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INVESTIGATION OF THE HEPATOPROTECTIVE EFFECT OF ANACARDIUM IN AN ETHANOL-INDUCED HEPATITIS MODEL

Akhmadova Maftuna

Master,

National University of Uzbekistan

maftuna19@gmail.com

Yunusova Muslima

Acting associate professor of the

National University of Uzbekistan, PhD

muslima8390@gmail.com

Seit-Asan Lenara

Research Intern at

National University of Uzbekistan

seitasanlenara@gmail.com

Mukhamedova Mukambar

Lecturer at the Lyceum at TashPMI

mmukambarbio@gmail.com

Abstract. This article presents the results of experimental studies on the hepatoprotective effects of the plant-derived polyphenolic compound anacardine in models of acute ethanol-induced liver injury. The relevance of the research lies in the limited availability and efficacy of local hepatoprotective drugs in the global pharmaceutical market, highlighting the need for effective, low-toxicity, and affordable plant-based alternatives. The study was conducted in accordance with international ethical standards using Wistar rats, and liver injury was induced using 33% ethanol. Anacardine was administered orally and intraperitoneally in various doses. The therapeutic efficacy was assessed by biochemical markers such as ALT, total protein, lipid peroxidation products (DCs and TBARS), and hepatoprotection coefficients. The results demonstrated that an oral dose of 100 mg/kg and an intraperitoneal dose of 5 mg/kg of anacardine showed the most significant improvement in liver function indicators, suggesting optimal hepatoprotective effects. Compared to standard treatment with Karsil, anacardine exhibited comparable or even superior results in some parameters. The findings support the potential use of anacardine as a natural hepatoprotective agent in liver injury associated with alcohol toxicity.

Keywords: anacardine, hepatoprotection, polyphenols, ethanol-induced liver injury, biochemical markers, experimental hepatology, oxidative stress.

Introduction. At present, the share of effective domestic hepatoprotectors in the global pharmaceutical market is small, and similar foreign medicinal products also constitute a limited proportion. Taking this into account, there is a growing need to discover new agents that can enhance liver resistance to toxic damage. In this context, preference should be given to herbal

preparations, which are generally characterized by low toxicity and a sufficiently broad and effective therapeutic action.

Materials and Methods. The studies were conducted in accordance with international recommendations of the European Convention for the protection of vertebrate animals used in experimental and other scientific purposes, using 240 mature white Wistar rats of both sexes, weighing between 170–280 grams [1].

Acute alcoholic liver damage was induced by intraperitoneal administration of a 33% ethanol solution. The experimental use of the polyphenol anacardine was performed using both oral and intraperitoneal routes of administration in the model animals.

The studied anacardine polyphenol was administered orally at doses of 100, 300, and 500 mg/kg body weight twice daily for 12 days through gastric intubation, and intraperitoneally at doses of 5, 15, and 25 mg/kg twice daily for 12 days. It should be noted that the tested polyphenol was administered 5 days prior to the introduction of the hepatotoxin (carbon tetrachloride or ethanol), and continued during the 7 days of toxin administration — that is, the polyphenol was given 1 hour before the injection of carbon tetrachloride or ethanol [3,7].

The functional activity of the liver was evaluated based on the duration of sleep in animals [4], which reflects the condition of the microsomal system involved in the metabolism of xenobiotics, particularly sodium ethaminal. The experiment was conducted according to the method of V.V. Gatsura [7]. After administering the investigational compound for 14 days to ethanol-induced model animals, sodium ethaminal was injected intraperitoneally at a dose of 40 mg/kg. The sleep duration (in the lateral position) was recorded in minutes. Statistical results were processed and expressed as the arithmetic mean (M) and its standard error (m).

Sodium ethaminal and the studied anacardin polyphenol were administered via the oral route at various doses (100, 300, and 500 mg/kg), and intraperitoneally at different doses (5, 15, 125 mg/kg). On the 14th day of administration, under conditions of acute toxic liver injury induced by ethanol solution, the compounds were introduced intraperitoneally at a dose of 40 mg/kg [6]. Animals receiving the same amount of purified water served as the control group.

Results. According to the obtained results, compared to the control group, administration of anacardin resulted in a significant and reliable reduction in sleep duration, with a maximum reduction observed at the 100 mg/kg dose — 55.7%. In our study, the focus was on determining the optimal therapeutic dose of the investigational compound that would normalize disrupted biochemical indicators in hepatocytes under ethanol-induced liver injury conditions.

In our experiments, the content of diene conjugates was initially determined. The concentration of diene conjugates was expressed in μmol/L. According to the results obtained in ethanol-induced liver pathology, when the dose was increased to 300 mg/kg, the total protein concentration in blood serum significantly increased by 22% compared to the control group, while the levels of DC (diene conjugates) and TBARS (thiobarbituric acid-reactive substances) decreased significantly by 102.29% and 131.22%, respectively. However, this effect was lower than that observed at the 100 mg/kg dose.

Further increasing the dose of anacardic acid to 500 mg/kg did not produce any additional positive effect on these parameters and did not result in statistically significant changes. Based on the hepatoprotection coefficient value and the degree of biochemical marker recovery, the highest coefficient (49%) was observed in animals receiving 100 mg/kg of anacardic acid. This value was

reliably higher than those observed at doses of 100, 300, and 500 mg/kg (which were 24%, 22%, and 18%, respectively).

In the next stage of the study, to determine the effective dose of anacardic acid when administered intraperitoneally and to confirm its efficacy at 5 mg/kg, the dynamics of biochemical marker changes were investigated in a model of ethanol-induced toxic liver injury at different doses (5, 15, and 25 mg/kg).

When the dose was increased to 15 mg/kg, the total protein level in blood serum rose by 22% compared to the control, while the levels of DC and TBARS decreased significantly by 102.29% and 131.22%, respectively. However, these values were still lower than those observed at the 5 mg/kg dose. A further increase in the dose to 25 mg/kg did not enhance the effects and did not lead to significant changes.

According to the hepatoprotection coefficient, taking into account the recovery of biochemical parameters, the highest coefficient (49%) was recorded in animals that received anacardic acid at a dose of 5 mg/kg. This was reliably higher than the coefficients observed at doses of 15 and 25 mg/kg (which were 24% and 22%, respectively).

The general parameters of the obtained data, including integral indicators of survival, detailed biochemical analysis of serum and liver, as well as data calculated by the hepatoprotection coefficient, allow suggesting a dose of 5 mg/kg of anacardine administered intraperitoneally as an effective therapeutic dose.

Conclusion. In ethanol-induced hepatitis models, comparison of the hepatoprotective effect of anacardine with Karsil showed that, relative to the control group, the concentration of diene conjugates in blood serum was corrected by 49%, and the content of thiobarbituric acid reactive substances (TBARS) was reduced by 36.4% in blood serum and by 41.7% in liver tissue. The increase in alanine aminotransferase (ALT) activity, as a marker of cytolysis, and the decrease in total protein levels were also normalized under the influence of the administered compound.

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