



THE MORPHOLOGICAL FEATURES OBSERVED IN RATS FOLLOWING THE INDUCTION OF DESTRUCTIVE PNEUMONIA ARE CHARACTERIZED BY A DYNAMIC PROGRESSION OF ACUTE INFLAMMATION, TISSUE DESTRUCTION, AND SUBSEQUENT REPARATIVE (FIBROTIC) CHANGES IN THE LUNG PARENCHYMA AND PLEURA

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<https://doi.org/10.5281/zenodo.17865429>

ARTICLE INFO

Received: 07th December 2025

Accepted: 08th December 2025

Online: 09th December 2025

KEYWORDS

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ABSTRACT

currently, respiratory system diseases occupy a leading position among pediatric pathologies. Pneumonia in children is a form of acute respiratory infection affecting the lung parenchyma, considered a severe, septic disease accompanied by inflammation in the alveoli, alveolar septa, and the terminal part of the bronchial tree [3, 4, 5]. The current relevance of the disease lies in its observation across all age groups of children, accompanied by purulent inflammation in the lungs and pleura. Simultaneously, there are certain problems in its prevention, diagnosis, and treatment processes [1, 2, 3, 6].

Relevance: currently, respiratory system diseases occupy a leading position among pediatric pathologies. Pneumonia in children is a form of acute respiratory infection affecting the lung parenchyma, considered a severe, septic disease accompanied by inflammation in the alveoli, alveolar septa, and the terminal part of the bronchial tree [3, 4, 5]. The current relevance of the disease lies in its observation across all age groups of children, accompanied by purulent inflammation in the lungs and pleura. Simultaneously, there are certain problems in its prevention, diagnosis, and treatment processes [1, 2, 3, 6].

Research aim: to determine the dynamics of pathological changes in the lungs of rats with induced destructive pneumonia through morphological, morphometric, and histochemical study using the Hematoxylin-eosin and Van Gieson methods, establishing the correlation between inflammation (infiltration), destruction (bullae formation), and reparative processes (fibrosis).

Research methods and methodology: Taking into account the various principles of modern scientific knowledge, we developed our research methodology to adequately address the stated objective. The planned and conducted studies, based on general scientific and specific methods, were aimed at solving the posed tasks.

Research results:

On the 7th day after modeling destructive experimental inflammation in the lung parenchyma of rats, the lung tissue appeared swollen, relatively enlarged, and denser. Redness, vascular dilation, and infiltration were detected on the lung surface. The pleura was

preserved, with thickening of fibrous tissue, venous fullness of blood vessels, vascular congestion in the parenchyma, and a small amount of serous-hemorrhagic fluid in the pleural cavity.

Upon examining a lung fragment on the 7th day of the experiment using the Hematoxylin-eosin staining method, the following changes were observed.

Moderate vascular congestion of the lung parenchyma and interalveolar septal vessels was observed.

On the 14th day of the study, congestion of the lung parenchyma and interalveolar vessels was observed. Inter-alveolar septa were infiltrated by lymphocytes, neutrophils, and eosinophils. Clear focal infiltration in the lung tissues, alveolar septum, and around the alveolar ducts indicated an inflammatory process characteristic of destructive pneumonia. Alveolar septal erosion—air-filled expansion of alveolar spaces, i.e., bulla—was observed in the lung tissue. Fibrous changes indicated the replacement of inflamed areas of the lung with connective tissue.

On the 21st day of the study, vascular congestion of the lung parenchyma was observed, with interalveolar septa infiltrated by lymphocytes, neutrophils, and eosinophils. Clear focal infiltration in the lung tissues, alveolar septum, and around the alveolar ducts indicated an inflammatory process characteristic of destructive pneumonia.

Conclusions:

1. In the experimental destructive pneumonia model (in rats), the simultaneous progression of destructive-inflammatory and regenerative-fibrotic processes was observed in the lungs from day 7 to day 21 of the disease. Analysis conducted with Hematoxylin-eosin staining showed inflammation-specific infiltration in the lung tissue (lymphocytes, neutrophils, macrophages), vascular congestion, and the formation of bullae (air-filled expansion) resulting from the destruction (erosion) of alveolar septa.

2. The results of Van Gieson staining indicated that fibrous changes begin as early as the early stages of inflammation (day 7) in the lung tissue. Thickening of the interalveolar septa and blood vessel walls, and an increase in collagen fibers in the intercellular spaces (especially on days 14 and 21) were noted.

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