensic chemical methods have proven insufficiently sensitive to identify the cause of "toxic hepatitis," which has the worst prognosis for recovery and has already claimed hundreds of lives.

Histological changes in the structure of organs and tissues have been noted in poisonings by all toxins without exception. Morphological analysis of autopsy materials from 48 individuals who died in the Chelyabinsk region from an unidentified toxin revealed damage to all internal organs, with predominant liver injury.

Objective: The goal of this study was to determine the morphological and thanatogenetic features of toxic hepatopathy caused by this unidentified chemical toxin.

The study analyzed cases of death from acute poisoning by an unknown toxin, which had a similar clinical picture. A syndromic approach was used in the evaluation. Study materials included supplementary data, patient hospital records, and forensic medical examination reports. Organ fragments were fixed in a 10% neutral formalin solution. After washing in water, part of the material underwent standard paraffin embedding, while duplicate samples were cut on a freezing microtome. Lung tissue micropreparations were prepared using stepwise serial sections from pathologically altered tissues and subpleural areas of each lung lobe where no visible changes were observed. Histological changes were examined under a light microscope using standard, additional, and histochemical staining methods (hematoxylin-eosin, Sudan III, Sudan IV, Nile blue sulfate).

Results: Necrosis and pronounced fatty degeneration of hepatocytes with accumulation of neutral fats were observed around central veins and in the midzone of classical liver lobules. Parenchymal fatty Sudanophilic degeneration developed in internal organs.

General light microscopy was not informative for identifying circulatory, ventilatory, and inflammatory changes in the lungs. Additional staining for fat in the lungs revealed Sudanophilic inclusions in capillaries and arterioles, which dissolved in alcohols during standard paraffin embedding. In some cases, fat droplets migrated through the venous system. It is hypothesized

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that hepatic fat necrosis, occurring against the background of preexisting chronic hepatitis and cirrhosis, could be the source of adipose emboli in the lungs.

Fat droplets in blood vessels, amid exudative and proliferative cellular reactions, were found in inflammatory lung foci. It is likely that adipose embolism, during survival, contributed to the development of circulatory-hypoxic processes in the lungs, the addition of pathogenic microflora, and the formation of morphologically diverse infarct-pneumonias.

Autopsy material analysis revealed the following macroscopic changes in the lungs: cone-shaped and irregularly oval consolidation foci, mottled in appearance, yellow or yellow with red rims, forming in the subpleural (mantle) zone of the lung. Some contained areas of softening. Histological manifestations of thromboembolism and microbial embolism were absent.

Subpleural subatelectasis and emphysematous changes in the lung parenchyma were noted. Interstitial thickening was accompanied by productive cellular reactions in the stroma and parenchyma. Sudanophilic inclusions were located in the lumen and walls of small vessels and accumulated in alveolar macrophages. These findings, along with signs of pulmonary edema, also indicated the survival of embolism episodes.

In one case, adipose embolism and lung infarction became the source of adipose embolism in the heart and brain. The persistence of fat-containing particles in myocardial arterioles caused perifocal productive cellular reactions, accumulation of Sudanophilic masses in vascular wall histiocytes and cardiomyocytes. General microscopy revealed focal productive myocarditis with dystrophy, necrobiosis, and necrosis of parenchymal cells. The appearance of fat emboli in the brain led to the formation of giant multinucleated foreign body-type cells around Sudanophilic particles.

Gradual filling of lung capillaries with fat explained the progressive dystrophic changes and tissue hypoxia of internal organs, dyspnea, various clinical indicators, and unmanageable hypotension during the terminal period. In hospitalized patients, there was a decrease in bilirubin and positive dynamics in the course of hepatopathy against the background of hemodialysis and intensive care.

Cardiopulmonary failure was one of the causes of acute venous congestion in systemic organs, congestive hyperemia, diapedesis hemorrhages, paralysis, and progressive fibroplastic processes in the walls of terminal hepatic veins, spleen veins, and hypertension in the inferior vena cava and portal vein systems. This complication likely triggered the development of a mixed-genesis hemorrhagic syndrome in gastrointestinal and abdominal organs.

It is well known that adipose embolism, with blockage of 2/3 of the capillary network, leads to fatality.

Cases of adipose embolism have been described in mechanical trauma (massive bruising and crushing of subcutaneous fat, muscles, fractures), tumors, skeletal surgery, and liposuction. This list of etiological factors may be supplemented by massive toxic hepatic necrosis.

Conclusion: In "toxic hepatitis," parenchymal organs undergo fatty degeneration. Massive toxic hepatic necrosis is a cause of adipose embolism of the lungs and infarct-pneumonias. The lung infarct morphology in adipose embolism differs macroscopically and microscopically from that in thromboembolisms. Adipose embolism influences the development of cardiopulmonary failure, portal hypertension, and hemorrhagic syndrome in the terminal phase of toxic hepatopathy.

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