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Morphological Changes of Internal Organs in Experimental Interstitial Lung Lesions

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ABSTRACT

This study presents the results of a pathomorphological investigation of internal organs in rats with experimentally induced interstitial pneumonia caused by chronic tobacco smoke exposure. Pronounced inflammatory-destructive changes were revealed in the lung tissue, including peribronchial sclerosis, lymphoid infiltration, and emphysematous alterations. In addition to pulmonary pathology, dystrophic changes were observed in the liver, kidneys, heart, and brain, indicating systemic inflammatory response and multiple organ dysfunction. These findings highlight the pathogenetic role of chronic inflammation and provide a basis for further studies and therapeutic assessment.

Keywords: *interstitial pneumonia, tobacco smoke, pathomorphology, inflammation, internal organs, rats.*

INTRODUCTION

Interstitial lung diseases, including interstitial lung lesions, represent a serious clinical problem characterized by a chronic course, progressive fibrosis, and a high risk of multiple organ failure [1]. Particular attention is given not only to pulmonary pathology, but also to systemic alterations occurring in the body during chronic inflammation [2]. Pathomorphological changes in organs and tissues in interstitial pneumonia reflect complex pathogenetic mechanisms involving activation of the systemic inflammatory response, oxidative stress, and disturbances of metabolic processes [4].

One of the most reliable methods for reproducing interstitial pneumonia in experimental settings is the model of chronic exposure to tobacco smoke using the Kurlandsky chamber. This technique was first developed and proposed for the study of respiratory system pathologies, including chronic obstructive pulmonary disease and interstitial pneumonia [3]. The Kurlandsky chamber provides stable and controlled exposure of laboratory animals to tobacco smoke, enabling the modeling of inflammatory processes similar to those occurring in the human respiratory tract during long-term smoking [6].

The Kurlandsky chamber is a specialized device equipped with an automated cigarette combustion system, high-resolution optical instruments for tissue analysis, and a system for controlling smoke concentration and ventilation. The use of this chamber makes it possible to obtain high-quality images, study morphological and biochemical changes in lung tissues at the cellular and subcellular levels, and evaluate the effectiveness of various treatment methods [3]. This approach is widely applied in biomedical research as an effective model of chronic respiratory diseases.

The aim of the present study was to investigate morphofunctional changes in tissues and internal organs in an experimental rat model of interstitial lung injury in-

duced by chronic tobacco smoke exposure, followed by an analysis of the effectiveness of different therapeutic approaches.

MATERIALS AND METHODS

A total of 164 outbred white rats weighing 150–200 g were used to reproduce interstitial lung injury. The pathological model was established by daily exposure to tobacco smoke in a Kurlandsky chamber for 60 days. Smoke was generated by burning commercially available Pall Mall cigarettes (Uzbekistan), ensuring stable concentrations of nicotine and combustion products. The control group consisted of intact animals.

Morphological methods. To assess the effects of the studied agents, morphological examination of internal organs was performed at the Republican Pathoanatomical Center of the Ministry of Health of the Republic of Uzbekistan (Director — Prof. D.A. Nishanov, Consultant — Prof. Kh.Z. Tursunova). After euthanasia, internal organs were dissected, and tissue fragments sized 0.5 × 0.5 cm were excised.

Tissues were fixed in 10% neutral buffered formalin, dehydrated in graded ethanol, and embedded in paraffin. Histological sections of 5 μm thickness were prepared using a Leica rotary microtome and stained with hematoxylin and eosin. Cell nuclei were stained dark blue-black, and the cytoplasm appeared dark violet.

Ethical standards. The study was conducted in accordance with ethical principles and international guidelines for the care and use of laboratory animals.

RESULTS

Microscopic examination of rat lung tissue stained with hematoxylin and eosin revealed a preserved parenchymal architecture. Alveolar ducts and alveoli had thin interalveolar septa lined with a single layer of squamous epithelium. The alveolar lumina were free, filled with air, without evidence of exudation or infiltration. Interalveolar capillaries were moderately congested, and endothelial cells remained intact.

The structure of terminal and respiratory bronchioles was clearly distinguishable: the mucosa was lined with a single layer of cuboidal epithelium without dystrophic changes; no mucous or cellular content was present in the lumina. Peribronchial and perivascular connective tissue structures showed no signs of edema or cellular infiltration. Cartilaginous plates of the bronchi and bundles of smooth muscle fibers were preserved, with no pathological alterations. Overall, the morphological picture corresponded to that of normal lung tissue in healthy animals (Fig. 1).

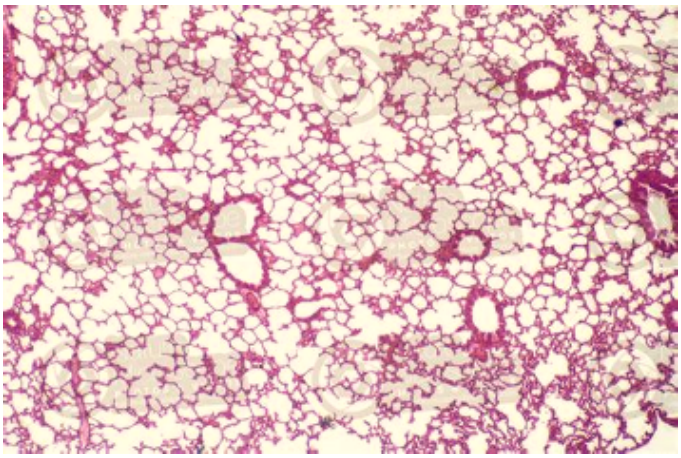

Fig. 1. Rat lung (control). Preserved histoarchitecture of alveolar tissue. Staining: hematoxylin and eosin. Magnification ×100.

Morphological changes were naturally most pronounced in the lungs and were characterized by chronic interstitial bronchopneumonia (Figs. 2, 3, 4, 5).

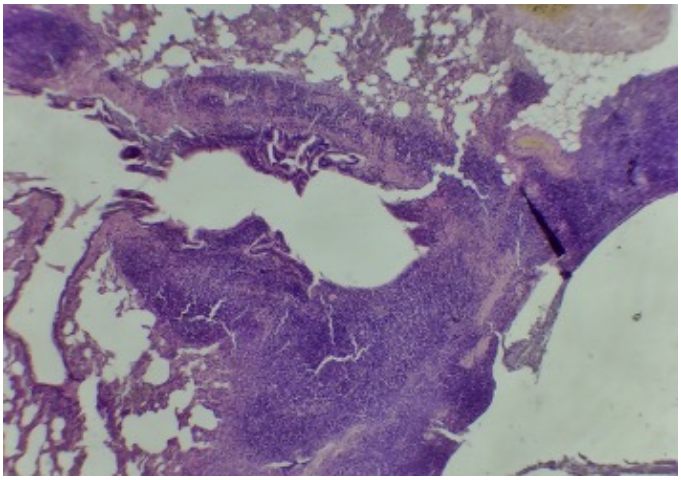

Fig. 2. Thickened bronchial wall due to sclerosis and inflammatory infiltration; proliferation of bronchial epithelium; hyperplasia of peribronchial lymphoid tissue. Staining: hematoxylin and eosin. Magnification 10×40.

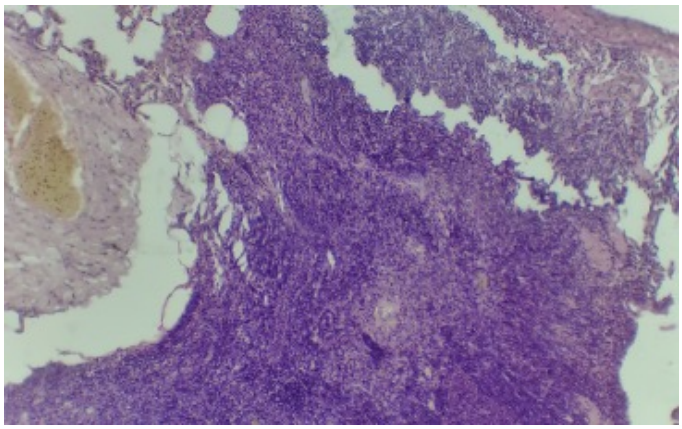

Fig. 3. Thin pleura; alveoli free and air-filled; congestion observed; bronchi and bronchioles free; epithelium single-layered columnar; interstitial infiltration, peribronchial and perivascular lymphoid infiltration, and hyperplasia of peribronchial lymphoid tissue. Staining: hematoxylin and eosin. Magnification 10×40.

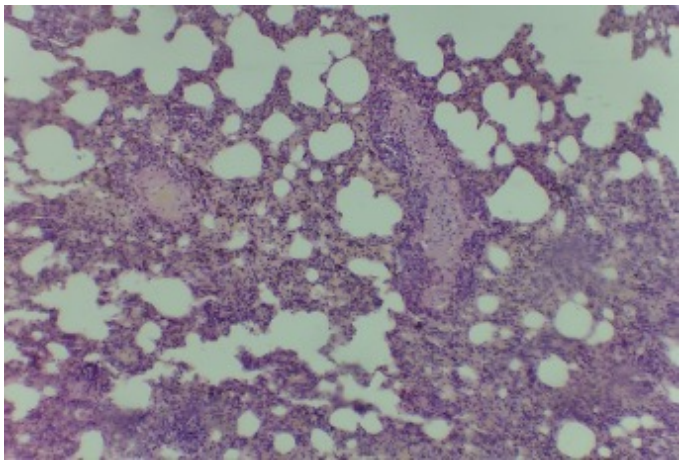

Fig. 4. Increased airiness (emphysematous changes) of alveoli with focal septal ruptures; perivascular sclerosis; thickening of interalveolar septa. Staining: hematoxylin and eosin. Magnification 10×10

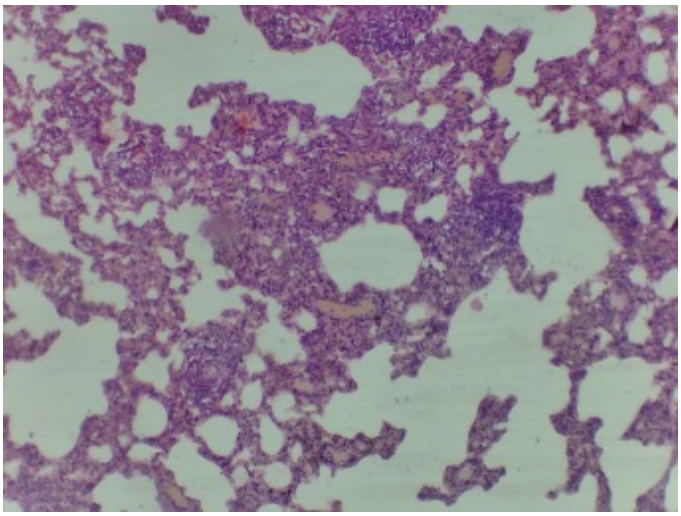

Fig. 5. Emphysematous alveoli with septal ruptures; inflammatory infiltration of the interstitium and perivascular regions. Staining: hematoxylin and eosin. Magnification 10×20.

Changes in the brain were associated with chronic hypoxia (Fig. 6).

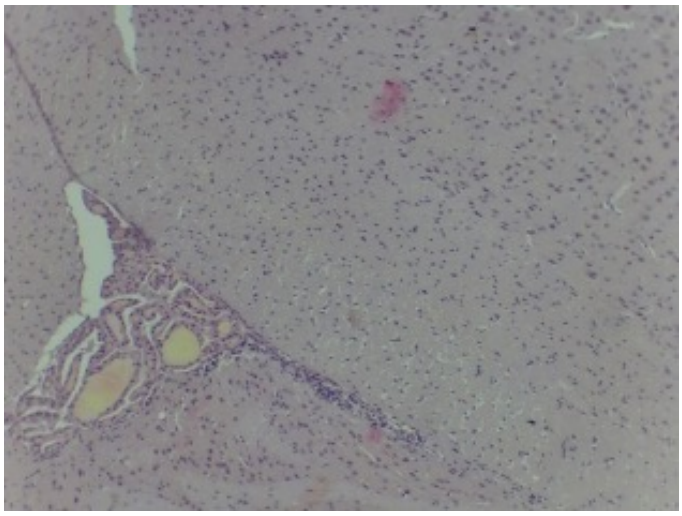

Fig. 6. Brain — pia mater with moderate edema and vascular congestion. Moderately expressed perivascular and pericellular edema. Staining: hematoxylin and eosin. Magnification 10×10.

In parenchymal organs — liver, kidneys, and heart — circulatory disturbances and dystrophic changes were identified (Figs. 7, 8, 9, 10).

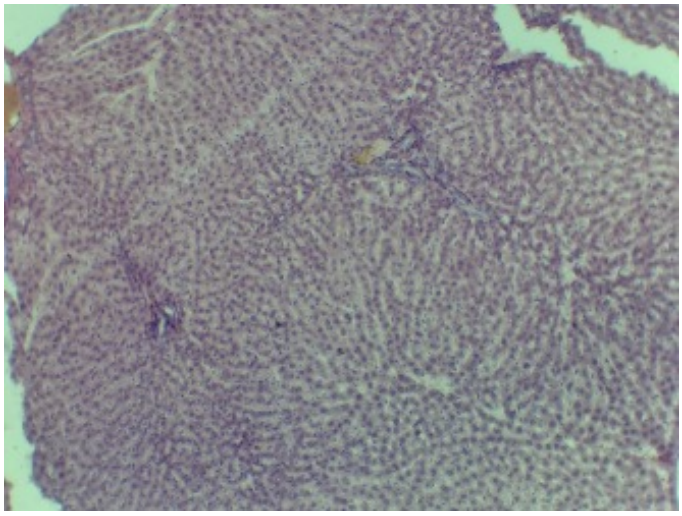

Fig. 7. Liver — thin capsule; lobular and trabecular structure of hepatocytes preserved; portal triads clearly expressed. Interstitial vessels unevenly congested. Hepatocytes with light nuclei and distinct nucleoli. Staining: hematoxylin and eosin. Magnification 10×10.

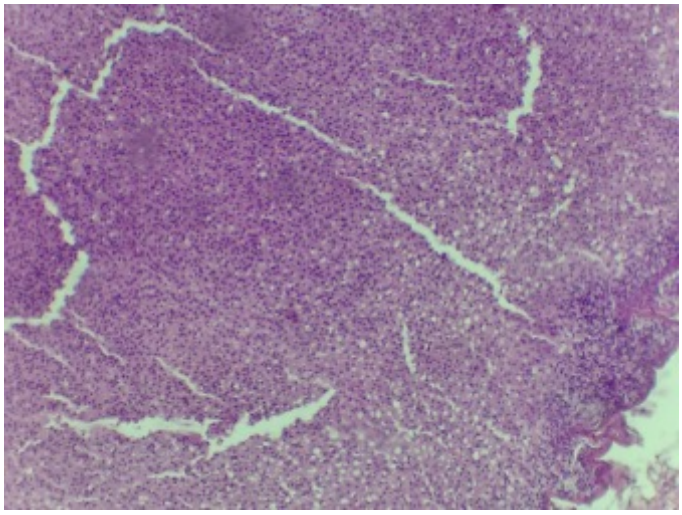

Fig. 8. Liver — vacuolar degeneration of hepatocytes at the periphery of lobules. Staining: hematoxylin and eosin. Magnification 10×10..

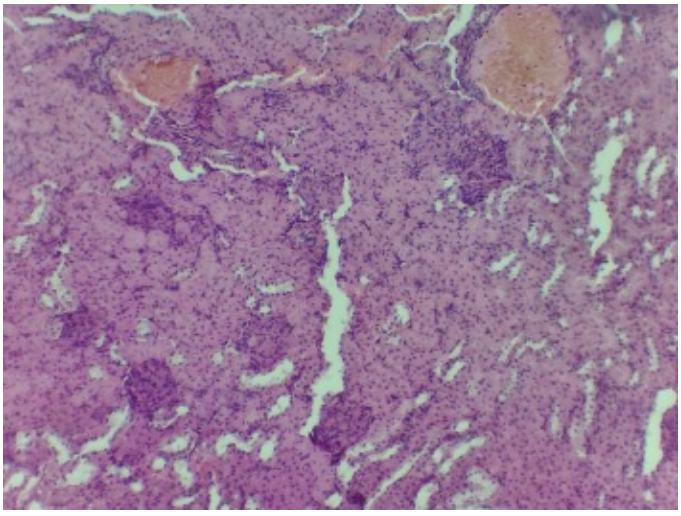

Fig. 9. Kidney — thin capsule; glomeruli of oval or irregular oval shape; Bowman's capsule thin, cavity free of pathological contents; glomerular capillary loops with uneven congestion. Epithelium of convoluted tubules swollen; straight tubules free.

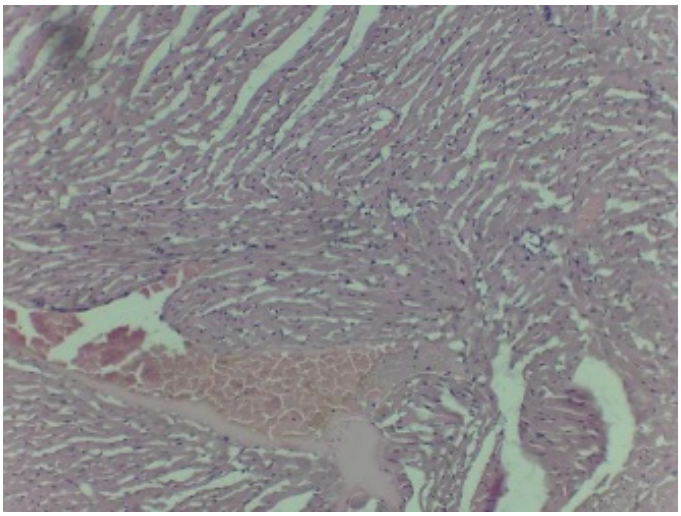

Fig. 10. Heart — marked vascular congestion in the stroma and intermuscular edema. Staining: hematoxylin and eosin. Magnification 10×10.

In the spleen, the capsule was thin; trabeculae poorly expressed; congestion of the red pulp was observed; lymphoid follicles with eccentrically located central arterioles, without germinal centers, but with distinct boundaries (Fig. 10).

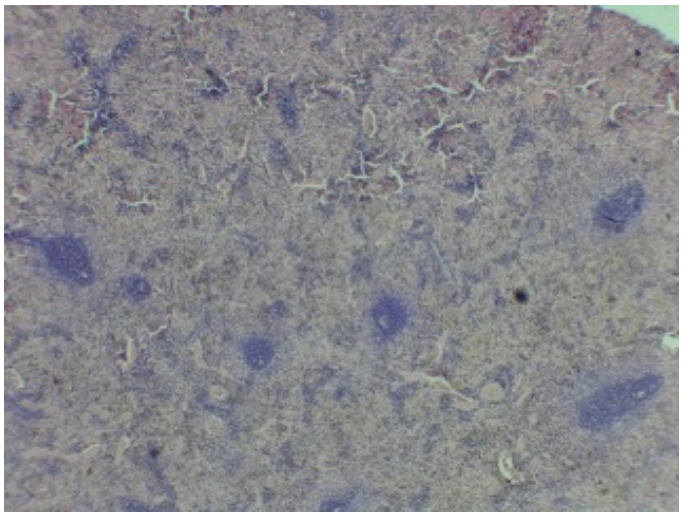

Fig. 11. Spleen. Staining: hematoxylin and eosin. Magnification 10×4.

CONCLUSION

The results of the present study convincingly demonstrate that chronic exposure to tobacco smoke in an experimental model leads to the development of interstitial lung injury, characterized by pronounced morphological changes in lung tissue, including hyperplasia of peribronchial lymphoid tissue, sclerosis, emphysematous remodeling of alveoli, and interstitial infiltration. Along with pulmonary pathology, systemic alterations were identified, including signs of chronic hypoxia in the brain, dystrophic and circulatory disturbances in the liver, kidneys, and heart, as well as morphofunctional changes in the spleen.

The revealed morphological abnormalities confirm the activation of the systemic inflammatory response and the involvement of multiple organs in the pathological process of interstitial pneumonia. This highlights the need for a comprehensive approach to the study of the pathogenesis of this disease.

The presented pathological model using the Kurlandsky chamber demonstrates high reproducibility and informativeness, making it a valuable tool for subsequent studies of the molecular mechanisms of organ damage, the evaluation of inflammatory biomarkers, and the testing of new therapeutic strategies for interstitial lung diseases.

REFERENCES

1. Chuchalin A.G. Pulmonology. National guidelines. Moscow: GEOTAR-Media; 2013.
2. Feldman C., Anderson R. Pneumonia as a systemic illness. Curr Opin Pulm Med. 2018;24(3):237–243.
3. Kayumov Yu.D., Tadzikhodjaeva Yu.Kh. Experimental model of chronic obstructive pulmonary disease with tobacco smoking in the study of membrane-destructive processes. Bulletin of the Association of Pulmonologists of Central Asia. 2020;18(1–4):22–26.
4. Hecker L., et al. NADPH oxidase-4 mediates myofibroblast activation. Nat Med. 2009;15(9):1077–1081.
5. Restrepo M., et al. Complication of community-acquired pneumonia. Semin Respir Crit Care Med. 2016;37(6):897–904.
6. Aliberti S., et al. Community-acquired pneumonia. Lancet. 2021;398(10303):906–919.

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Eksperimental interstitsial o‘pka shikastlanishida ichki a’zolarning morfologik o‘zgarishlari

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Toshkent davlat tibbiyot universiteti

Annotatsiya

Mazkur maqolada tamaki tutuni bilan uzluksiz ta’sir qilish orqali yuzaga keltirilgan eksperimental interstitsial pnevmoniya holatida kalamushlarning ichki a’zolaridagi patomorfologik o‘zgarishlar tahlil qilingan. Tadqiqot davomida o‘pkada yallig‘lanish va destruksiyalashuv jarayonlari — peribronxial skleroz, limfoid infiltratsiya va emfizematoz o‘zgarishlar aniqlangan. Bundan tashqari, jigar, buyrak, yurak va miya to‘qimalarida distrofik o‘zgarishlar qayd etilib, bu holat tizimli yallig‘lanish javobi va ko‘p a’zoli disfunktsiyani ko‘rsatadi. Olingan natijalar surunkali yallig‘lanishning patogenetik ahamiyatini ko‘rsatib, keyingi terapevtik tadqiqotlar uchun asos bo‘la oladi.

Kalit so‘zlar: o‘pkaninig interstitsial jarohatlanishi, tamaki tutuni, patomorfologiya, yallig‘lanish, ichki a’zolar, kalamushlar.

Морфологические изменения внутренних органов при экспериментальном интерстициальном поражении лёгких

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Аннотация

В данной работе представлены результаты патоморфологического исследования внутренних органов крыс при экспериментальной интерстициальном поражении легких, вызванной хроническим воздействием табачного дыма. Установлены выраженные воспалительно-деструктивные изменения в лёгочной ткани, включая перибронхиальный склероз, лимфоидную инфильтрацию и эмфизему. Наряду с лёгочной патологией выявлены дистрофические изменения в печени, почках, сердце и головном мозге, что указывает на развитие системного воспалительного ответа и полиорганной дисфункции. Полученные данные подчёркивают патогенетическую значимость хронического воспаления и могут служить основой для дальнейших исследований и оценки эффективности терапии.

Ключевые слова: интерстициальное поражение легких, табачный дым, патоморфология, воспаление, внутренние органы, крысы.