UDC: 616.3-001.17:616.24-091.8

MORPHOLOGICAL AND MORPHOMETRIC CHANGES IN THE LUNG TISSUE OF 1-, 3-, AND 6-MONTH-OLD OUTBRED WHITE RATS FOLLOWING A 70% ACETIC ACID–INDUCED CHEMICAL BURN OF THE DIGESTIVE TRACT

Ismoilov Zafar Zaripovich
Bukhara State Medical Institute named after Abu Ali ibn Sino, Bukhara, Uzbekistan
1 A. Navoi Street, Bukhara, Uzbekistan
Tel.: +998 (65) 223-00-50
E-mail: info@bsmi.uz

Abstract. The study evaluated the morphological and morphometric changes in the lung tissue of 1-, 3-, and 6-month-old outbred white rats following a chemical burn of the digestive tract induced by 70% acetic acid. Histological examination demonstrated severe destructive remodeling of the pulmonary parenchyma, including rupture and fusion of alveolar septa, formation of large emphysematous air spaces, areas of alveolar collapse, diffuse inflammatory infiltration, perivascular edema, and diapedetic microhemorrhages. The number of alveoli per microscopic field decreased by 31.4–33.7% in 1-month-old rats, 30.2–32.7% in 3-month-old rats, and approximately 40% in 6-month-old rats compared with age-matched controls. The thickness of the alveolar septa increased to 14.25±0.63, 15.38±0.71, and 16.91±0.83 μm, respectively. The area of peribronchial inflammatory infiltration progressively increased with age, reaching 458,800 μm² in 6-month-old rats. Van Gieson staining revealed collagen accumulation and early peribronchial fibrosis in the oldest animals. These findings demonstrate that a digestive tract burn induced by 70% acetic acid causes severe secondary pulmonary injury, the extent of which increases with age and progresses from acute necrotic-inflammatory damage to early fibrotic remodeling.

Keywords: acetic acid, chemical burn, digestive tract, lung tissue, alveoli, inflammation, fibrosis, morphometry, outbred white rats.

Aim of the Study The aim of the study was to determine the age-related morphological and morphometric changes in the lung tissue of 1-, 3-, and 6-month-old

outbred white rats following a chemical burn of the digestive tract induced by 70% acetic acid.

Materials and Methods

The experimental study involved outbred white rats divided into three age groups: 1-month-old rats weighing 48–65 g, with a mean body weight of 56.5±5.1 g; 3-month-old rats weighing 158–263 g, with a mean body weight of 210.5±8.0 g; and 6-month-old rats weighing 280–340 g, with a mean body weight of 312.4±9.3 g. Age-matched intact animals served as controls.

A chemical burn of the digestive tract was experimentally induced using a 70% acetic acid solution. At the predetermined experimental endpoint, lung tissue samples were collected for histological and morphometric examinations. Histological sections were stained with hematoxylin and eosin to evaluate the general architecture of the pulmonary parenchyma and with Van Gieson stain to identify collagen fibers and fibrotic changes.

The number of alveoli in the peripheral and central regions of the lung was counted per microscopic field. The thickness of the interalveolar septa and the area of peribronchial inflammatory infiltration were measured. The condition of the bronchiolar walls, pulmonary vessels, alveolar spaces, and interstitial tissue was also assessed. The results were expressed as mean values with statistical variation, and differences at $p<0.05$ were considered statistically significant.

Results

Histological examination following exposure to 70% acetic acid demonstrated severe structural damage to the lung tissue in all age groups. The pulmonary parenchyma lost its normal uniform architecture. Some alveoli became enlarged and merged into large emphysematous air spaces because of multiple ruptures of the interalveolar septa, whereas other areas of the lung were characterized by marked alveolar collapse and atelectasis.

In the 1-month-old rats, extensive areas of the pulmonary parenchyma showed destructive alterations. The alveolar walls demonstrated signs of necrotic damage and intense eosinophilic staining. The interalveolar septa were markedly thickened and infiltrated by inflammatory cells. Erythrocyte extravasation from the pulmonary capillaries, diapedetic hemorrhages, and focal exudative changes were also observed.

The number of alveoli in the peripheral regions of the lung decreased to 4.62±0.68 per microscopic field compared with 6.74±0.79 in the control group, representing a reduction of 31.4%. In the central regions, the number of alveoli

decreased from the control value of 7.67±0.70 to 5.08±0.55, corresponding to a reduction of 33.7%. The thickness of the interalveolar septa increased from 9.75±0.49 μm in the control group to 14.25±0.63 μm, representing an increase of approximately 46%. The area of peribronchial inflammatory infiltration reached 157,600 μm².

In the 3-month-old rats, the pathological process became more pronounced. The walls of the terminal bronchioles were markedly thickened, while diffuse inflammatory infiltration persisted throughout the lung parenchyma. Large emphysematous alveolar spaces, rupture and fusion of adjacent alveoli, edema of the periarterial tissue, and multiple diapedetic microhemorrhages were observed. The bronchial and bronchiolar walls were surrounded by extensive inflammatory infiltrates.

The number of alveoli in the peripheral regions was 4.91±0.47 compared with 7.03±0.80 in the age-matched control group, representing a reduction of 30.2%. In the central regions, the number decreased from 7.97±0.80 to 5.36±0.62, corresponding to a reduction of 32.7%. The thickness of the interalveolar septa reached 15.38±0.71 μm compared with 10.03±0.79 μm in the control group, representing an increase of approximately 53% ($p<0.05$). The peribronchial infiltration area increased to 212,400 μm².

The most severe morphological changes were detected in the 6-month-old rats. The pulmonary parenchyma contained multiple necrotic foci, extensive inflammatory infiltrates, large emphysematous air spaces, and areas of alveolar collapse. Numerous ruptured interalveolar septa caused the fusion of adjacent alveoli. Periarterial edema and diapedetic hemorrhages were widespread. The lumina of the bronchi contained eosinophilic mucus, while their walls were markedly thickened.

The number of alveoli in the peripheral regions decreased to 5.02±0.61 compared with 8.35±0.98 in the control group. In the central regions, it decreased to 5.44±0.59 compared with the control value of 9.03±0.69. Thus, the reduction in the number of alveoli in both regions was approximately 40%.

The thickness of the interalveolar septa reached its maximum value of 16.91±0.83 μm compared with 11.24±0.57 μm in age-matched controls, representing an increase of approximately 50% ($p<0.01$). The area of peribronchial inflammatory infiltration increased to 458,800 μm², which was approximately 2.9 times greater than that observed in 1-month-old rats.

Van Gieson staining revealed pronounced collagen accumulation around the bronchi and within some interalveolar septa in the 6-month-old animals. Marked collagenization of the bronchial wall, peribronchial fibrotic changes, and persistent

inflammatory infiltration indicated the transition of acute necrotic-inflammatory injury to early fibrotic remodeling.

Discussion

A chemical burn of the digestive tract induced by 70% acetic acid caused not only local corrosive injury but also severe secondary damage to the lung tissue. The observed pulmonary alterations may be associated with a systemic inflammatory response, microcirculatory disturbances, increased vascular permeability, and the entry of inflammatory mediators and toxic products of tissue destruction into the circulation. The reduction in the number of alveoli per microscopic field reflected rupture and fusion of alveolar walls, formation of enlarged emphysematous spaces, and collapse of other alveolar regions. The progressive thickening of the interalveolar septa was associated with edema, inflammatory infiltration, vascular congestion, and subsequent collagen accumulation.

Although all age groups demonstrated severe pulmonary damage, the pathological process was most pronounced in 6-month-old rats. These animals showed the greatest relative reduction in alveolar number, the maximum thickness of the interalveolar septa, the largest area of inflammatory infiltration, and the appearance of early fibrotic changes. Therefore, the severity of secondary pulmonary injury following a 70% acetic acid–induced digestive tract burn demonstrated a clear age-dependent pattern.

Conclusion

A chemical burn of the digestive tract induced by 70% acetic acid caused severe destructive, inflammatory, vascular, and early fibrotic changes in the lung tissue of outbred white rats.

In 1-month-old rats, the pathological process was characterized by early necrotic damage to the alveolar walls, septal thickening, erythrocyte extravasation, and diffuse inflammatory infiltration. The number of alveoli decreased by 31–34%, while septal thickness increased by approximately 46%.

In 3-month-old rats, diffuse infiltration, bronchiolar wall thickening, emphysematous enlargement of the alveolar spaces, perivascular edema, and microhemorrhages became more pronounced. The number of alveoli decreased by 30–33%, and septal thickness increased by approximately 53%.

The most severe alterations were observed in 6-month-old rats. The number of alveoli decreased by approximately 40%, septal thickness increased to 16.91±0.83 μm, and the peribronchial infiltration area reached 458,800 μm². The development of

collagenization and peribronchial fibrosis indicated progression from acute necrotic-inflammatory injury to early fibrotic remodeling.

Thus, the severity of lung tissue injury caused by a digestive tract burn with 70% acetic acid increased with the age of the animals. The identified morphometric parameters may be used as objective criteria for assessing the degree of secondary pulmonary damage following severe chemical burns of the digestive tract.

References

1. Contini S., Scarpignato C. Caustic injury of the upper gastrointestinal tract: a comprehensive review // *World Journal of Gastroenterology*. – 2013. – Vol. 19, No. 25. – P. 3918–3930. DOI: 10.3748/wjg.v19.i25.3918.
2. Cheng H.T., Cheng C.L., Lin C.H. et al. Caustic ingestion in adults: the role of endoscopic classification in predicting outcome // *BMC Gastroenterology*. – 2008. – Vol. 8. – Article 31. DOI: 10.1186/1471-230X-8-31.
3. Bird J.H., Kumar S., Paul C., Ramsden J.D. Controversies in the management of caustic ingestion injury: an evidence-based review // *Clinical Otolaryngology*. – 2017. – Vol. 42, No. 3. – P. 701–708. DOI: 10.1111/coa.12819.