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Morphology of Chronic Hepatopathy in Combination of Chronic Drug and Chronic Alcohol Intoxication

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Abstract Consumption of alcoholic beverages by individuals suffering from drug addiction leads to more pronounced damage to liver cells, which is confirmed by a number of pathomorphological changes. In particular, there is an intensification of fatty degeneration of hepatocytes, an increase in the activity of inflammatory processes in the liver parenchyma, the presence of neutrophils in the inflammatory infiltrate, as well as a significant increase in the area of portal tracts due to connective tissue hyperplasia [1,3]. These changes reflect the complex effect of ethanol and narcotic substances on hepatocytes, leading to progressive fibrosis and potential transformation into cirrhosis.

Keywords Liver, Chronic intoxication, Alcohol

1. Introduction

One of the most typical diseases of drug addicts (especially with intravenous drug use) is considered to be chronic hepatitis of viral etiology. However, the data of modern literature on the nature and prevalence of damage to this organ in poisoning with narcotic drugs, as a rule, are incomplete and extremely contradictory [4]. In particular, the role of drugs in the pathogenesis of liver damage remains unclear. There are no data on liver damage in combined intoxication with narcotic drugs and alcohol. The issue of differences in the course and morphology of viral hepatitis against the background of drug addiction and in people who do not use drugs remains unresolved [2,5].

2. Material and Methods

At this stage of our research, we used the histomorphometric method of studying liver tissue in chronic alcohol intoxication (CAI), chronic opiate intoxication and their combination.

3. Results and Discussion

No significant differences were found in the morphometric study of parameters such as the ratio of the area of hepatocytes, their nuclei, perisinusoidal spaces and sinusoids to the total area of the parenchyma in the field of view. The proportion of the parenchyma area accounted for by fat vacuoles in isolated drug poisoning (IDP) was 0.01, in the group with combined drug poisoning (CDP) – 0.05 ($P<0.05$).

When measuring the average thickness of the central vein wall, no statistically significant differences were found between the three groups. Some authors consider sclerosis of the walls of the central veins, leading to their compaction, to be a diagnostic criterion for CAI. According to Yu.I. Pigolkin (2004) this sign is also characteristic of CNI, including IONV, and therefore it can be used only as a criterion for chronic exogenous intoxication without specifying its type. Fibrosis of the walls of the central veins in drug addicts is apparently not associated with alcohol consumption, but has another explanation.

The proportion of the parenchyma area occupied by intralobular infiltrates in IONV was 0.0003, and in the SONV group - 0.0009 ($P<0.05$).

Portal tracts were expanded in all studied groups due to the growth of connective tissue, and in drug addicts also due to inflammatory infiltration and proliferation of the bile ducts. The average cross-sectional area of the portal tract in IONV was 58076±7450.9 μm^2, in SONV – 51962.0±6761.4 μm^2 (the difference is statistically insignificant). In the comparison group (KHAI) this value was 31608.7±9156.0 μm^2 ($P<0.05$).

A similar pattern was found when measuring the average perimeter of the portal tract cross-section, which was 1237.5 ±76.9 μm against the background of IONV, 1353.3±88.6 μm with SONV, and 764.8±131.8 μm with alcohol intoxication ($P<0.05$). The proportion of the perimeter of the portal tract cross-section that falls on the foci of destruction of the border plate in drug addicts was, on average, 0.1 with IONV and 0.09 with SONV (insignificant). In the comparison group (CHAI), the border plate remained intact in all the studied visual fields. The number of bile ducts per portal tract was higher in IONV than in SONV (3.68±0.54 and 2.63±0.6, respectively, ($P<0.05$). In the comparison group (KHAI), it

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was 1.2±0.7 (P<0.05). The diameter of the ducts in the study groups did not differ significantly.

The proportion of the portal tract cross-sectional area accounted for by the nuclei of inflammatory infiltrate cells in SONV and IONV had no reliable differences, but was significantly smaller in patients with CAI (20.3% of the portal tract area in IONV, 22.1% in SONV, 8.9% in CAI). In CAI, blood vessels predominated (10.1% of the portal tract area in IONV, 18.2% in SONV, 33.4% in CAI). The proportion occupied by connective tissue fibers was approximately the same in all groups and amounted to 33-40%. The cross-sectional area of the bile ducts in SONV and CAI was relatively similar, whereas in IONV it was slightly larger (5.0% of the portal tract area in IONV, 3.3% in SONV, 3.2% in CAI). Data on the cellular composition of the inflammatory infiltrate of the portal tracts and the number of cells of each type per 1 μm2 in all three study groups are presented in the table.

In IONV, the infiltrate contains fewer neutrophils (P<0.05) and slightly more lymphocytes than in drug poisoning (P<0.05); macrophages and, especially, fibroblasts (P<0.05) predominate. The proportion of neutrophils differs significantly from that in SONV only in cases of acute alcoholic hepatitis, when it can reach 49.2%. Eosinophils and plasma cells were absent in portal tracts in CAI, whereas in IONV and SONV, their numbers were approximately the same. The results obtained allow us to recommend the study of such liver tissue parameters as the proportion of parenchyma accounted for by fat vacuoles and intralobular infiltrates, the perimeter and cross-sectional area of the portal tract, and the length of the foci of destruction of the border plate for the differential diagnosis of CNI and CAI. along the perimeter of the portal tract, the average number of ductules in the portal tract, the proportions of the portal tract section occupied by inflammatory infiltrate cells and vessels, the proportions of fibroblasts, macrophages, lymphocytes, neutrophils, lymphocytes and plasma cells in the inflammatory infiltrate.

Our studies have shown that alcohol consumption by drug addicts leads to increased damage to hepatocytes, which is manifested by increased fatty degeneration, increased activity of lobular hepatitis, the appearance of neutrophil admixture in the inflammatory infiltrate and increased sclerotic processes leading to an increase in the perimeter of the cross-section of portal tracts due to the proliferation of connective tissue. Quantitative analysis of these processes can be recommended for use by forensic experts (histologists) when differential diagnostics of IONV, SONV and CAI is necessary.

4. Conclusions

Our studies have shown that markers of chronic alcohol intoxication are found in those who died from combined acute alcohol and opiate poisoning significantly more often than in those who died from isolated acute opiate poisoning, which can be explained by the role of ethanol in the development of these morphological changes.

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