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Morphological Changes in the Colon Wall of White Rats in Experimental Pneumosclerosis

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ABSTRACT

This article presents morphological changes in the colon wall of white-bred rats in experimental pneumosclerosis.

KEYWORDS: experiment, pneumosclerosis, white-bred rats, colon wall, morphology.

Morphological and morphometric changes were detected in the colon tissue of white-bred rats in the experimental group on the 32nd day of the development of experimental pneumosclerosis in the lungs. According to the results of morphological examination, a violation of the normal 4-layer structure of the intestine was observed. There were significant changes in the cellular composition of the mucous membrane of the colon of the control group. The death of tall columnar epitheliocytes and goblet cells was observed. The thickness of the wall surrounding the crypts in the lamina propria of the mucosa was 3.1-5.3 μm in the control group, while in the experimental group this indicator increased to 4.2-6.9 μm.

Unlike the control group, when the crypts were viewed along the area of the lamina propria, as a result of the death of several crypts, the formation of cavities was observed, and inside these cavities, leukocyte cells were accumulated, indicating that an inflammatory process was taking place.

was 100 μm. Under the microscope, the crypts were characterized by weakly oxyphilic cytoplasm, and the goblet cells underwent dystrophic changes. The number of goblet cells in each crypt was 6-8, which indicates a decrease compared to the control group. The number of lymphocytes and macrophages in the field of view increased, with 75 lymphocytes counted in one field of view.

Although the muscularis mucosae had a 2-layer structure, the muscularis mucosae showed signs of degradation. The submucosa showed signs of inflammation and was filled with loose fibrous connective tissue, fat cells, and a large number of fibroblasts. The total thickness of the submucosa and muscularis mucosae averaged 560.87 μm, which is higher than in the control group. In the inner muscularis mucosae, fiber disorganization was observed, including an increase in blood vessels and nerve fibers.

Such changes are considered to be the toxic effect of nitrogen dioxide (NO2) applied to the colon

for the development of experimental pulmonary pneumosclerosis.

Analysis of morphometric parameters in the colonic tissues of white-bred rats between the control and experimental groups (pneumosclerosis induced by nitrous oxide) showed significant changes. In the experimental group, a clear thickening of the walls of the colonic crypts was observed: in the range of 4.2–6.9 μm (in the control group, this indicator was 3.1–5.1 μm). The diameter of the crypts was significantly reduced: in the experimental group, it was 2.5–4.3 μm, and in the control group, it was 9.4–9.7 μm. Also, when the submucosa and muscularis mucosa were measured together, a thickening was also detected - in the experimental group, this indicator was 560.87 μm, and in the control group, it was 492.43 μm. A decrease in the number of goblet cells in the crypts was also observed. In the experimental group, this indicator was in the range of 6–8, and in the control group, it was 7–19. A decrease in the number of goblet cells indicates a violation of the secretory function of the intestine, which indicates the occurrence of pathological processes.

In addition, a significant increase in the number of leukocytes between the crypts was observed: in the experimental group, their number was 117, while in the control group this indicator was 57. Such an increase in the number of leukocytes indicates the presence of a strong inflammatory process in the tissues, which is a consequence of the pneumosclerosis-inducing effect of nitrogen oxide. These changes confirm the occurrence of pathological processes in the tissues and indicate that the factors of influence used in the experimental conditions lead to morphological changes.

These results prove that factors that cause pneumosclerosis in experimental conditions significantly affect the morphometric and functional state of the intestine, allowing us to conclude that physiological processes are disrupted and inflammatory reactions are activated.

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