European Journal of Molecular & Clinical Medicine
ISSN 2515-8260 Volume 7, Issue 2, 2020

FREQUENCY OF HAPTOGLOBIN PHENOTYPES AND PATHOLOGICAL CHANGES OF THE LIVER DURING POISONING BY DRUGS ON THE BACKGROUND OF ALCOHOL INTOXICATION

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Abstract: Mortality from the consequences of drug addiction and chronic alcohol intoxication and the health disorder associated with it constitute an increasingly significant proportion of the total mortality rate.
Keywords: pathologic, phenotypes, haptoglobin, intoxication

In recent years, the problem of drug addiction and alcoholism has become acute due to the epidemiological and social danger of these diseases, which pose a real threat to the mental and physical health of young and reproductive populations [1, 3]. Special attention is paid to the problem of chronic alcoholism, which has acquired both social and medical significance.

Mortality from the consequences of drug addiction and chronic alcohol intoxication and the health disorder associated with it constitute an increasingly significant proportion of the total mortality rate.

The liver is the main target organ in drug and alcohol addicts. Pathological process in the liver is one of the main somatic complications resulting from direct toxic influence of the applied means [2].

Resistance of an organism to toxic influences is determined by the nature of immune response, which is genetically determined. Observations show that there is a link between an organism's resistance to certain diseases and the haptoglobin phenotype [4, 5].

Haptoglobin (Hp) is a genetically polymorphic protein present in blood in the form of two homozygous (Hp 1-1 and Hp 2-2) and one heterozygous variant (Hp 2-1). In the literature, there is evidence that homozygous phenotypes Hp many diseases acquire an adverse course.

Purpose of the work. To study the distribution of haptoglobin phenotypes and pathomorphological changes occurring in the morphofunctional zones of the liver tissue during drug intoxication against the background of alcohol intoxication.

Materials and methods. Autopsy and histological study as well as determination of haptoglobin phenotypes in serum were carried out in 117 observations, 78 of which were men and 39 women aged 16 to 69 years. A control group of 59 cases was selected to compare the results with the violent death category.

Results and its discussion. The results of a morphological study of the liver of people who died from chronic poisoning with narcotic substances in combination with alcohol showed that degenerative changes in the liver parenchyma are more pronounced and often

manifest in a diffuse large-droplet nature with the development of massive foci of fatty necrosis (Fig. 1), in 95.3% of cases. In this case, the beam structure of hepatocytes is not detected in the liver tissue, they are completely destroyed and hepatocytes are transformed into fat-containing cells with the formation of massive foci of lipogenic necrosis. Where the hepatocyte nuclei are almost completely destroyed in the form of karyolysis, karyopyknosis and karyorrhexis. In their circumference, the appearance of empty lakes is noted due to the paralytic expansion of both sinusoids and perisinusoidal spaces. Only on the periphery of the lobules of the liver is the preservation of the largest portion of the parenchyma without necrosis and fatty degeneration observed, where significant swelling of hepatocytes due to matte degeneration of the cytoplasm is detected (Fig. 2). In some of them, the destruction of nuclei in the form of karyolysis or vacuolization of karyoplasm in the form of "empty nuclei" is noted. Cholestasis and a macrophage reaction are also noted in areas of pronounced vacuole dystrophy of hepatocytes. In other parts of the liver, the relative beam structure of the parenchyma is preserved, but with small and large droplet fatty degeneration of hepatocytes. In these areas of the liver parenchyma, compensatory lipocytic hyperplasia is observed in the form of a single and group accumulation in perisinusoidal spaces (Fig. 3).

Fat dystrophy of hepatocytes with formation of fatty necrosis and lipogranulomas was revealed in the form of characteristic diagnostic signs of combined liver damage by drugs and alcohol, with predominance of protein dystrophy centrolobular foci of colliquative necrosis, which was observed in 56,7% of cases. Dyscirculatory disorders in the form of increased permeability of sinusoid walls were accompanied by hemosiderosis of both hepatocytes and stromal histiocytic cells. There were pockets of liver parenchyma with predominance of protein dystrophy with the appearance of coarse protein, typical for alcoholic liver damage with the appearance of Mallory cells, we found in 52.8% of cases. An accumulation of lymphoid and macrophage cells was found around the abnormal protein (Fig.4). The expansion of Disse space and activation of reticuloendothelial cells in the form of phagocytic inclusions was observed in the circumference of such sites.

In addition to dystrophic changes and lymphatic infiltration in periportal tracts, sclerosis development in the form of expansion of connective tissue fibrous structures and reproduction of histiocytic cells with formation of relatively thick fibrous bundles is revealed (Fig.5). What is proved in the van Gieson histochemical study by the manifestation of sclerosis foci in the form of red-brown collagen fiber bundles (Fig.6) around the portal pathways was revealed in 78,4% of our cases.

The results of the serum haptoglobin study showed that the distribution of Hp phenotypes in the main group differs from that in the control group. There has been a reliable increase in the number of bodies with Hp 1-1 phenotype due to a reliable decrease in the number of Hp 2-2 phenotype holders. This indicates that persons with Hp 1-1 phenotype are prone to death in chronic drug poisoning against the background of alcohol compared to those who have Hp 2-2 phenotype in their serum. Naturally, a combination of several factors is required for lethal disease to occur. One of the predisposing factors may be the presence of the Hp 1-1 phenotype.

Conclusions. Thus, the use of alcohol in addicts leads to increased damage to hepatocytes, which is manifested by increased fat dystrophy, increased activity of lobular hepatitis, the appearance of an admixture of neutrophils in inflammatory infiltration and increased sclerotic processes.

Individuals with the Hp 1-1 phenotype are more likely to be fatal and that the presence of the Hp 1-1 phenotype should be considered a risk factor.

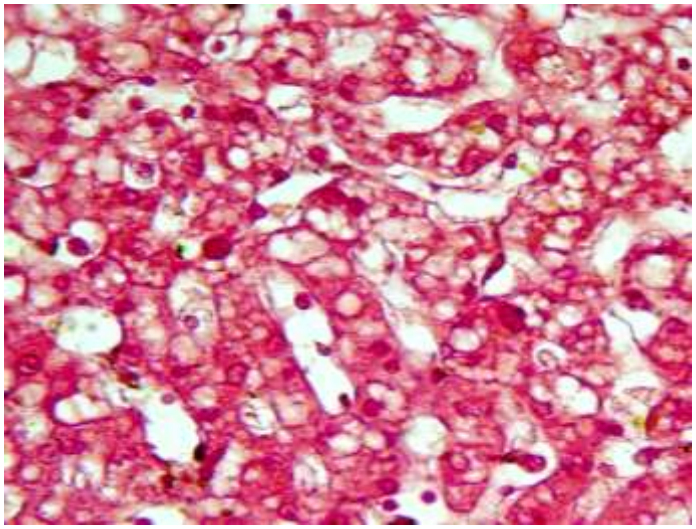

Fig. 1. Large drop fat dystrophy of hepatocytes in chronic drug poisoning combined with alcohol. Color: hematoxylin eosin. Enlargement: approx. 10, vol. 40.

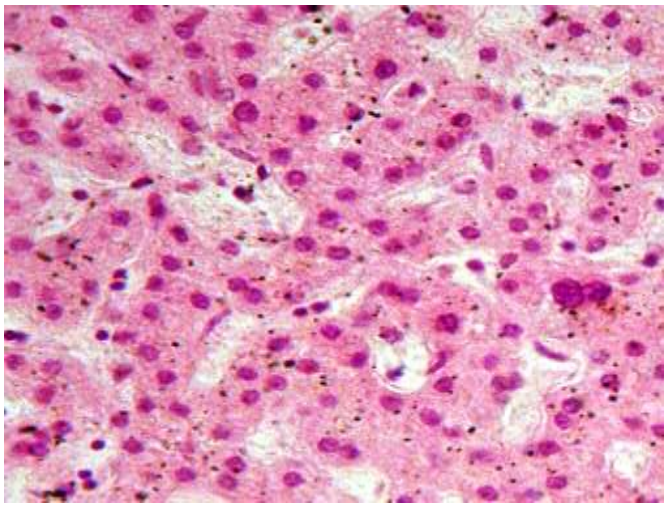

Fig. 2. Swelling of hepatocytes, lipofuscinosis of stellar endotheliocytes at periphery of liver lobes. Color: hematoxyline-eosin. Increasing: approx. 10, vol. 40.

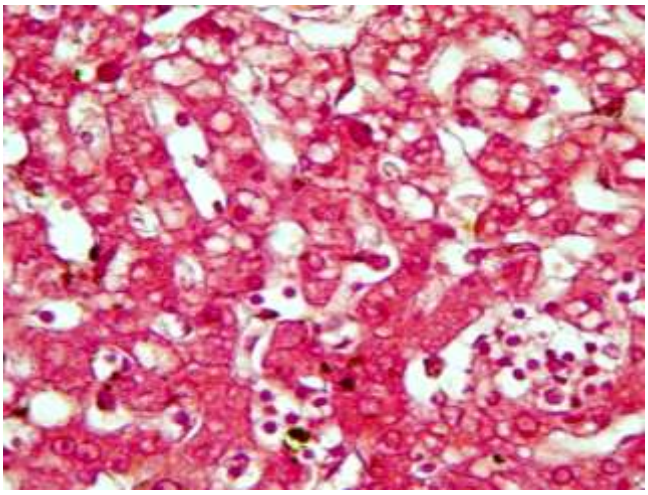

Fig. 3. Fat dystrophy of hepatocytes, nuclear vacuumization and lipocyte hyperplasia. Color: hematoxyline-eosin. Increasing: approx. 10, vol. 40.

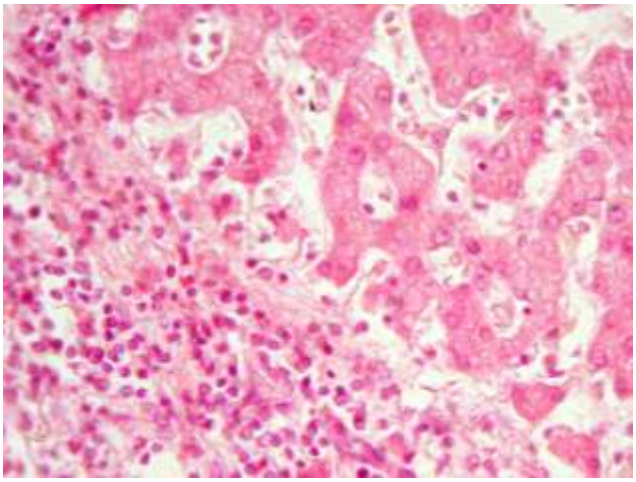

Fig. 4. Appearance of Mallory protein and lymphatic, leukocytic infiltration. Color: hematoxyline-eosin. Increasing: approx. 10, vol. 40.

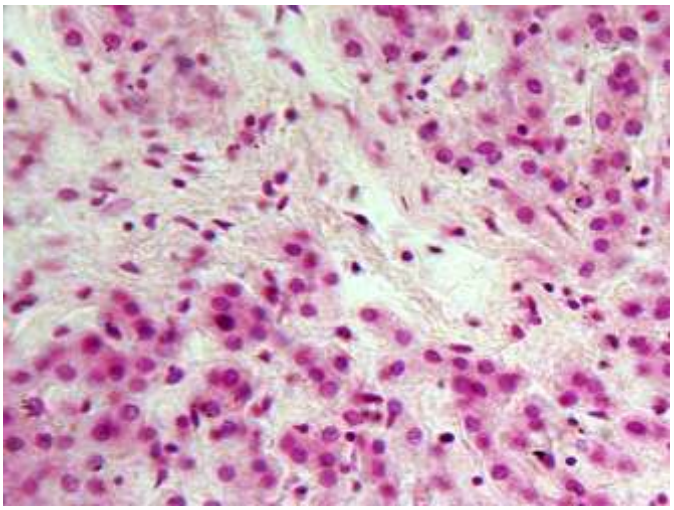

Fig. 5. Periportal sclerosis in chronic drug poisoning combined with alcohol. Color: hematoxyline-eosin. Increasing: approx. 10, vol. 40.

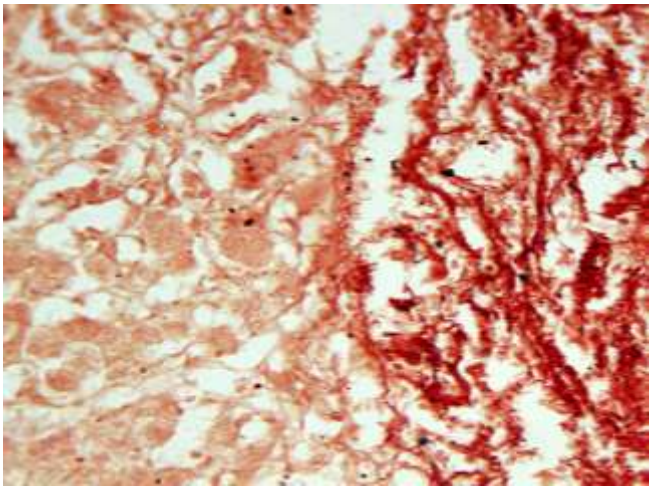

Fig. 6. Increasing collagen fiber content in periportal areas. Painting: Van Guizon. Increase: approx. 10, vol. 40.

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