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MORPHOLOGICAL AND PATHOLOGICAL CHANGES IN THE LUNGS DUE TO COVID-19

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Abstract: The COVID-19 pandemic has significantly impacted not only global healthcare systems but also the human body. The SARS-CoV-2 virus damages both the upper and lower respiratory tracts, leading to severe inflammatory processes in the lungs. The acute and chronic phases of this disease can cause serious pathological conditions such as alveolar damage, microthrombosis, and fibrosis.

This article provides a comprehensive review of the morphological and pathological changes in the lungs due to COVID-19, explaining how the virus affects alveolar cells, triggers inflammatory processes, and leads to long-term complications.

Keywords: COVID-19, SARS-CoV-2, lung pathology, alveolar damage, fibrosis, thrombosis, cytokine storm, microthrombosis, diffuse alveolar damage (DAD), pulmonary fibrosis.

Main Section

Mechanism of COVID-19 Entry and Damage to the Lungs

COVID-19 is transmitted through airborne droplets and reaches the lungs via the upper respiratory tract. The virus binds to ACE2 receptors and primarily infects alveolar epithelial cells. This process occurs in the following stages:

1. Entry of the Virus into Alveolar Cells

The S-spike protein of the SARS-CoV-2 virus binds to type II pneumocytes in the alveoli.The virus enters the cells, replicates its RNA, and destroys them.

2. Immune Response and Inflammation

Cytokines (IL-6, IL-1β, TNF-α) released by infected cells initiate an inflammatory response.Neutrophils and macrophages migrate to the alveoli to eliminate the pathogens.

3. Microthrombosis and Vascular Damage

The inflammatory process damages endothelial cells, leading to thrombosis. This disrupts blood circulation in the alveoli, causing hypoxemia and respiratory failure.

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4. Pulmonary Fibrosis and Long-Term Damage

After the acute inflammation phase, fibroblasts become activated in the alveoli. The accumulation of connective tissue leads to pulmonary fibrosis, resulting in long-term breathing difficulties.

Morphological Changes in the Lungs Due to COVID-19

COVID-19 induces various pathological processes in lung tissue. The most significant ones include:

1. Diffuse Alveolar Damage (DAD)

DAD is one of the main pathological features of COVID-19 pneumonia. It progresses in three phases:

Exudative Phase=> Severe inflammation and fluid accumulation in the lungs. Damage to alveolar walls, formation of hyaline membranes.Patients experience severe respiratory distress.

Proliferative Phase=> Activation of fibroblasts leads to the regeneration of cells. Thickening of alveolar walls but loss of elasticity.

Fibrotic Phase=> Pulmonary fibrosis develops due to excess connective tissue. This impairs lung function, causing long-term complications.

2. Microthrombosis and Circulatory Disorders

In 30-50% of COVID-19 patients, thrombi are found in pulmonary capillaries.

Consequences include:

Pulmonary embolism: Thrombi obstruct large arteries, leading to acute respiratory failure.

Impaired gas exchange: Oxygen deficiency occurs.

Pulmonary hypertension: Increased vascular pressure puts strain on the right side of the heart.

3. Cytokine Storm and Alveolar Inflammation

One of the most dangerous mechanisms of COVID-19 is the cytokine storm. The immune system overreacts, producing excessive inflammatory mediators (IL-6, IL-1β, TNF-α). Uncontrolled inflammation damages the alveoli. Patients deteriorate rapidly, often requiring mechanical ventilation.

Long-Term Effects of COVID-19 on the Lungs

The long-term consequences of COVID-19 pneumonia include:

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1. Pulmonary Fibrosis: Loss of elastic tissue disrupts normal lung function.

2. Chronic Respiratory Failure: Alveolar dysfunction causes persistent shortness of breath.

3. Post-COVID Syndrome: Patients may suffer from prolonged fatigue, breathing difficulties, and cardiovascular issues.

Pathological Features and Clinical Manifestations

The pathological changes in the lungs due to COVID-19 can manifest in various clinical forms. The main symptoms include:

Acute Respiratory Failure: Impaired oxygen exchange due to alveolar tissue damage.

Hypoxemia: Reduced oxygen levels in the blood due to impaired gas exchange.

Pulmonary Fibrosis: Long-term COVID-19 pneumonia leads to scarring of lung tissue, restricting lung capacity.

Hemorrhagic Pneumonia: Bleeding in the alveoli and rupture of small blood vessels may occur.

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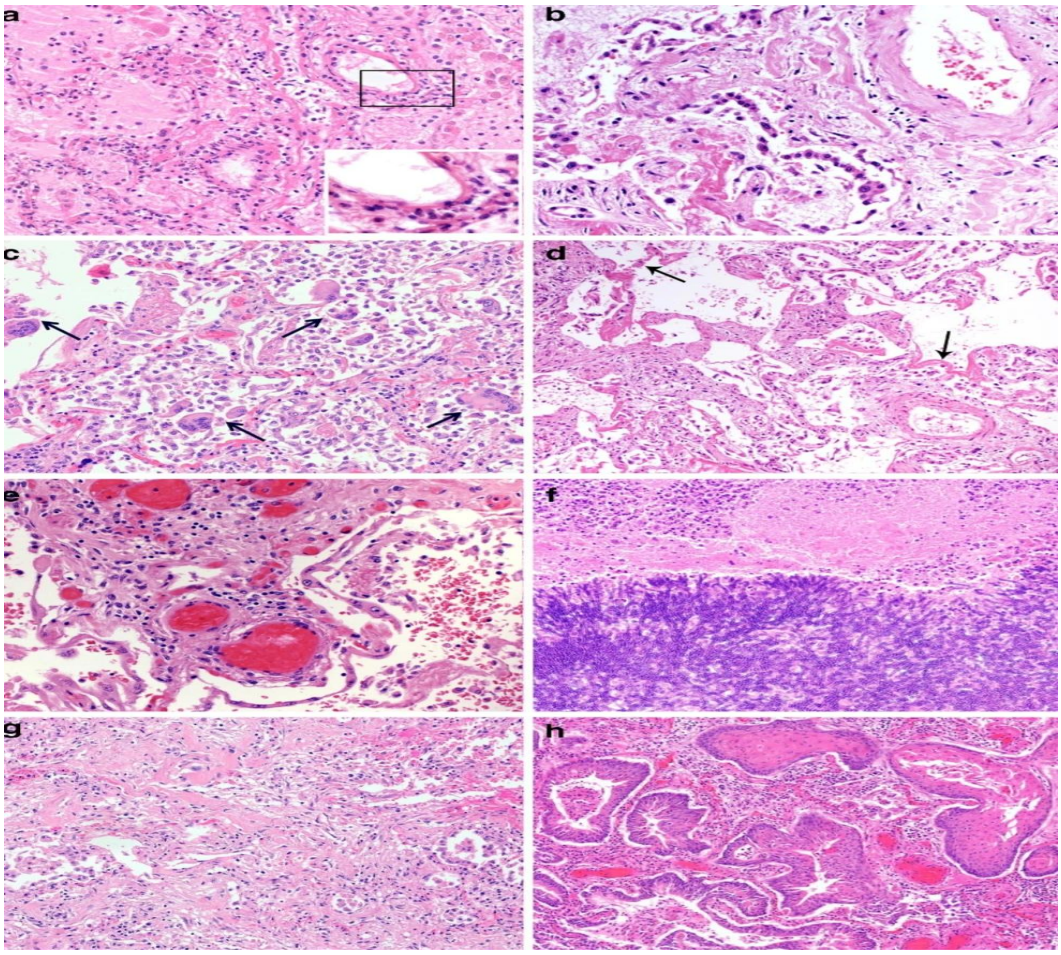

Lethal Pulmonary Pathology in COVID-19

1. Early Lung Damage: Alveolar edema, endothelial necrosis, microthrombosis, and endothelialitis.Progressive inflammatory vascular damage with nuclear debris and granulocytes.

2. Exudative Phase of DAD: Hyperplasia of type II pneumocytes.

3. Formation of Giant Intra-Alveolar Cells: Observed in the exudative phase of DAD.

4. Hyaline Membranes and Fibrin Deposits: Present in the exudative phase of DAD.

5. Thrombosis in Small and Medium-Sized Vessels: Occurs in the early stages of lung disease.

6. Invasive Aspergillosis: A rare form of superinfection in the late phase of DAD.

7. Organizing Phase of DAD: Loose connective tissue fibrosis in interstitial and intramural spaces. Intra-alveolar plugs.

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8. Late Phase of DAD Organization: Massive bronchopulmonary squamous metaplasia.

Conclusion

COVID-19 infection causes complex morphological and pathological changes in lung tissue. Processes such as diffuse alveolar damage, capillary thrombosis, and pulmonary fibrosis contribute to the severity of the disease. Early diagnosis, intensive treatment, and rehabilitation are crucial in minimizing long-term complications of COVID-19 pneumonia. Treatment strategies should focus on antiviral drugs, immunomodulators, and pulmonary rehabilitation.

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