COMMUNICATION ALCOHOLIC LIVER DAMAGE LEADS TO DISTURBANCE PORPHYRIN METABOLISM

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Purpose: Investigate the porphyrin metabolism caused by alcohol intoxication in rats.

Materials and methods used for the experiment, two groups of 20 rats each. First group every day for 90 days were injected intraperitoneally 40% ethanol, the second group control - healthy animals. Porphyrin exchange characterized by the level of coproporphyrin and uroporphyrin in urine; porphyrins in hepatocytes was determined by fluorescent microscopy. Prolonged administration of ethanol to rats results in the development of liver cirrhosis and disturbance of porphyrin metabolism in animals. After long-term administration of ethanol one group was divided into two subgroups: 1a - the rats that had disturbed porphyrin metabolism and 1b - animals that have found no such changes. Porphyrin exchange characterized by the level of p-aminolevulinic acid, porphobilinogen (Method Mauzerall and Granick), uroporphyrin (using Hoffman), coproporphyrin and uroporphyrin in urine (using Koskelo), the accumulation of porphyrins in the hepatocytes was determined by fluorescent microscopy. Urine collection, animals were placed in a special place - urinals. The daily amount of urine ranged from 2.5 to 18.5 ml. Porphyrins in the urine by a 1 every 3 days using a spectrophotometer SF-4F, pretreated urine by the method of Brugsch and Berman. Statistical processing of the results was performed by analysis of variance, t-Student test. Liver damage in various stages of intoxication studied morphological, morphometric and biochemical methods of investigation.

Results.The study showed that animals 1a group showed increases in protoporphyrins (21,6 ± 2,9 nmol per 1 g of creatinine, $P < 0.05$ versus control) and coproporphyrin (289,2 ± 29,9 nmol 1 g of creatinine, $p < 0.05$ versus control) in urine. Biochemical studies of the functional state of the liver showed a significant increase in total bilirubin in the group with abnormal porphyrin metabolism in comparison not only to control, but with the group without affecting porphyrin metabolism. Significantly increased rates of total lipids, cholesterol, and transaminases compared to intact animals. The results show that chronic administration of alcohol to animals not only leads to liver toxicity and the development of acute hepatitis, but also to disturbance of porphyrin metabolism. Such disturbances were observed only in some animals (4%, $p < 0.05$), which obviously had a genetic predisposition to gemoporhyria. Alcohol intoxication has identified this pathology.