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Commentary

Pathophysiology and Diagnosis of Chronic Obstructive Pulmonary Disease

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DESCRIPTION

Chronic, partially reversible inadequate airflow (airflow limitation) and the inability to fully exhale (air entrapment) are symptoms of COPD, a progressive lung disease. The condition of the tiny airways and emphysema are the cause of the inadequate airflow (the breakdown of lung tissue). People respond differently to these two factors' respective contributions. Lung hyperinflation is preceded by air trapping.

Small airways disease, also known as bronchial and alveolar remodelling in the lung, is ultimately what causes COPD to develop as a major and chronic inflammatory response to inhaled irritants. Therefore, emphysema and airway remodelling with peripheral airway narrowing are to blame for the change in lung function. When cilia and mucus production are dysregulated, mucociliary clearance is significantly affected. It indicates that small airway illness, also known as chronic bronchiolitis, is the condition that leads to the development of emphysema. Two categories of white blood cells, neutrophils and macrophages, are the inflammatory cells in question. Cytotoxic T cells are also involved in smokers, and some COPD patients show eosinophil involvement similar to that in asthma. Inflammatory mediators like chemotactic factors contribute to this cell response. Other processes that contribute to lung damage include oxidative stress, which is released by inflammatory cells and caused by the high levels of free radicals in tobacco smoke, as well as the breakdown of the connective tissue in the lungs by proteases (particularly elastase), which are insufficiently inhibited by protease inhibitors. Emphysema results from the breakdown of the lungs' connective tissue, which consequently affects airflow and the absorption and expulsion of breathing gases. General strength The wasting that frequently develops in COPD may be partially brought on by inflammatory mediators that the lungs release into the blood.

The airways narrow as a result of internal inflammation and scarring. The difficulty to exhale fully is a result of this. When breathing out, there is a significant decrease in airflow because the airways are being compressed by the chest's pressure. This might cause more air from the previous breath to remain in the lungs when the next breath is taken, increasing the amount of air in the lungs at any given time. This is a process known as air trapping, which is immediately followed by hyperinflation. Exercise-induced hyperinflation is associated with shortness of breath in COPD because partial lung filling makes breathing in less comfortable. Exacerbations can potentially worsen hyperinflation. There might also exhibit a degree of irritant-induced airway hyper responsiveness comparable to that seen in asthma.

Low ventilation from airway blockage, hyperinflation, and a diminished desire to breathe can result in low oxygen levels and subsequently high carbon dioxide levels in the blood. These conditions can lead to poor gas exchange and poorer oxygenation. Airway inflammation is also worsened during exacerbations, which leads to greater hyperinflation, decreased expiratory airflow, and worsened gas transfer. This can result in low blood oxygen levels, and emphysema causes the capillaries in the lungs to break down. If these conditions persist for a long time, they can constrict the arteries in the lungs. Both of these disorders have the potential to lead to pulmonary heart disease, also referred to as cor pulmonale in the past

Diagnosis

Anyone over the age of 35 to 40 with shortness of breath, a chronic cough, sputum production, or frequent winter colds should be evaluated for COPD if they have a history of exposure to risk factors for the condition. The diagnosis is then verified using spirometry.

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