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MOLECULAR MARKERS OF HEPATOCYTE INJURY: EXPRESSION OF BECLIN-1, BAX, AND GRP78 DURING CHRONIC ALCOHOL EXPOSURE IN FEMALE RATS

Background. The identification of molecular markers of liver pathology induced by chronic alcohol consumption is important for understanding the mechanisms of alcohol-induced liver injury, early diagnosis, and development of targeted, individualized therapy. Investigating the interplay between autophagy, apoptosis, and endoplasmic reticulum stress may facilitate the development of integrated biomarker panels reflecting the extent and nature of hepatocellular damage. **Methods.** The study was conducted on sexually mature female Wistar rats. Experimental rats received 20% ethanol as a sole source of fluid for 110 days. The activity of serum biochemical markers was measured using a biochemical analyser. The expression levels of the Beclin-1, BAX, caspase-3, and GRP78 in liver tissue were determined by Western blot analysis. Relative protein content was expressed as arbitrary units (a.u.) of optical density. **Results.** Long-term ethanol consumption causes hepatocellular injury, as evidenced by increases in serum the ALT, AST, and GGT by 3.6-, 2.1-, and 2.7-fold, respectively. Chronic alcohol intoxication resulted in pronounced complex alterations in hepatocytes. In particular, the relative expression levels of the proapoptotic protein BAX and caspase-3 in hepatocytes of the experimental group decreased 2.1- and 1.4-fold, respectively, compared with those in intact animals ($p < 0.05$). Concurrently, alterations in the functioning of the autophagy system were observed. The content of Beclin-1 protein decreased 1.71-fold compared with intact animals, which may indicate the initial stages of suppression of autophagic processes in hepatocytes under prolonged ethanol-induced toxic stress. In addition, a 1.77-fold reduction in the expression level of the endoplasmic reticulum stress marker, the chaperone GRP78, relative to the intact animal group ($p < 0.05$), presumably indicates exhaustion of the adaptive endoplasmic reticulum response to oxidative stress, potentially contributing to the hepatocyte death and disease progression. **Conclusions.** Rapid depletion of the GRP78 chaperone pool and disruption of the autophagic pathway in the setting of suppressed apoptosis may reflect the molecular mechanisms underlying the accelerated development of alcohol-associated liver injury. Considering the increased sensitivity of the female organism to the hepatotoxic effects of ethanol, evaluation of the expression of BAX, Beclin-1, and GRP78 may be considered the candidate molecular markers for determining the disease course and the risk of progression of alcohol-associated liver injury.

Keywords: apoptosis, autophagy, ER stress, alcohol intoxication.

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