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EPITHELIUM ULTRASTRUCTURE OF THE BRONCHI OF EXPERIMENTAL ANIMALS IN ACUTE LUNG INJURY

Abstract*: In acute lung injury (ALI), some changes that affect both the ciliary epithelium apparatus and the structure of intracellular organelles of ciliated cells and Clara cells occur in the ultrastructural organization of the bronchiolar epithelium. Thedystrophicanddestructive changes found in electron microscopy appear to be the result of an intervention with an inflammatory disease affecting not only the large bronchi butalsothebronchioles.*

Key words*: alveolar-capillary membrane, acute lung injury, endotoxemia, electron microscopic studies, ciliated cells*

Language*: English*

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Introduction

Acute lung injury (ALI) is an acute inflammation disorder that disrupts the endothelial and epithelial barriers of the lungs [2,p.24;4,p.441;5,p.1075]. The alveolar-capillary membrane consists of microvascular endothelium, interstitium, and alveolar epithelium[1,p.74]. Cellular characteristics of ALI include loss of alveolar-capillary membrane integrity, excessive transepithelial migration of neutrophils, and release of pro-inflammatory cytotoxic mediators [3,p.229; 7,p.207; 8,p.313]. Biomarkers found on epithelium and endothelium that are involved in inflammatory and coagulation cascades predict morbidity and mortality in ALI[6,p.244].

The research aimed to determine the morphofunctional reorganization of the epithelium in acute lung injury.

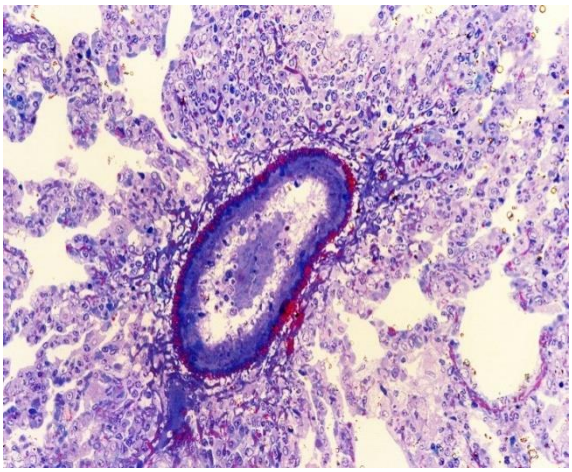
Materials and Methods

The material for the study was the distal airways (bronchioles) with a diameter of less than 2 mm, isolated from the lungs of 10 white rats, in which ALI was modeled in acute endotoxemia [1]. The animals were decapitatedunder thiopental anesthesia with calypsol. The material for electron microscopy was fixed by immersion in a mixture of 2.5% glutaraldehyde, 2.5% paraformaldehyde, and 0.1% picric acid solutions in phosphate buffer (pH = 7.4) for 15 minutes. The material obtained from the lungs of 10 healthy white rats was used as a control. Further processing of the material of bronchioles from the lungs - dehydration and filling in Araldite and Epon-812 was performed following the generally accepted method [8]. Semi- and ultrathin sections were obtained using a Leica EM UC7 ultramicrotome. Semi-thin sections (1–2 μm) were stained using trichrome staining (methylene blue-azure II- fuchsin stain) according to F. D'Amico (2005) and examined under a Zeiss light microscope (PromoStar) [7]. The images were photographed with a Canon digital camera (Japan). Ultrathin sections (50-70 nm) were stained with a 2% saturated aqueous solution of uranyl acetate, then with a 0.4% solution of pure lead citrate (Serva) in a 0.1 M NaOH solution. For viewing and photographing stained and unstained ultrathin sections, an electron microscope JEM-1400 (Japan) at an accelerating voltage of 80-120 kV was used.

Results and discussion

Total results of light-optical and electron microscopic studies on the bronchial mucosa showed that an inflammatory process with a predominance of degenerative - exudative changes had already developed in the early stages. A plethora of blood

vessels, stasis, cell clusters were observed with an admixture of more or fewer amounts of cells (macrophages, lymphocytes, leukocytes), fibrin filaments, and diapedesis. An increase in vascular permeability also occurred (Fig. 1)



Fugure.1. Predominance of degenerative - exudative changes in the bronchial mucosa
Staining: hematoxylin-eosin
MAG: 280x.

Significant alterative changes were manifested by the destruction of epitheliocytes, the accumulation of a large amount of mucus, and neutrophilic leukocytes (Figure 2).

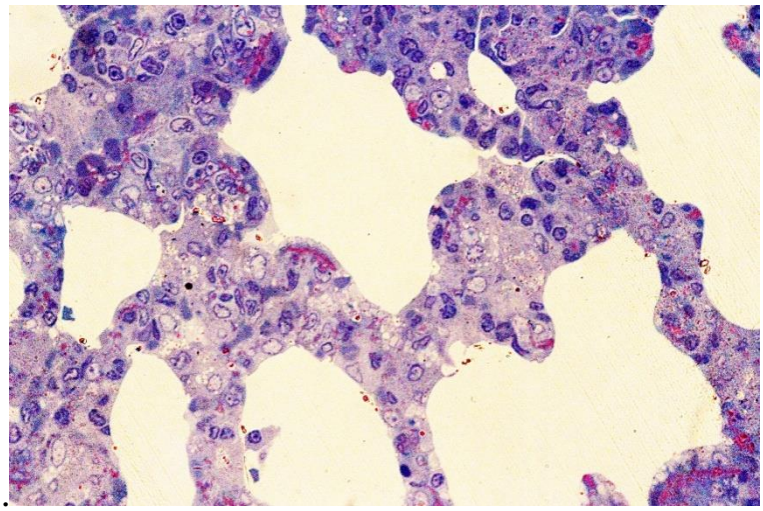


Figure 2. Significant alterative changes in the bronchial mucosa
Staining: hematoxylin- eosin
MAG: 280x.

Ultrastructural analysis of ciliated cells revealed destruction of some of the cilia. On the apical surface of ciliated cells, cytoplasmic outgrowths, resembling microvilli, 0.5 - 0.6 μm in length, appeared, at the base of which plasmalemma invaginations were determined (Figure 3).

Cells with well-developed tubules of the rough endoplasmic reticulum, which were located mainly in the supranuclear area of the cells, dominated in the ciliated cells.

Cells with fragmentation and vacuolization of the tubules of the rough endoplasmic reticulum were also identified among the ciliated cells. The Golgi complex was well developed in these cells. The cisterns of the Golgi complex were expanded and contained substrates of medium electron density. In the immediate vicinity of the Golgi complex, lysosomes with a diameter of 0.5-0.7 μm occurred, which fused with mitochondria. Clusters of numerous ribosomes and polysomeswere also found there (Figure 4).

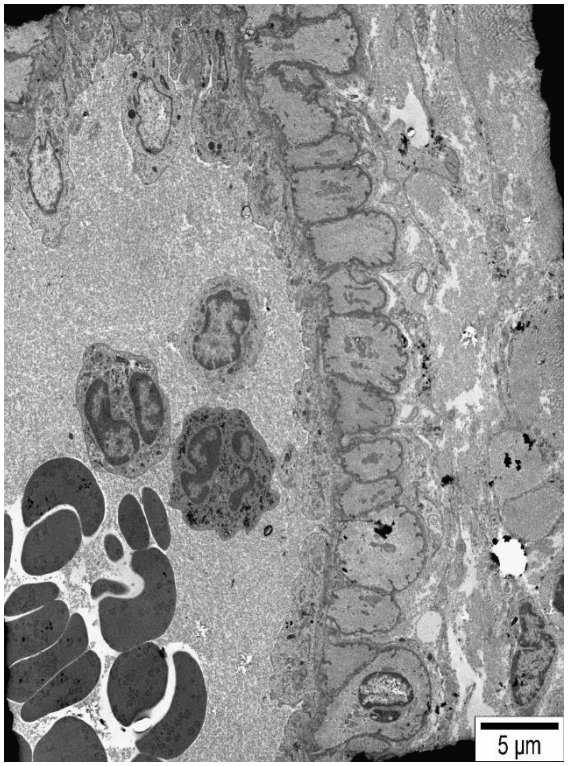


Figure 3. Electronogram. Destruction of some cilia, plasmalemma invaginations
MAG: 5000x

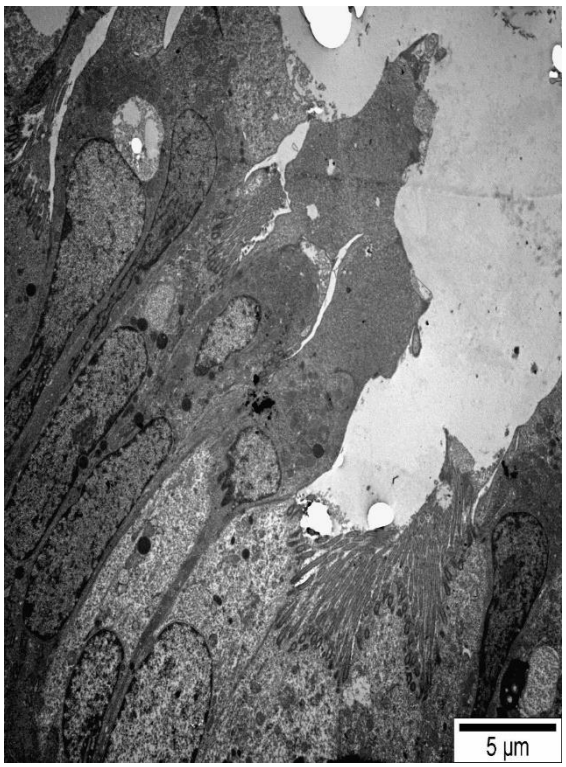


Figure 4. Electronogram. Cells with well-developed tubules of the rough endoplasmic reticulum dominated in the ciliated cells.
MAG: 5000x

The basal cells of the integumentary epithelium of the bronchial mucous membrane had a large nucleus with pronounced nucleoli; elements of the endoplasmic reticulum were few. Free ribosomes and tonofibrils were oriented along the basal membrane. Mitochondria were small and had a one-electron density matrix.

The structure of the basal membrane of the integumentary epithelium, as a rule, changed. Besides, unchanged basal membranes were found at the sites of translocation of lymphoid elements from the lamina propria of the mucous membrane.

Changes in the epithelium of the trachea and bronchi were characterized by pronounced hyperplasia of the goblet cells. Expanded cisterns of the cytoplasmic reticulum, densely covered with ribosomes, were recorded in them. The endoplasmic reticulum was surrounded by mitochondria. It should be noted that the extrusion of granules of mucoid secretion was realized by the mechanism of holocrine secretion, i.e., a part of the cytoplasm of the glandulocyte was rejected with the involvement of fragments of mitochondria and ribosomes in this process.

Under these experimental conditions, swelling of the endothelial cells of the vessels of the microcirculatory bed, leading to a narrowing of the lumen of the hemocapillaries, was noted. In this case, there were contacts between the plasmalemma of endothelial cells and formed elements of the blood with a simultaneous increase in the number of pinocytic vesicles concentrated in the apical part of the cytoplasm (Figure 5).

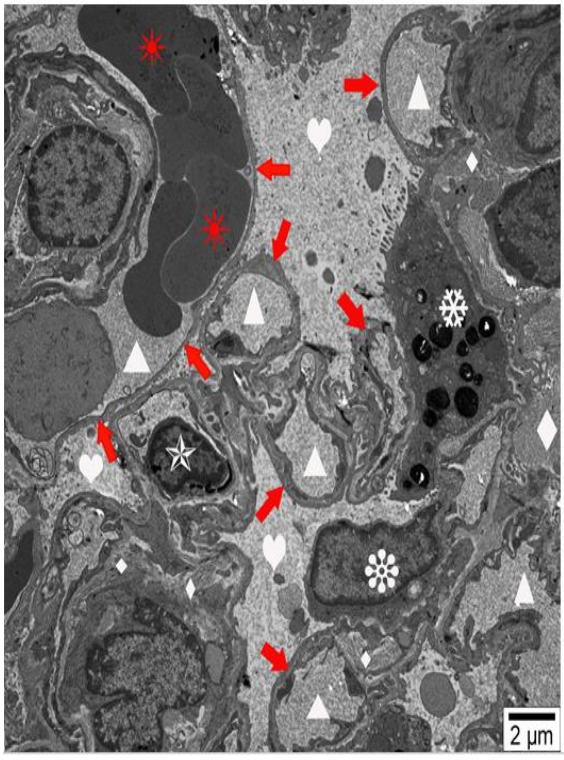


Figure 5.Electronogram. Swelling of vascular endothelial cells of the microcirculatory bed, leading to a narrowing of the lumen of the hemocapillaries. Contacts between plasmalemma of endothelial cells and formed elements
MAG: 12500x

Thus, the phenomena of catarrhal-sclerotic bronchitis developed in the walls of the intrapulmonary bronchi.In the background of local destructive processes, foci of granulation tissue with an abundance of newly formed microcirculation vessels (arterioles, capillaries) developed in the connective tissue of the lamina propria of the mucous membrane.The described changes in the walls of the bronchi were usually mosaic in nature. Their intensity increased as the diameter of the bronchi decreased, reaching a maximum in the bronchioles. Under these conditions, we also detected various morphological variants of obstructive bronchitis (catarrhal, catarrhal-sclerotic) as well as morphological signs of the inflammatory process chronization. Plasmorrhage, hemorrhages developed in the wall of the bronchi, the number of lymphocytes, plasma cells, macrophages, granulocytes, and mast cells increased significantly. Inflammatory changes were localized mainly around the end sections and excretory ducts of the bronchial glands. Very large infiltrates contained lymphocytes, as well as single eosinophils, polymorphonuclear

leukocytes, and macrophages. With the addition of purulent inflammation in the infiltrates, polymorphonuclear leukocytes predominated. The wall of the bronchioles in some cases underwent purulent fusion. As the process progressed, a significant amount of mucous and/or purulent content accumulated in the lumen of the bronchi, which aggravated the processes of bronchial obstruction.

In the long term, morphological signs of the development of severe forms of bronchitis were recorded, with a tendency to their chronization. Productive inflammation, especially in the small bronchi, ultimately leads to the transformation of the epithelium and obliteration of the bronchi with purulent masses, which in total, can result in the development of irreversible obstruction of the airways.

Conclusion

Thus, we have established facts indicating the formation of pronounced morphofunctional changes, reorganization of the epithelium of the airways leading to a tendency towards chronization of the inflammatory process in acute lung injury, in experimental animals under these experimental conditions.

References:

1. Bernard, G.R., Artigas, A., Brigham, K.L., Carlet, J.. Falke, K., Hudson, L., Lamy, M., LeGall, J.R., Morris, A., & Spragg, R. (1994). Report of the American-European Consensus conference on acute respiratory distress syndrome: definitions, mechanisms, relevant outcomes, and clinical trial coordination. Consensus Committee. *J Crit Care*., 9: 72–81.
2. Bhattacharya, J., & Westphalen, K. (2016). Macrophage-epithelial interactions in pulmonary alveoli. *SeminImmunopathol.* 2016, 23-26.
3. Puig, F., Herrero, R., Guillamat-Prats, R., Gómez, M.N., Tijero, J., Chimenti, L., Stelmakh, O., Blanch, L., Serrano-Mollar, A., Matthay, M.A., & Artigas, A. (2016). A new experimental model of acid- and endotoxin-induced acute lung injury in rats. *Am J Physiol Lung Cell Mol Physiol.* 2016 Aug 1, 311 (2): L229-37.
4. Esteban, A., Fernandez-Segoviano, P., Frutos-Vivar, F., Aramburu, J.A., Najera, L., Ferguson, N.D., Alia, I., Gordo, F., & Rios, F. (2004). Comparison of clinical criteria for the acute respiratory distress syndrome with autopsy findings. *Ann Intern Med*. 141:440–445.
5. Ferguson, N.D., Meade, M.O., Hallett, D.C., & Stewart, T.E. (2002). High values of the pulmonary artery wedge pressure in patients with acute lung injury and acute respiratory distress syndrome. *Intensive Care Med*., 28: 1073–1077.
6. Johnson, E.R., Matthay, M.A. (2010). Acute lung injury: epidemiology, pathogenesis, and treatment. *J Aerosol Med Pulm Drug Deliv.,* 2010 Aug, 23(4): 243-52.
7. Voelkel, N.F., Czartolomna, J., Simpson, J., & Murphy, R.C. (1992). *FMLP causes eicosanoid-dependent vasoconstriction and edema in lungs from endotoxin-primed rats*. Am Rev Respir Dis.
8. Comhair, S.A., Xu, W., Mavrakis, L., Aldred, M.A., Asosingh, K., & Erzurum, S.C. (2012). Human primary lung endothelial cells in culture. *Am J Respir Cell Mol Biol.* 2012 Jun, 46(6): 723-30.
9. Yang, Y., Li, Q., Deng, Z., Zhang, Z., Xu, J., Qian, G., & Wang, G. (2011). Protection from lipopolysaccharide-induced pulmonary microvascular endothelial cell injury by activation of hedgehog signaling pathway. *MolBiol Rep.* 2011 Aug, 38(6): 3615-22.
10. Wind, J., Versteegt, J., Twisk, J., van der Werf, T.S., Bindels, A.J., Spijkstra, J.J., Girbes, A.R., & Groeneveld, A.B. (2007). Epidemiology of acute lung injury and acute respiratory distress syndrome in The Netherlands: a survey. *Respir Med*. 2007 Oct, 101(10):2091-8.