FEATURES OF THE GLUTATHIONE SYSTEM FUNCTIONING IN THE SCIATIC NERVES IN RATS WITH TOXIC PERIPHERAL NEUROPATHY RELATED TO THE DRUG KOKARNIT

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Toxic neuropathy is one of the peripheral neuropathy types that occurs because of the harmful effects of toxic substances on the peripheral nerves1. Due to the development of neuropathies with various genesis, different metabolic changes are observed, like the balance between pro- and antioxidant systems of the organism. The aim of this study was to investigate changes in the glutathione antioxidant system during development of paclitaxel-induced peripheral neuropathy, and the influence of the drug Kokarnit on these parameters.

Materials and Methods. The experiment was conducted on 40 white non-linear male rats, which were divided into 4 groups. Group 1 - intact animals, in groups 2-4 toxic neuropathy was modulated by paclitaxel in animals. Then group 2 had injections of the saline once per day during 9 days, group 3 - 0.5% lidocaine hydrochloride, group 4 - Kokarnit dissolved in 0.5% lidocaine hydrochloride (World Medicine) at a dose of 1 mg / kg. Content of reduced, oxidized glutathione and glutathione reductase was determined in nervus ischiadicus after autopsy.

Results. Development of neuropathy in experimental animals was confirmed by the Randall-Selitto test and the tail flick method. In the development of paclitaxel-induced neuropathy in animals, the content of reduced and oxidized glutathione decreased by 26% (p <0.01) and 53% (p <0.001), respectively, while glutathione reductase activity did not change significantly. Content of oxidized glutathione in animals treated with lidocaine was higher by 53% (p <0.05) than in animals with neuropathy. In rats which
were treated with Kokarnit, its content tended to increase compared to animals with neuropathy and was lower than in intact rats. The activity of glutathione reductase due to the administration of lidocaine did not change, but due to the administration of Kokarnit its values were 30% higher (p <0.05) than in intact animals. Development of toxic neuropathy in rats reduces the content of reduced and oxidized glutathione and does not change activity of glutathione reductase in the peripheral nerves. Kokarnit does not increase the glutathione content but increases the activity of glutathione reductase. In view of the obtained results, it is necessary to continue to study mechanisms of the toxic nephropathy development due to the Kokarnit effect.

References.


Key words: glutathione system, neuropathy, Kokarnit

Accepted for printing on 29 Oct 2017