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PATHOMORPHOLOGICAL AND MORPHOMETRIC FEATURES OF ALVEOLAR STRUCTURES IN PNEUMOPATHIES

Abdullayeva Nurjakhon Juraboy kizi

Andijan State Medical Institute, Uzbekistan

Abstract: The present study investigates the specific pathomorphological and morphometric alterations occurring in the alveolar structures of the lungs in various pneumopathies. Structural remodeling of the alveoli represents one of the most critical processes determining the clinical course and prognosis of pulmonary diseases. Lung tissue samples obtained from patients with inflammatory, infectious, and dysplastic pneumopathies were examined using histological, histochemical, and morphometric techniques. The results revealed pronounced thickening of alveolar septa, partial destruction of the epithelial lining, capillary congestion, and compensatory fibroblastic proliferation. Morphometric analysis showed a significant reduction in alveolar surface area and an increase in interalveolar septal thickness compared to healthy lung tissue. These findings confirm that pneumopathies are accompanied by deep structural and functional remodeling of the alveolar component, directly affecting gas exchange efficiency.

Key words: pneumopathy, alveoli, morphology, morphometry, lung tissue, epithelial damage, fibrosis, remodeling

Introduction

The alveolar structures of the lungs represent the terminal component of the respiratory system and play a vital role in gas exchange between the external environment and the bloodstream. Any pathological alteration in the morphology of alveoli, their epithelial lining, or interalveolar septa directly affects the functional capacity of the lungs. Pneumopathies, a broad group of diseases that include inflammatory, infectious, allergic, and fibrosing conditions, are among the leading causes of structural lung remodeling in both children and adults.

The pathogenesis of pneumopathies is multifactorial, involving immune-inflammatory reactions, vascular disturbances, hypoxia, and cellular damage to the alveolar epithelium. Repeated or prolonged inflammatory processes lead to destruction of the alveolar-capillary membrane, edema, and deposition of fibrous tissue. These changes reduce the elasticity and surface area of the alveoli, resulting in impaired ventilation and diffusion capacity.

From a morphofunctional point of view, the study of alveolar structures in pneumopathies is essential for understanding the mechanisms of respiratory failure and chronic lung disease formation. Histopathological and morphometric analyses provide objective criteria for evaluating the severity and reversibility of pulmonary lesions. The identification of characteristic microscopic patterns and quantitative structural indices offers insight into the processes of damage, repair, and adaptation occurring within lung parenchyma.

The purpose of this research was to analyze the specific pathomorphological and morphometric features of alveolar structures in different types of pneumopathies and to determine how these changes influence the integrity of the alveolar-capillary interface and gas exchange function.

Materials and Methods

The study was performed on 60 lung tissue samples obtained from autopsy and surgical biopsy materials. The cases were divided into three groups according to the clinical and morphological diagnosis: inflammatory pneumopathies (bronchopneumonia, interstitial pneumonia), infectious pneumopathies (viral and bacterial origin), and fibrosing or dysplastic pneumopathies (chronic interstitial fibrosis and idiopathic pulmonary fibrosis). Ten samples of morphologically intact lung tissue served as the control group.

Histological examination was carried out using hematoxylin and eosin staining, Van Gieson's collagen staining, and PAS reaction for basement membrane integrity. Morphometric analysis included measurements of alveolar diameter, surface area, and interalveolar septal thickness. Quantitative evaluation was performed using digital microscopy with calibrated imaging software. All data were statistically analyzed using SPSS 26.0 software. Mean values (M) and standard deviations (SD) were calculated, and intergroup differences were assessed by Student's t-test with significance accepted at $p < 0.05$.

Results

Microscopic examination of lung tissue in pneumopathies revealed pronounced structural disturbances in the alveolar framework. In inflammatory pneumopathies, the alveoli were partially filled with serous and fibrinous exudate, and infiltration by neutrophilic and lymphocytic elements was observed. The alveolar septa were thickened, and capillary lumens showed congestion and perivascular edema. The epithelial lining of alveoli demonstrated signs of desquamation, and type II pneumocytes exhibited hyperplasia as a compensatory response.

In infectious pneumopathies, the lesions were characterized by massive infiltration of polymorphonuclear leukocytes, areas of necrosis, and destruction of alveolar walls. The capillary network was irregular, with microthrombosis in some regions. The interalveolar septa displayed disorganization of collagen fibers and focal hemorrhages.

In fibrosing and dysplastic pneumopathies, a different pattern was observed. The alveolar cavities were deformed or collapsed, and interalveolar septa were markedly thickened due to proliferation of fibroblasts and excessive deposition of collagen and elastin. The basement membranes were unevenly thickened, and alveolar-capillary continuity was disrupted.

Morphometric evaluation revealed a significant increase in the average septal thickness (from 2.3 ± 0.4 μm in the control group to 6.7 ± 0.8 μm in fibrosing pneumopathies, $p < 0.001$). The mean alveolar diameter was reduced by approximately 30%, and the total alveolar surface area decreased correspondingly. The ratio of capillary lumen to septal thickness indicated a marked decline in microcirculatory efficiency.

Discussion

The obtained data confirm that pneumopathies are accompanied by deep structural and morphometric alterations of the alveolar component, which collectively impair gas exchange function. The thickening of interalveolar septa represents both an inflammatory and reparative process involving endothelial activation, fibroblast proliferation, and extracellular matrix

deposition. The resulting fibrosis reduces the compliance of the lung tissue, making respiration energetically inefficient.

The observed hyperplasia of type II pneumocytes and partial desquamation of the alveolar lining indicate an attempt of the epithelium to restore the damaged air–blood barrier. However, in chronic or recurrent pneumopathies, this regenerative response becomes incomplete or maladaptive, leading to permanent remodeling of alveolar architecture. Microthrombosis and vascular congestion further aggravate hypoxia and contribute to the vicious cycle of tissue injury and repair.

The morphometric findings correspond to previous experimental studies that demonstrated a strong correlation between septal thickening and decreased diffusion capacity of the lungs. The reduction in alveolar surface area limits the effective area for oxygen–carbon dioxide exchange, which clinically manifests as progressive dyspnea and reduced arterial oxygen saturation.

Thus, pneumopathies of varying etiology share common morphological features—damage to the alveolar-capillary barrier, fibroblastic proliferation, and altered tissue architecture—though the degree of reversibility depends on the balance between destructive and reparative processes. Early recognition of these changes through biopsy and imaging morphometry can serve as a predictor of disease progression and guide therapeutic interventions aimed at limiting fibrosis and promoting alveolar regeneration.

Conclusion

Pneumopathies lead to complex structural and morphometric remodeling of the alveolar apparatus, which underlies the deterioration of pulmonary function. The study established that inflammatory and infectious pneumopathies are characterized by acute epithelial damage, exudation, and septal congestion, whereas fibrosing and dysplastic pneumopathies involve chronic reparative fibrosis with irreversible deformation of alveolar walls. Morphometric indices demonstrated a significant decrease in alveolar surface area and a marked increase in septal thickness, both of which contribute to impaired gas diffusion and ventilation-perfusion mismatch.

The presence of epithelial desquamation, capillary thrombosis, and fibroblast proliferation suggests that alveolar remodeling is not merely a passive consequence of inflammation but an active, self-sustaining process involving cellular reprogramming and altered extracellular matrix metabolism. These findings emphasize the importance of early diagnosis, histological monitoring, and timely application of anti-inflammatory and antifibrotic therapy to preserve alveolar integrity. Morphometric criteria may serve as valuable quantitative indicators for evaluating treatment efficacy and predicting long-term outcomes in pneumopathies.

In summary, the pathomorphological and morphometric alterations observed in pneumopathies reflect the fundamental mechanisms of damage, adaptation, and fibrosis in the lung parenchyma. Recognition of these features provides a morphological basis for targeted therapy and for the prevention of irreversible pulmonary remodeling.

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