

Renal Morphology in Experimental Pulmonary Pneumosclerosis

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Abstract: In clinical practice, special importance is attached to the elimination of pathological processes that occur in the human body after damage to lung tissue. Currently, among the various problems of clinical pulmonology, the world continues to attract the attention of researchers due to the constantly growing number of patients with fibrosis of the lungs, difficulties in diagnosing patients and insufficient development of treatment schemes. At present, pulmonary fibrosis – patients with respiratory failure and chronic pulmonary heart disease, and complications of internal organs, especially in the gastrointestinal system, are noteworthy, requiring that measures should be taken to predict the nature of the disease in advance, to reduce the share of disability among patients.

Keywords: pulmonary fibrosis, morphology, kidney, glomerulonephritis.

Research objective: study and analysis of renal tissue morphology in experimental pulmonary pneumosclerosis.

Material and methods: the study used 50 rats, which were separated by age into monthly as well as their corresponding control groups. In order to achieve the modeling of pulmonary fibrosis, white-breed rats were selected and the experimental model "pulmonary fibrosis with nitrogen dioxide through the breathing of animals from toxic gases in closed boxing" was used. The room in which the rats were kept complied with the requirements for the conditions of keeping this type of laboratory animal (t 20-24 Co, humidity 60%, light/darkness 12 hours/12 hours). Water was given without restrictions, and when feeding animals all sexually mature rats were quarantined for a week, transferred to a normal vivarium regime after somatic or infectious diseases were excluded.

Before starting the experiment, the animals underwent a two-week quarantine and were trained to be in the experimental chamber (BGMI at the Ethics Committee and Commission requirements for the control and use of laboratory animals). The first group of rats (intakt) was a control group, White broodless rats fed on a simple standard diet to compare the results in the experiment with another group. And in the second group of our experiment, white non-breeding rats caused pulmonary pneumosclerosis for 20 days.

The reaction of the metal with nitric acid resulted in the release of vapors of the toxic substance nitrogen dioxide, which caused inflammatory connective tissue changes in the lung parenchyma when prolonged poisoning for 20 days. The concentration of the poisoning substance in the air was released by chemical and mathematical equations. Animals were coughed up for 30 minutes with an interval of three times a day, 15 minutes between poisonings to the effect of the poisoning substance. Ventilation intervals were carried out in order to release the accumulated carbon dioxide gas, which is released in the release of animal breath.

Table № 1 Distribution of animals according to the content of experience

Groups (c-control, e-experience)	Experience content	Animal youth	Total number of animals (*extinct rats number)
		3month	
I c	Control	20	20
II e	White rats caused by pulmonary pneumosclerosis for 20 days	30(5*)	30(5*)
total		50(5*)	50(5*)

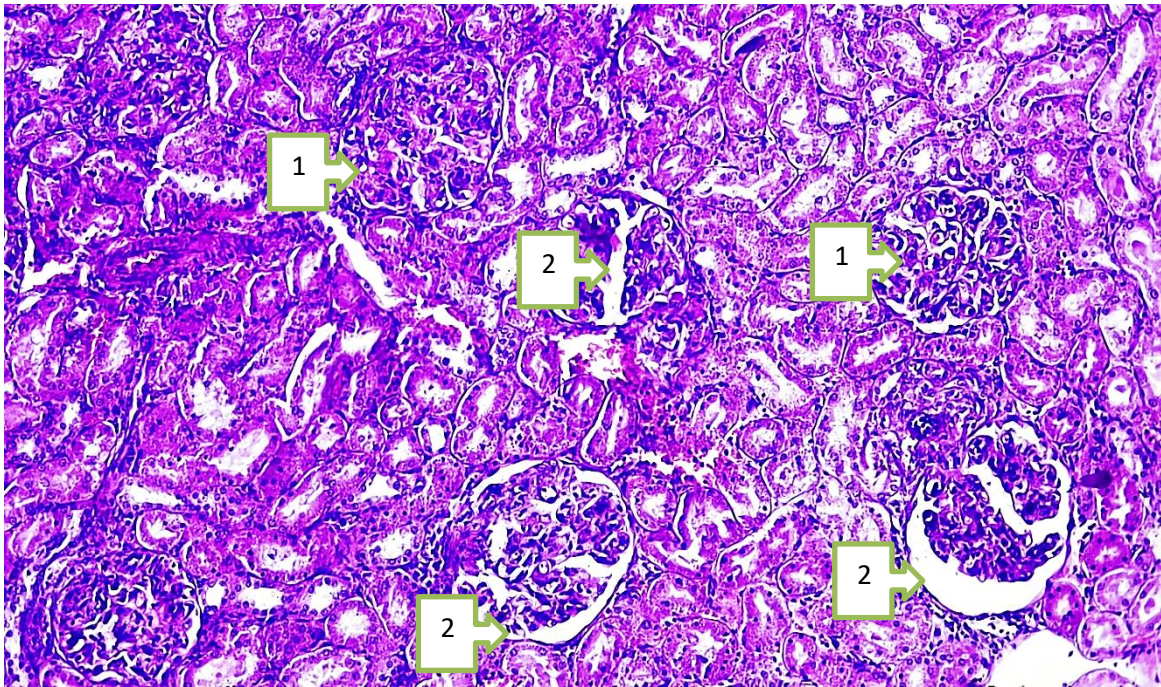
After the animals were decapitated on an empty stomach, the abdominal cavity was opened, the kidneys were separated, the organ was cut in half and fixed in a 10% formalin neutralized solution, hardened for 72 hours, then washed in running water for 2 hours, passed through alcohols with an increased concentration for dehydration, and bricks were prepared with paraffin pouring. 5-8 μm thick incisions were made from them and the overall histological structure was studied by staining in hematoxylin-eosin dye. The cuts were studied under the light microscope of the firm "Leyka" and pictures were taken from their desired areas. Micropreparations were photographed under a microscope measuring 4x10, 10x10, 40x10, 100x10.

Results and conclusions

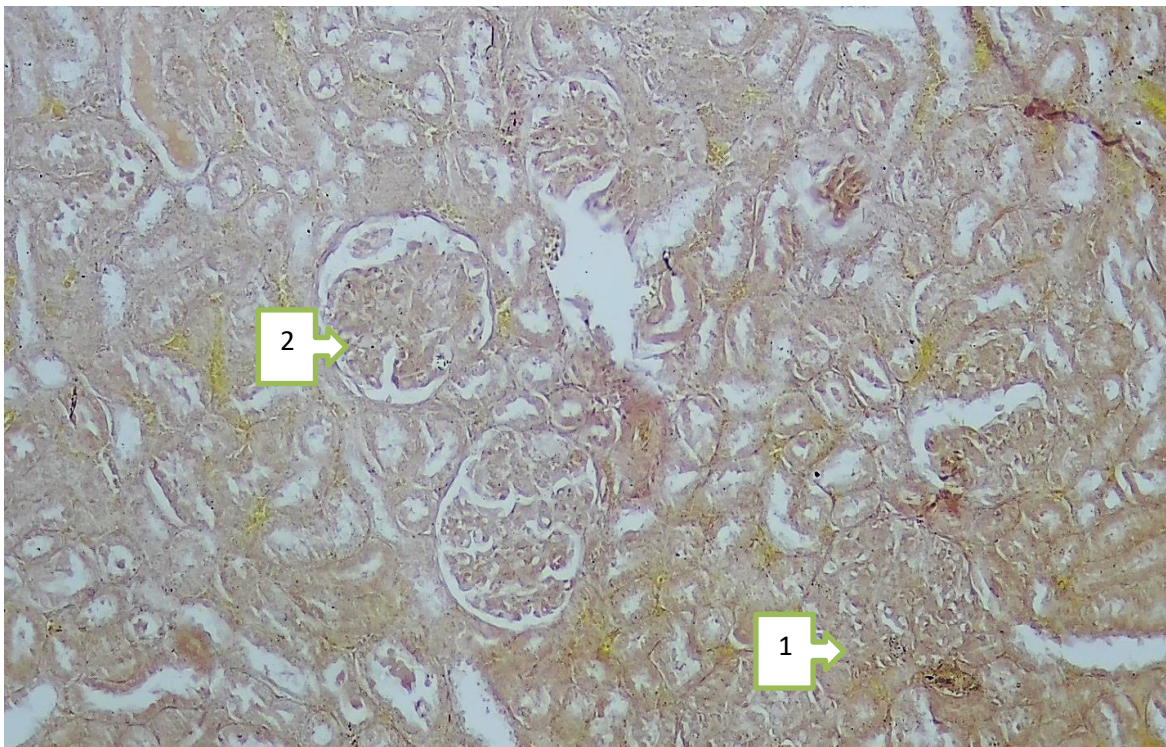


Macroscopic view of the kidney

As a result of the study, it was found that white non-breeding rats belonging to the first main group did not visually observe pronounced changes in the external macroscopic appearance of the kidney organ, when viewed microscopically, pathomorphological signs characteristic of membrane-proliferative glomerulonephritis (figure 2-3) were observed in the kidney tissue. In particular, it was observed that at the expense of the apparent proliferation of mesangiocytes, the Coptic appeared multicellular and dilated, the mesangial Matrix expanded, the cells inside the Coptic appeared fragmented at the expense of proliferative and sclerotic changes, morphological changes characteristic of neutrophilic inflammation appeared, dystrophic and necrobiotic changes appeared in the proximal and distal canals.



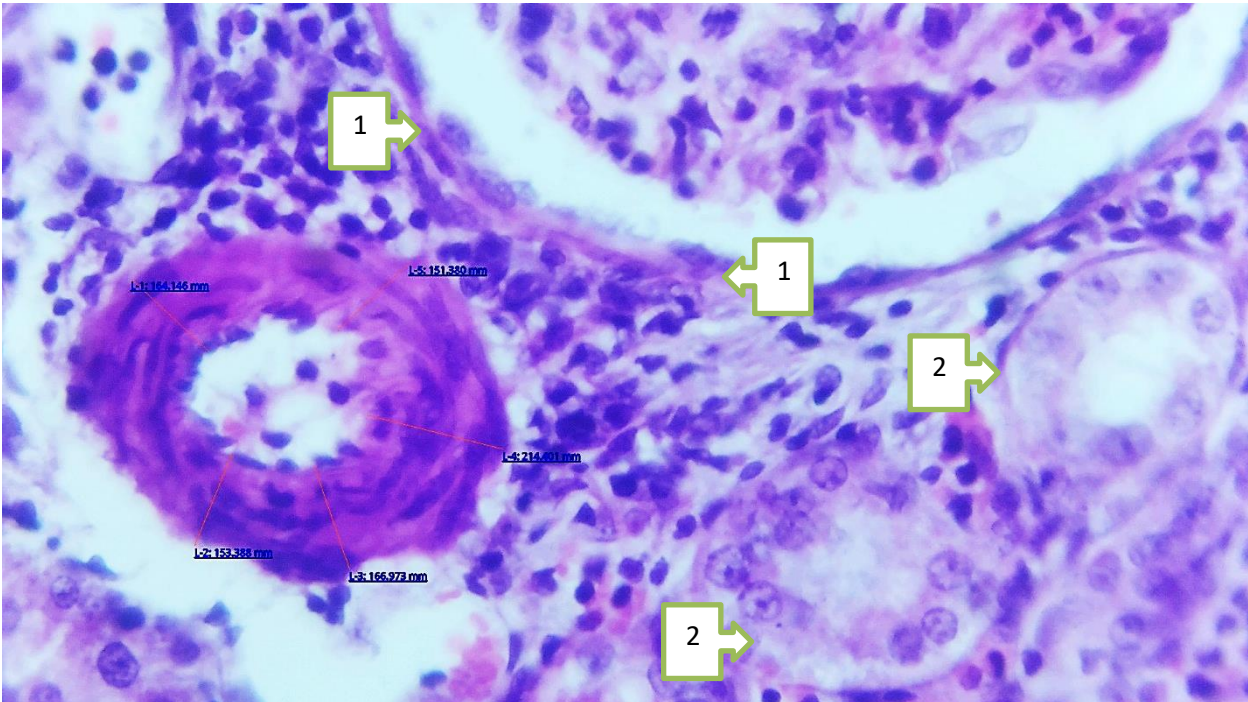
Picture1. Microscopic view of white-breed rat kidney tissue. Membrane-proliferative glomerulonephritis. 1. At the expense of the obvious proliferation of mesangiocytes, the balls appear multicellular and dilated, the mesangial matrix is enlarged. 2. At the expense of proliferative and sclerotic changes, the cells inside the clutch appear to be fragmented. Paint G-E. AB 4x18 ok.



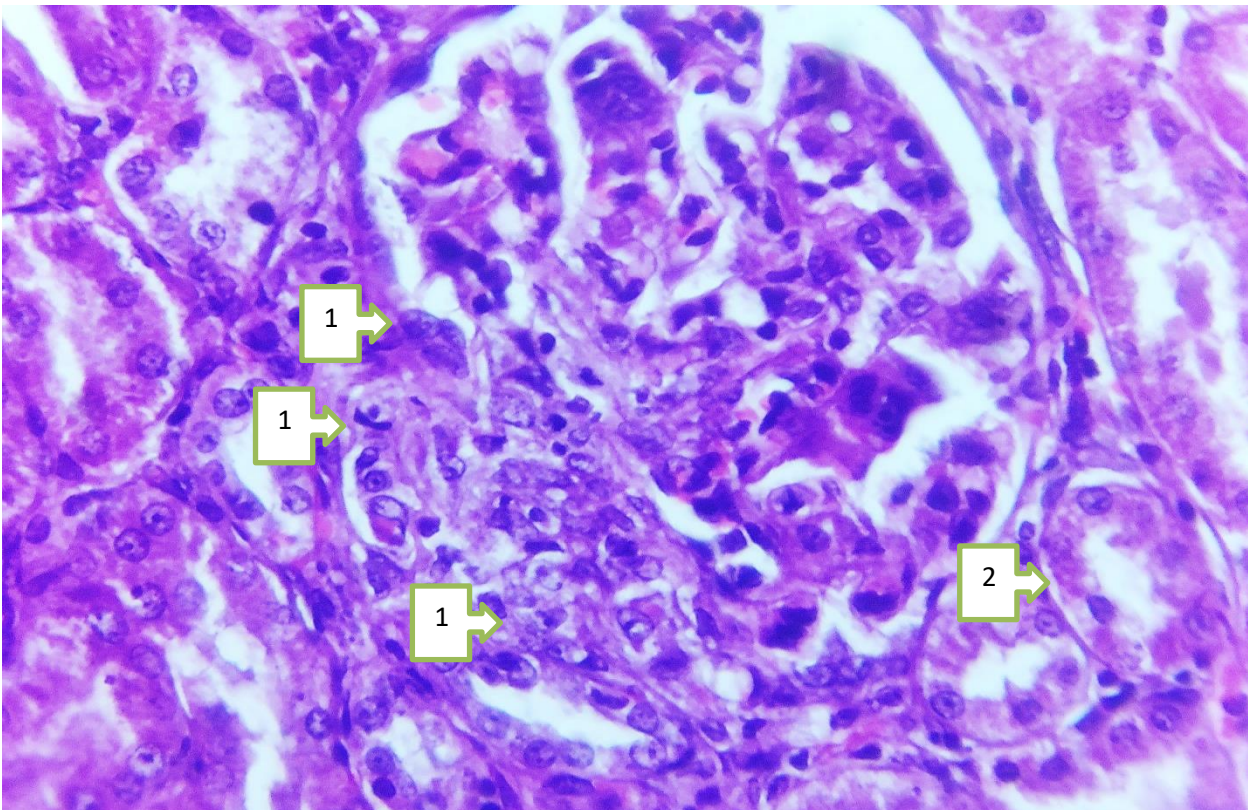
Picture 2. Microscopic view of white-breed rat kidney tissue. Membrane-proliferative glomerulonephritis. 1. At the expense of the obvious proliferation of mesangiocytes, the balls appear multicellular and dilated, the mesangial matrix is enlarged. 2. At the expense of proliferative and sclerotic changes, the cells inside the clutch appear to be fragmented. Paint van-Gison. AB 4x18 ok.

Micropreparations made from experimental animal kidneys, as can be seen from the picture of histological drug in the mirror of the quoted item, it was observed that there were morphological changes characteristic of inflammation in the kidney tissue, diffuse expansion of the basal membrane,

along with changes characteristic of most membranoproliferative glomerulonephritis, and leukocytic infiltration in the surrounding tissue, dystrophic-necrobiotic changes in the proximal and distal These signs are considered to be characteristic of membranous nephropathy.



Picture 3. Microscopic view of white-breed rat kidney tissue. Membranoproliferative glomerulonephritis. 1. Diffuse expansion of the basal membrane and leukocyte infiltration in the surrounding tissue. 2. Dystrophic-necrobiotic changes in proximal and distal ducts. Painted with hematoxylin-eosin, OB 40x20 ok.



Picture 4. Microscopic view of white-breed rat kidney tissue. Membranous Glomerulonephritis. 1. Accumulation of deposits in the basal membrane (immunocomplexes). 2. Dystrophic necrobiotic changes in the ducts. Painted with hematoxylin-eosin, ob 4x20 ok.

In conclusion, under the conditions of the experiment, the first group of white non-breeding rats, in which pulmonary pneumosclerosis was called, were found to have various morphological changes in kidney tissue. These were observed to appear multicellular and dilated at the expense of apparent proliferation of mesangiocytes in kidney tissue, mesangial Matrix dilated, proliferative and sclerotic changes at the expense of cell cleavage within the cap, neutrophilic inflammatory-specific morphological changes appeared, dystrophic and necrobiotic changes appeared in proximal and distal ducts.

The second group of white-noded bats found signs of nodular periarteriitis in the surrounding tissue due to dystrophic-necrobiotic changes in proximal and distal ducts, appearance, including proliferative and sclerotic changes in cells within the coptochap, diffuse expansion of the basal membrane, and leukocyte infiltration in the surrounding tissue.

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