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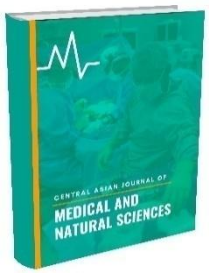

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Morphological Changes of the Stomach in Chronic Gastritis

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Abstract: In the work, 80 outbred white outbred rats of four months of age, weighing 170-200 g of the gastric mucosa with experimental gastritis, were studied. Animals were taken out of the experiment at 30 days of age by instantaneous decapitation of animals under ether anesthesia. After opening the abdominal cavity, microscopic changes in the stomach were examined.

Morphological studies of stomach tissues were performed under a Lake microscope. In the control groups of rats, the total thickness of the stomach wall in its cardia and at the junction with the duodenum remained unchanged; the mucosa and submucosa were well developed. In the second group of animals, when using non-steroidal anti-inflammatory drugs, dystrophic changes, desquamation of the mucous membrane prevailed, in some places hemorrhage and plethora of blood vessels, and the number of glandular structures began to decrease.

Keywords: atrophic gastritis, rats, stomach, morphology.

Introduction

Chronic gastritis is a chronic disease, perhaps the most common internal human disease, which, in terms of prevalence, severity, complications, occupies the main place among diseases of the gastrointestinal tract. The prevalence of chronic gastritis in the world population is very high and ranges from 50 to 80%. In Russia, this figure is at the same level. [5,7,8]. Chronic gastritis was first described by the German physician G.E. Stahl in 1728, who revealed an inflammatory process in the gastric mucosa. In the past, many scientists believed that chronic gastritis was a histological finding, but not a disease. In 1982, Australian scientists B. Marshall and R. Warren discovered H. pylori and suggested that this microorganism is capable of causing chronic gastritis. The role of the bacterium in the development of chronic gastritis was fully proven in 1983.

The highest frequency of its occurrence is in highly developed countries. Most often, this pathology occurs in people of working age (from 40-50 years). To date, a significant influence of the nature of nutrition on the development, functional and adaptive properties of the organs of the digestive system of humans and animals in ontogenesis has been shown [4,6,10]. At the same time, the main attention in such studies was paid to the chemical composition of food and diet.

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Thus, the study and development of issues of early diagnosis, prevention and treatment of chronic gastritis are redirected to reduce the incidence of this pathology.

Purpose of the study. Based on the above, we were to summarize our observations in the study of chronic gastritis and study an experimental model of chemical gastritis caused by non-steroidal anti-inflammatory drugs.

The material of the study was 80 outbreed white outbreed rats of four months of age, weighing 170-200 g. Animals of the control group were kept under normal vivarium conditions on natural food for rodents, the basis of which was whole wheat grain, as well as vegetables cut into large pieces. Group I: The control group received a daily oral dose of 1 ml of isotonic saline for seven days. Group II: Animals received two doses of paracetamol (100 ml/kg) intraperitoneally, the first dose on the 1st day of the experiment and the second dose on the 4th day of the experiment for seven days. During the experiment, the behavior of animals, their appearance, body weight were evaluated. The object of the study was the stomach, which was fixed in Karnau's solution, prepared from paraffin blocks, micro preparations 5-7 mm thick using an MPS-2 sledge microtome, then stained with hematoxylin and eosin.

Results and discussion. An experimental model of chemical gastritis was developed, which makes it possible to identify structural and functional changes in the gastric mucosa and muscular membrane in animals with long-term use of non-steroidal anti-inflammatory drugs. The results of the experiment showed that in animals, due to the use of non-steroidal anti-inflammatory drugs, the following changes are observed. When studying the control group of biopsies, it was found that the normal gastric mucosa remained largely unchanged. Group II: when animals were injected intraperitoneally with two doses of paracetamol (100 ml / kg), the first dose on the 1st day of the experiment, and the second dose on the 4th day of the experiment, the following changes were noted for seven days, signs of superficial gastritis, mucus, followed by a decrease in the number of glandular elements (Fig. No. 1 and 2).

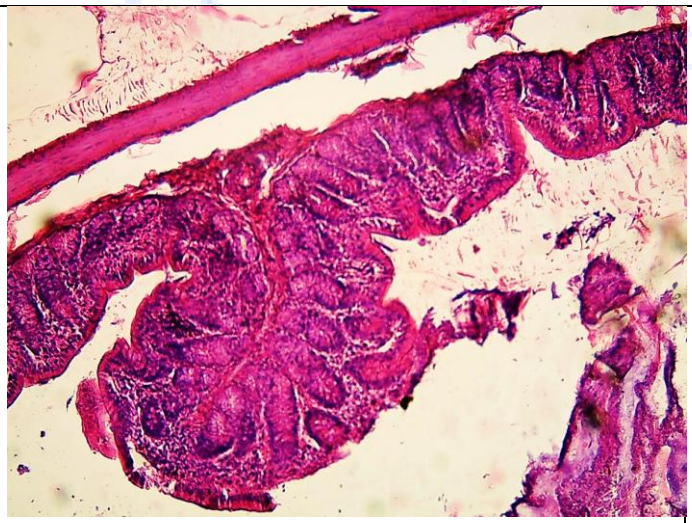

Figure 1. The mucous membrane of rats, the first day of the experiment. Desquamation of the surface epithelium, edema and plethora. Env.G-E. SW: 10x20.

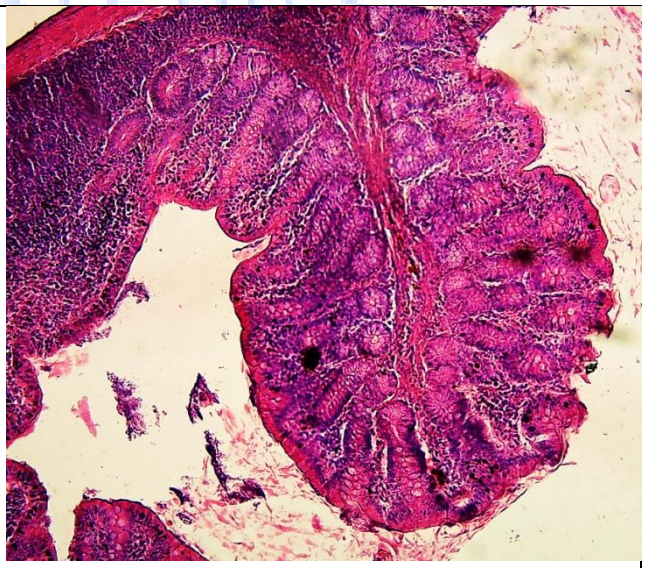

Figure 2: The mucous membrane of rats, multiple hemorrhage and dystrophic and necrotic changes in the epithelium, a pronounced inflammatory infiltrate of the submucosa. Duration 7 days. G-E. SW: 10x20.

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In animals at various stages of the disease, microscopy revealed a thickening of the mucous membrane due to hyperemia and edema, punctuate hemorrhages, and acute erosions. Depending on the duration of action of NSAIDs, lymphohistiocytic infiltrate began to progress and atrophic and sclerotic changes were added.

Conclusions. The study shows that in chemical gastritis, morphological changes begin with superficial gastritis and then progress depending on the action of NSAIDs. In animals at various stages of the disease, microscopy revealed a thickening of the mucous membrane due to hyperemia and edema, punctate hemorrhages, acute erosions. Depending on the duration of the action of NSAIDs, atrophic and sclerotic changes began to progress and joined. The highest frequency of chemical gastritis was found in animals in recent days, and atrophic gastritis in animals is characterized by a lack of specificity.

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