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RESPIRATORY SYSTEM PATHOLOGY: STRUCTURAL AND FUNCTIONAL CHANGES OF THE LUNGS

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Abstract. Respiratory system pathology represents a significant area of modern medicine due to the high prevalence and clinical impact of lung diseases. The lungs are highly specialized organs responsible for gas exchange, making them particularly vulnerable to a wide range of pathological processes [1,2,3]. This article examines the structural and functional changes of the lungs in various pathological conditions, including inflammatory, infectious, and chronic diseases. Special attention is given to morphological alterations such as edema, fibrosis, and tissue remodeling, which contribute to impaired respiratory function. The relationship between histopathological changes and clinical manifestations is also analyzed, highlighting the importance of early diagnosis and timely intervention. Understanding the underlying mechanisms of lung pathology provides valuable insights for improving diagnostic approaches and therapeutic strategies in respiratory medicine[4,5,6].

Keywords: lung pathology, respiratory system, structural changes, functional impairment, pulmonary diseases, inflammation, fibrosis, edema, hypoxia, gas exchange, histopathology, morphological changes, pathogenesis, acute and chronic lung diseases

INTRODUCTION

The respiratory system plays a vital role in maintaining homeostasis by ensuring continuous gas exchange between the external environment and the internal milieu[7,8,9]. The lungs, as the central organs of this system, provide a large surface area for efficient oxygen uptake and carbon dioxide elimination. Their delicate alveolar-capillary structure, however, also makes them highly vulnerable to various damaging factors, including infectious agents, toxic substances, environmental pollutants, and immune-mediated processes[10,11].

In recent decades, the global burden of respiratory diseases has increased significantly due to urbanization, industrialization, smoking, and environmental degradation. Both acute and chronic lung conditions—such as pneumonia, chronic obstructive pulmonary disease (COPD), asthma, and interstitial lung diseases—contribute substantially to morbidity, disability, and mortality. These disorders are characterized by complex pathological processes that affect both the structure and function of lung tissue[13].

From a pathological anatomy perspective, lung diseases involve a wide spectrum of morphological changes. These include inflammatory cell infiltration, vascular alterations, alveolar damage, interstitial thickening, fibrosis, and airway remodeling. Such structural abnormalities disrupt normal lung architecture and impair physiological functions, particularly ventilation and perfusion, ultimately leading to reduced efficiency of gas exchange. In severe cases, these changes may progress to respiratory failure.

At the cellular level, pathological processes in the lungs often involve epithelial injury, endothelial dysfunction, activation of inflammatory pathways, and extracellular matrix remodeling. The interplay between these mechanisms determines the severity and progression of disease. For instance, persistent inflammation may lead to chronic structural damage, while inadequate repair mechanisms can result in irreversible fibrosis[12].

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Advances in histopathology, imaging techniques, and molecular biology have significantly improved the understanding of lung pathology. Modern research emphasizes not only morphological assessment but also the correlation between structural alterations and clinical manifestations. This integrated approach is essential for early diagnosis, accurate disease classification, and the development of targeted therapeutic strategies.

Therefore, studying the structural and functional changes of the lungs in pathological conditions is of great scientific and clinical importance. A comprehensive understanding of these processes allows for better interpretation of disease mechanisms and contributes to improving outcomes in patients with respiratory disorders.

Research objective. The objective of this study is to investigate the structural and functional changes of the lungs in various pathological conditions of the respiratory system. The research aims to analyze morphological alterations in lung tissue, including inflammatory processes, edema, fibrosis, and remodeling, and to evaluate their impact on respiratory function. Additionally, the study seeks to establish a correlation between histopathological findings and clinical manifestations in order to improve understanding of disease mechanisms, enhance diagnostic accuracy, and contribute to the development of effective therapeutic strategies in respiratory medicine.

Materials and methods. This study is based on a comprehensive analysis of lung pathology using both morphological and clinical approaches. Lung tissue samples were obtained from biopsy and autopsy materials, including cases of acute and chronic respiratory diseases. The selected samples represented a range of pathological conditions such as inflammatory, infectious, and degenerative lung disorders.

Histological examination was performed using standard staining techniques, including hematoxylin and eosin (H&E), to assess general tissue architecture and cellular changes. In selected cases, special stains and immunohistochemical methods were applied to identify specific cellular components, inflammatory markers, and patterns of tissue remodeling. Microscopic evaluation focused on identifying structural alterations such as edema, fibrosis, inflammatory infiltration, vascular changes, and alveolar damage.

Methods: Histological analysis of lung tissue samples was performed using standard hematoxylin and eosin (H&E) staining to evaluate general morphology. Additional special staining and immunohistochemical techniques were applied to identify inflammatory cells, fibrotic changes, and vascular alterations. Microscopic examination focused on assessing edema, fibrosis, cellular infiltration, and alveolar damage. Clinical and laboratory data were analyzed to correlate morphological findings with functional impairment. Comparative and descriptive methods were used to distinguish between acute and chronic pathological processes.

Results. The analysis demonstrated age-related differences in the structural and functional alterations of lung tissue across various pathological conditions. The study population was divided into three age groups: young (18–35 years), middle-aged (36–60 years), and elderly (>60 years).

In the young group (18–35 years), acute inflammatory changes predominated. Histological examination revealed pronounced neutrophilic infiltration, mild to moderate alveolar edema, and relatively preserved lung architecture. Structural damage was generally limited, and functional impairment was minimal, with only slight disturbances in gas exchange.

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In the middle-aged group (36–60 years), both acute and chronic pathological features were observed. There was moderate inflammatory infiltration with a mixed cellular pattern, including neutrophils, lymphocytes, and macrophages. Alveolar wall thickening, interstitial edema, and early fibrotic changes were more evident. Functional assessment indicated moderate impairment of ventilation and gas exchange.

In the elderly group (>60 years), chronic and degenerative changes were predominant. Histological findings included extensive fibrosis, marked interstitial thickening, and significant remodeling of lung architecture. Vascular congestion and reduced capillary density were also observed. Inflammatory infiltration was mainly lymphocytic and macrophage-dominated. These structural changes were associated with pronounced functional impairment, including severe reduction in gas exchange efficiency and increased incidence of respiratory insufficiency.

In chronic pathological conditions, the most prominent features included diffuse interstitial fibrosis, destruction of alveolar septa, and significant architectural distortion of lung parenchyma. Fibrotic thickening led to reduced elasticity of lung tissue and narrowing of small airways. Bronchiolar remodeling with epithelial hyperplasia and goblet cell metaplasia was frequently observed. These changes were associated with long-standing inflammatory activity and impaired tissue repair mechanisms.

Vascular changes included endothelial damage, capillary rarefaction, and in some cases, pulmonary hypertension-related remodeling of small arteries. These alterations contributed to worsening ventilation-perfusion mismatch.

Functionally, there was a clear progression of respiratory impairment corresponding to the severity of structural damage. Mild dysfunction was associated with acute reversible changes, whereas chronic fibrotic transformation resulted in significant and often irreversible reduction in gas exchange capacity, decreased lung compliance, and development of chronic respiratory insufficiency.

Age-stratified analysis confirmed that elderly patients exhibited more advanced structural destruction, higher fibrosis scores, and more pronounced functional decline compared to younger groups. In contrast, younger patients showed predominantly reversible inflammatory changes with better recovery potential.

Overall, the severity of morphological alterations and functional decline increased with age, demonstrating a clear correlation between aging and progression of lung pathology.

Conclusion. The study demonstrates that pathological processes in the lungs are characterized by significant structural and functional alterations that vary depending on the nature and duration of the disease as well as the patient's age. Acute conditions are mainly associated with inflammatory changes and reversible tissue damage, whereas chronic processes lead to progressive fibrosis, architectural remodeling, and persistent impairment of lung function.

A clear correlation was identified between morphological changes and the degree of functional disturbance, particularly in gas exchange efficiency. Age-related analysis showed that the severity of structural damage and functional decline increases with advancing age, with elderly individuals exhibiting more pronounced and often irreversible changes.

These findings highlight the importance of early detection and timely intervention in respiratory diseases to prevent progression to chronic and debilitating stages. A comprehensive

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understanding of the relationship between structural alterations and functional impairment in the lungs is essential for improving diagnostic accuracy and optimizing therapeutic strategies in modern respiratory medicine.

As practical recommendations, a multistage clinical algorithm is proposed:

1. Initial assessment: Evaluation of patient history, risk factors (smoking, occupational exposure, infections), and presenting respiratory symptoms.
2. Clinical examination: Physical examination focusing on respiratory rate, auscultation findings, and signs of respiratory insufficiency.
3. Laboratory diagnostics: Basic blood tests, inflammatory markers, and, when indicated, microbiological analysis.
4. Imaging studies: Chest X-ray and/or computed tomography (CT) to identify structural abnormalities in lung tissue.
5. Functional assessment: Evaluation of pulmonary function using spirometry and assessment of gas exchange (e.g., oxygen saturation, blood gases).
6. Morphological verification: Histological and, if necessary, immunohistochemical examination of lung tissue (biopsy) to confirm diagnosis.
7. Differential diagnosis: Distinguishing between acute and chronic conditions, infectious and non-infectious processes, and identifying specific disease entities.
8. Treatment strategy selection: Development of individualized therapeutic approaches based on the underlying pathology and severity of functional impairment.
9. Monitoring and follow-up: Regular assessment of clinical status, lung function, and response to treatment to prevent disease progression and complications.

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