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Morphofunctional State Of The Wall Of Bronchi And Lung Tissue Structures Of Different Caliber In Experimental Bronchoectasis

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ABSTRACT

Objective: *This study aims to investigate the structural and functional changes in the bronchial wall and lung tissue induced by experimental bronchiectasis in a rabbit model.* ***Method:*** *A total of 34 adult rabbits weighing 2-2.5 kg were subjected to bronchiectasis induction using a modified method involving the insertion of a sterile Capron thread into the tracheal cavity. The rabbits were examined at intervals of 3 to 6 months post-induction, with histological and morphometric analyses conducted on their respiratory tissues.* ***Results:*** *Significant findings included the presence of purulent-mucous exudate, thickening of the bronchial mucosa, and inflammatory infiltrates comprising lymphocytes, neutrophils, and histiocytes. The thickness of the bronchial walls and changes in lung tissue, including emphysema and atelectasis, were also observed, indicating progressive disease over the study period. Morphometric analysis showed a marked increase in the number of cells in the bronchi and lung tissues at 3, 4, and 5 months.* ***Novelty:*** *This study contributes new insights into the pathological progression of bronchiectasis in an animal model, demonstrating the dynamic changes in bronchial structure and inflammatory responses over time, which may have implications for understanding the disease in human populations.*

INTRODUCTION

Bronchoectasis is an acquired chronic disease of the bronchopulmonary system, characterized by infiltrative and sclerotic changes in the peribronchial cavity and purulent inflammatory processes in the dilated, deformed bronchi [1].The incidence rate of children aged 0 to 14 living in developed countries is considered to be low-e.g. 0.5 per 100,000 children in Finland and 3.7 per 100,000 children in New Zealand. However, the incidence rate in Aboriginal children in central Australia reaches 200 per 100,000 children [1], [2]. Due to the unsatisfactory level of medical care for children under 4 years of age after pneumonia in India, bronchiectasis is diagnosed in 212-2646 cases per 1million people [3], [4]. Studies have not been carried out in the Russian Federation to study the prevalence of bronchoectatic diseases in children. There are statistics on the prevalence of nosological forms corresponding to the codes XKT-10 J44 (another chronic obstructive pulmonary disease) and J47 (bronchoectasis) in children between the ages of 0 and 14: 98.3 per 100,000 in 2010 and 81.3 in 2011 [5].With repeated lung infections, it can be assumed that chronic inflammation of the respiratory tract is the main cause of bronchoectasis.

Bronchiectasis is a multi-factoringpathology, and now in its pathogenesis involves complex interactions between the organism and respiratory pathogeneswhich are the

factors of the environment. This interaction contributes to the deplorable folk of recurrent infections, tissue of the respiratory tract, impaired clearance, the destruction of structural elements of the wall of the bronchi and the formation of enlargement and obstruction of the small bronchi [1].Under the conditions of experimental pneumonia, epithelial and immune structures of the immune system, as well as regulatory structures are characterized by a synchronous increase in their morphofunctional activity [3], [6].

RESEARCH METHOD

In rabbits, the results of the study of the structural components of the bronchi wall of various calibers, immune inflammatory cells, as well as the observed changes in the parenchyma of lung tissue are presented, causing bronchiectasis in experimental conditions. The studies were carried out in 34 rabbits, adult, weighing 2-2.5 kg. In rabbits in the experiment, an examination was carried out after 3-6 months after a certain period of time after the cause of bronchiectasis. Material from 8-9 rabbits was analyzed in each period.

To cause experimental bronchoectasis, M.I.Zakharevsky and N.I.Created by Anichkov and L.V.Yatshenko and N.T.A modified method was used from the sides of Reichlin (1981). This method allowed the introduction of a sterile Capron thread into the tracheal cavity to trigger experimental bronchoectasis in rabbits after a certain period of time.

In the planned deadlines, experimental rabbits were lifeless in such a way as to comply with the rules of Bioethics, and their respiratory organs were taken for examination. According to the manual for the unification of histological and histochemical investigations, operational materials were processed, in which 10% neutral formalin was fixed at no less than 24 hours, and the lumps were dehydrated in alcohol with increased concentration. After that, the material was put on paraffin. In the next step, 5-6 Nm paraffin cuts were obtained from prepared paraffin blocks. The incisions were dyed on hematoxylin and eosin by all-histological methods, picrofoxin by Van-Gison, and resortsin-fuchsin by Weigert.

RESULTS AND DISCUSSION

In 3.4 months after the introduction of the bronchoectasis disease model, experimental rabbits were found to be macroscopic reddish - grey in their bronchi of various calibers, slightly hardened. Symptoms of edema and hyperemia are observed on the mucous membranes of the trachea and bronchi. A purulent - mucous exudate is detected at the tip of the large and Lobar bronchi cavity and Capron thread, a serous-purulent exudate of the middle and small caliber bronchi cavity.

The lungs of rabbits are light pink - reddish, swollen and densely concentrated, and atelectasis, emphysema and abscess foci are detected in some lung segments [7].

In microscopic examinations, the process of desquamation of the ciliated epithelium and hypertrophy and hypersecretion in goblet cells on the mucous membrane of bronchi

of various calibers were clearly manifested. Basal cell hyperplasia has also been diagnosed, and in some areas the nuclei have been found to be HyperChrome stained.

In the mucous membrane, cases of picrinophilia of collagen fibers, mucoid and fibrinoid staining of fibers and fuchsinophilia in muscle tissue were noted. In the mucosa and especially in the large and medium bronchi there are inflammatory infiltrates consisting of lymphocytes, neutrophils, histiocytes, fibroblasts, plasmatic cells. Small-caliber bronchi show signs of smooth muscle cell hypertrophy and fuchsinophilia of the muscle floor.

In the results of staining according to Weigert, it was noted that the unfavorable shape of the bronchial mucosa and the unequal distribution of the epithelium, thickening of the floor in some places. When staining with Van-gizone, foci of fibrosis covering picrinophilic and inorganic fuchsinophilic connective tissue fibers were identified, particularly evident in the small bronchi.

There were signs of Hemostasis and lymphostasis in small blood vessels in the mucous membrane, hypersecretion and enlargement of the corridors in serous and protein glands. In Months 3, 4 and 5 of the experiment, pulmonary microscopy shows destructive and inflammatory processes prominent in many alveoli, especially in the inter-alveolar barrier. The alveolar cavity was enlarged and interstitial barriers thinned, inflammation resulted in emphysematosis and the formation of airless foci, which were found to be located in more peribronchial tissues (Figure.1, 2, 3).

During these periods, the thickness of the mucous membrane of the large and Lobar bronchi in the morphometric analysis was 69.6 μm, which is 0.68 times more than the ratio to the control group. The mucous membrane of medium-caliber bronchi was found to be 0.64 times thick and 0.39 times that of small-caliber bronchi.

The thickness of the mucous membrane of the large and Lobar bronchi was 87.2 μm, which is 0.16 times more than in the control group. In the case of medium-caliber bronchi, this figure was 0.14 times, and in the case of small-caliber bronchi, 0.17 times. The fibrosis of the large and Lobar bronchi and the muscle floor are 263.7 μm thick, which is 0.48 times more than with the control group.

In medium-caliber bronchi, this figure was found to be 0.50 times that in small-caliber bronchi, 0.47 times. The adventisal membrane of the bronchial wall was also altered, with a thickness of 12.6 μm in the large bronchi, 0.73 times greater than the control group, 0.75 times in the medium-caliber bronchi, and 0.12 times in the small-caliber bronchi. Even in 5 months of the study, rabbits in the observation group were noted to grow reliably in the thickness of the bronchial mucosa. 3, 4, and 5 months after the experimental bronchiectasis, rabbits experienced an increase in the number of cells in bronchi and lung tissue of different calibers. In 3 months, LP increased by 11.39% in the large and Lobar bronchi, 14.43% in the medium-caliber bronchi and 13.39% in the small-caliber bronchi, and 10.31% in the respiratory part of the lung. MP grew by an average of 9-12%, while nm increased by 30.3% in the large and Lobar bronchi and 27.6% in the

middle bronchi. PP rates were also higher compared to the control group, and FP dropped significantly.

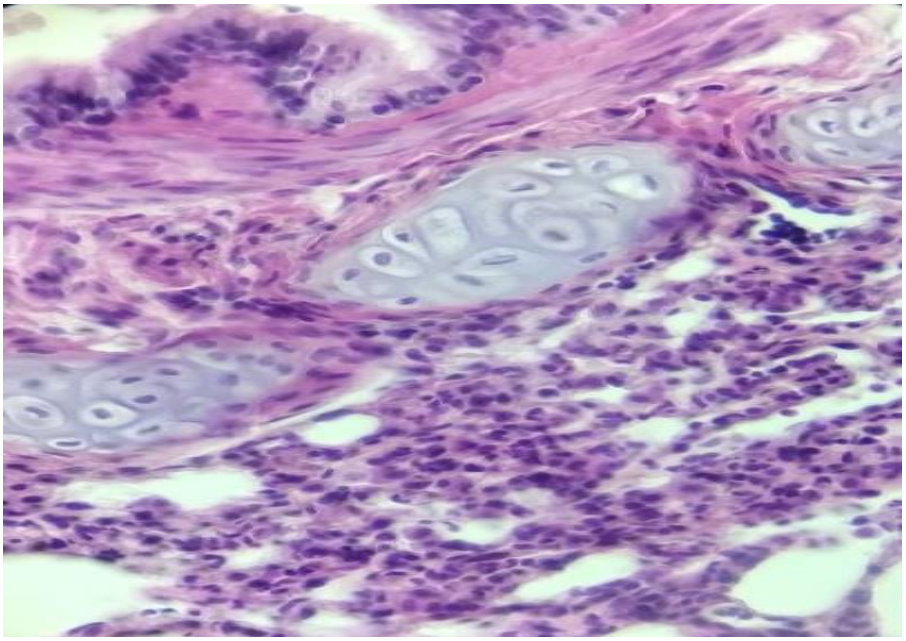

Figure 1. Rabbit's lungs.

In the figure 1 is experimental bronchiectatic disease. 3 months of observation. Plasmocytes are found in the cavity of respiratory bronchioles. Stained with hematoxylin and eosin. Magnification X400.

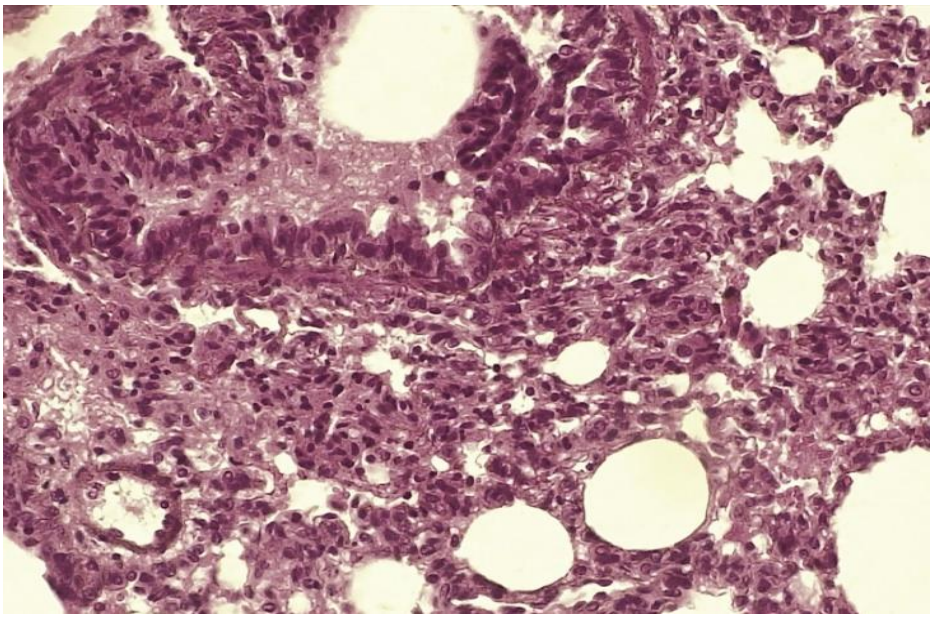

Figure 2. Rabbit's lungs.

In the figure 2 is experimental bronchiectatic disease. 3 months of observation. Plasmocytes are found in the cavity of respiratory bronchioles. Stained with hematoxylin and eosin. Magnification X400.

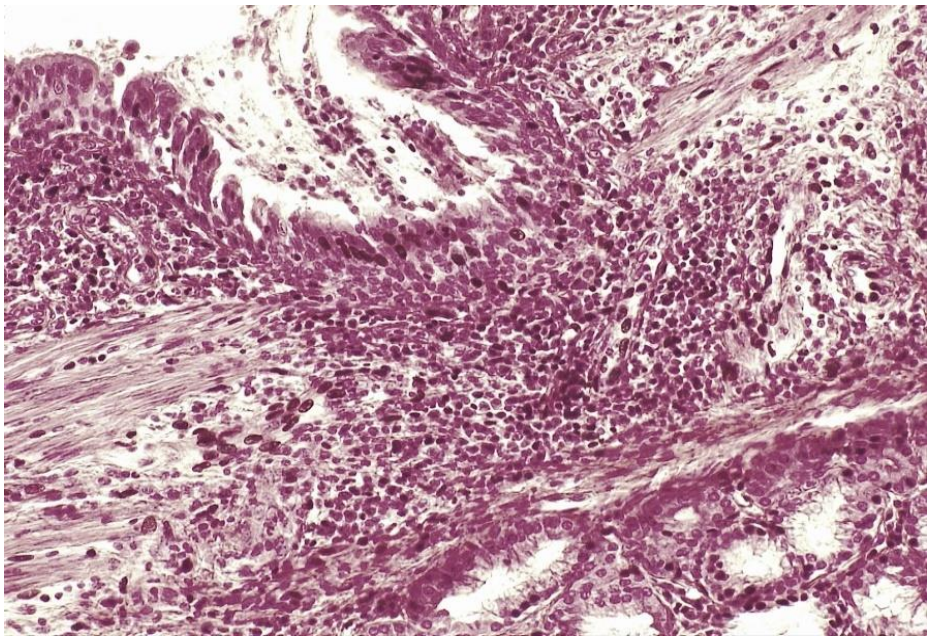

Figure 3. Rabbit's lungs.

In the figure 3 is experimental bronchiectatic disease. 5 months of observation. Plasmocytes are found in the cavity of respiratory bronchioles. Stained with hematoxylin and eosin. Magnification X400.

In 4 months, it was noted that LP rates increased by 14.13% in the large and Lobar bronchi and 12.67% in the pulmonary respiratory tract. MP, on the other hand, was found to increase by 10-20% and nm increased by 38.8% in large bronchi. In 5 months, LP increased by 15.28% in large bronchi, and mm by 13-25%. NP showed the highest growth rates-38.9%. In addition, it was observed that while PP increased by 15% compared to the control group, FP decreased.

Thus, 3.4.5 months after the experimental rabbit lung was called a model of bronchoectasis disease after intratracheal insertion of the Strand, there was an increase in alternative exudative processes in the respiratory organs of experimental rabbits.

CONCLUSION

Fundamental Finding : This study reveals that the experimental induction of bronchiectasis in rabbits leads to significant structural and functional changes in the bronchial wall and lung tissue over a period of 3 to 5 months. Notable findings include the presence of purulent-mucous exudate, thickening of the bronchial mucosa, and marked inflammatory infiltration in the respiratory tissues. **Implication :** The observed pathological changes highlight the importance of understanding the chronic inflammatory processes associated with bronchiectasis, which may inform future

therapeutic strategies and improve clinical management of affected individuals, particularly in populations at higher risk, such as children in developing regions. **Limitation :** The study is limited by its reliance on a single animal model (rabbits), which may not fully replicate the human condition of bronchiectasis. Additionally, the sample size and duration of the study may not capture the long-term progression of the disease. **Future Research :** Future studies should explore the molecular mechanisms underlying bronchiectasis and its progression in different animal models and human populations. Longitudinal studies examining the effectiveness of various treatment interventions and their impacts on inflammatory responses and lung function would also be beneficial.

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