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Białoszycka M. M., student Wydział Lekarski, Collegium Medicum *(Warmińsko-Mazurski Uniwersytet, Olsztyn, Polska)*
Scientific supervisor: Simonova I.V., senior lecturer of foreign languages department *(Vinnytsia National Pirogov Memorial Medical University, Vinnytsia, Ukraine)*

METHOD FOR CREATING CONDITIONS TO INDUCE ITS INTRINSIC ULCEROGENIC EFFECT OF DICLOFENAC (A NON-STEROIDAL ANTI-INFLAMMATORY DRUG) IN EXPERIMENTAL STUDIES ON RATS

It is well known that diclofenac, (a non-steroidal anti-inflammatory drug /NSAID), can cause damage to the stomach lining when administered orally. Such damage can range from erosions to ulcers of the gastric mucosa, with potential complications like perforation and bleeding. Given the widespread use of NSAIDs and the large variety of these drugs, there is a constant search for methods and approaches to mitigate their side effects, including their ulcerogenic effects. An essential component of such studies is the creation of experimental models of NSAID-induced ulceration in animals. These experiments are most commonly conducted on rats, with the drug being administered directly into the stomach via a probe at a specific dose, according to a scheme designed to model acute or subchronic ulcers. It is known that both humans and animals have a natural mechanism for protecting the gastric mucosa, in which mucin and mucus produced by the gastric lining cells play a key role. To enable diclofenac to exert its intrinsic ulcerogenic effect, neutralizing the protective influence of mucin and mucus on the gastric mucosa of the test animals were proposed. This can be achieved by introducing a 0.5-10% aqueous suspension of high-dispersion silica into the stomach cavity via a probe at a dose of 25-50 mg/kg [1], 20 minutes before administering the NSAID under study. High-dispersion silica quickly adsorbs mucin, the primary component of protective mucus, leading to accelerated absorption of diclofenac in the stomach [1]. The accelerated absorption of the NSAID is accompanied by its metabolic transformations, which, in turn, speed up changes in the gastric mucosa, leading to the development of erosions and ulcers. We selected a subchronic ulcer model for our study, involving the administration of the drug over 5 days. The research was conducted on 30 non-linear Wistar rats with an initial body weight of 210-260 grams, divided into three groups: 1 – control animals (intact), 2 – animals with NSAID-induced gastric ulcers (7 mg/kg of diclofenac for 5 days), and 3 – rats that received an aqueous suspension of silica at a dose of 25 mg/kg 20 minutes before diclofenac administration for 5 days. After euthanizing the rats, their stomachs were removed, and the extent of ulcerative lesions in the mucosa was quantitatively assessed [2]. In the group of animals pretreated with silica, the degree of ulceration was the most severe. Thus, the preliminary administration of the aqueous silica suspension into the stomach of rats during subchronic diclofenac use enhances the drug's ulcerogenic effect, providing a suitable model for experimentally replicating this pathology

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