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Pathophysiology and management of emphysema: From smoking cessation to surgical intervention.

Farah Tanaka*

Department of Respiratory Medicine, United Arab Emirates University, United Arab Emirates

***Correspondence to:** Farah Tanaka, Department of Respiratory Medicine, United Arab Emirates University, United Arab Emirates. E-mail: f.tanaka@uaeu.ac.ae

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Introduction

Emphysema, a primary subtype of chronic obstructive pulmonary disease (COPD), is characterized by irreversible destruction of the alveoli — the tiny air sacs in the lungs responsible for gas exchange. This progressive disease results in the loss of elastic recoil, air trapping, and reduced surface area for oxygen absorption, ultimately impairing respiratory function. While smoking remains the leading cause of emphysema, various environmental and genetic factors also contribute to its pathogenesis [1].

At the core of emphysema's pathophysiology is an imbalance between proteases and antiproteases in the lungs. Cigarette smoke and other inhaled irritants trigger an inflammatory response, releasing enzymes such as neutrophil elastase that degrade elastin and other structural components of the alveolar walls. Normally, these enzymes are counteracted by protective proteins like alpha-1 antitrypsin (AAT). In individuals with AAT deficiency — a genetic condition — this protective mechanism fails, making them highly susceptible to early-onset emphysema even without a smoking history [2].

There are several types of emphysema, categorized based on their anatomical distribution. Centrilobular emphysema, the most common form, primarily affects the upper lobes and is strongly linked to smoking. Panlobular emphysema involves uniform destruction of the alveoli throughout the lobule and is commonly associated with AAT deficiency. Paraseptal emphysema, typically seen in younger individuals, affects the distal parts of the acinus and is often linked to spontaneous pneumothorax [3].

Clinically, emphysema presents with symptoms such as progressive dyspnea (shortness of breath), chronic cough, wheezing, and decreased exercise tolerance. In advanced stages, patients may exhibit signs of hyperinflation (barrel chest), use of accessory muscles for breathing, and weight loss. Diagnosis is typically confirmed by spirometry, which shows a reduced FEV1/FVC ratio, and imaging studies such as high-resolution CT scans that reveal characteristic airspace enlargement and destruction of alveolar walls [4].

The cornerstone of emphysema management is smoking cessation, which is the only intervention proven to slow the progression of the disease. Quitting smoking significantly reduces inflammation, preserves remaining lung function, and decreases the frequency of exacerbations. Pharmacologic aids such as nicotine replacement therapy, varenicline, and bupropion, combined with behavioral counseling, greatly enhance cessation success rates [5].

Conclusion

In conclusion, emphysema is a complex, progressive lung disease with a multifaceted approach to management. From addressing modifiable risk factors like smoking to leveraging surgical innovations for advanced disease, the treatment strategy must be individualized and comprehensive. As research advances, there is growing hope for more effective therapies that can halt or even reverse some aspects of lung damage, ultimately improving the lives of those affected by this debilitating condition.

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