MORPHOLOGICAL ASSESSMENT OF LUNG DAMAGE IN YOUNG RATS CAUSED BY PARTIAL TRACHEAL OCCLUSION

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Abstract

A decrease in the lumen of the upper respiratory tract (the trachea) in newborn infants is the main cause of impaired development of lungs during postnatal ontogenesis.

Aim. To study the histological changes in the lung tissue of young sexually immature animals when modeling partial tracheal stenosis.

Materials and methods. Young male Wistar rats aged 25-28 days and weighing 42-54 g were divided into 4 groups: 1) control (intact); 2) animals with tracheal stenosis (7 days); 3) animals with tracheal stenosis (21 days); 4) animals with tracheal stenosis (7 days after surgery followed by observation for 2 weeks). Modeling of stenosis was reproduced by applying a ligature at the level of the upper quarter of the trachea. This caused a narrowing of the tracheal lumen by 25-30%. The histological changes of the lungs in animals of all experimental groups were studied.

Results. A significant structural damage to the alveoli and swelling of the alveolar acini were revealed on the 7th day of the development of tracheal stenosis. A partial reduction of pathomorphological changes was observed on day 21. The removal of the ligature from the trachea was accompanied by a gradual restoration of the normal histological structure of the animal lung tissue, while maintaining individual zones of hypoxic damage. At the same time, intra-alveolar edema, wedening of the alveolar lumen, a decrease in the thickness of their walls, structural disorders of the alveolar-capillary membrane, the accumulation of hemorrhagic contents, and destructive changes in the cells of the pulmonary acini were observed in the lung tissue. Long-term hypoxia (21 days) was accompanied by a partial regression of these damages with a simultaneous increase in the number of macrophages and massive death of pulmonary acini cells. In group 4 animals, after removing the ligature, in addition to the trachea damage the preservation of separate dystrophic changes was observed.

Conclusions. In tracheal stenosis model and acute hypoxic-respiratory hypoxia, dystrophic changes in the lung tissue were revealed. With an increase in the duration of hypoxic exposure, a gradual adaptation of the cellular elements and metabolic processes to the conditions of impaired oxygenation was observed. Restoration of lung ventilation accelerates the development of compensatory and regenerative processes in lung cells against the background of the preservation of pronounced structural and morphological disorders.

Keywords: hypoxia, lungs, tracheal stenosis, histological changes, acini.

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