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Understanding the Mechanisms that Lead COPD to Develop and Advance

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Introduction

Globally, chronic obstructive pulmonary disease (COPD) is a condition that is quite common and a leading cause of death. It is characterized by a chronic inflammatory process in the airways and lung parenchyma brought on by noxious particles or gases, mainly cigarette smoke, and is characterized by progressive airflow limitation. The primary anatomical abnormalities are small airways disease and parenchymal damage, while pulmonary vascular alterations also play a significant role in the disease. Pulmonary hypertension, a complication that may arise in more than 50% of patients with an advanced illness, may develop as a result of changes in artery anatomy. Pulmonary hypertension has been linked to lower survival times and more exacerbation events.

Description

Vascular remodeling in the pulmonary vasculature of COPD patients is evident and primarily affects small size vessels. The most noticeable characteristic of pulmonary muscle arteries is intimal hyperplasia. Although it is noticeable in vessels of all sizes, tiny arteries with a diameter of less than 500 m are where it is most noticeable. The arterioles are likewise muscularized and exhibit hypertrophy of the intima. The majority of investigations have failed to demonstrate any notable differences in the thickness of the muscle layer between COPD patients and controls, while changes in the tunica media are less obvious [1].

The proliferation of cells that express smooth muscle -actin and other mesenchymal markers like vimentin results in intimal hyperplasia. Additionally, collagen and elastic fibers are deposited. Patients with varying degrees of COPD severity exhibit pulmonary vascular remodeling, and the presence of pulmonary hypertension does not appear to be linked to a higher disruption of the arterial structure. Additionally, heavy smokers with normal lung function also experience intimal hyperplasia and muscularization of tiny pulmonary arteries of a similar size to that seen in COPD. In reality, research on experimental COPD models shows that pulmonary vascular alterations occur before the onset of emphysema. A greater number of inflammatory cells, primarily activated T lymphocytes with a majority of the CD8+ T cell subset, are found infiltrating the adventitia of the pulmonary muscle arteries in patients with COPD and smokers without airflow obstruction. Neutrophils, macrophages, and B lymphocytes are in small numbers and are comparable to control nonsmokers [2].

When examined under an electron microscope, the endothelial surface of the pulmonary arteries of COPD patients may exhibit areas of endothelial denudation and a loss of cell connections. Changes in endothelial function and the production of angiogenic and growth factors accompany changes in vessel structure in COPD. Individuals with end-stage COPD who underwent lung transplantation as well as patients with mild-to-moderate COPD have both been reported to have reduced endothelium-dependent relaxation, which indicates endothelial dysfunction of the pulmonary arteries. Reduced expression of the enzymes prostacyclin synthase (PGI2-S) and endothelial nitric oxide synthase (eNOS), which promote vascular contraction and cell proliferation, are related with endothelial dysfunction. Patients with COPD also have higher levels of vascular endothelial growth factor (VEGF) and the transforming growth factor-beta receptor-II (TGF-RII) in their pulmonary arteries [3].

Cigarette smoke impacts on COPD

Hypoxia and inflammation are two potential pathways that may be involved in the development of endothelial dysfunction and vascular remodeling in pulmonary arteries. The potential implications of cigarette smoke products as beginning elements of the pulmonary vascular derangement in COPD, however, have come under scrutiny recently. This claim is supported by circumstantial evidence, as most alterations seen in the pulmonary arteries of COPD patients have also been seen in the pulmonary arteries of non-smokers. According to research from our lab, "healthy" smokers who do not have airflow obstruction exhibit intimal hyperplasia, decreased eNOS expression, increased VEGF expression, and inflammatory cell infiltrate in the pulmonary arteries at levels similar to those of patients with COPD, which is obviously different from nonsmokers.

There are more parallels in lung gene expression between "healthy" smokers and patients with mild-to-moderate COPD than between the latter and those with end-stage COPD, according to a recent study analyzing genes potentially involved in pulmonary vascular alterations. This has led to the theory that tobacco smoke products are crucial in starting the vascular alterations that could ultimately lead to pulmonary hypertension in people with COPD [4].

Cells of progenitor

Intimal hyperplasia, which is brought on by the proliferation of cells that exhibit common mesenchymal and smooth muscle cell (SMC) markers but lack certain markers unique to mature SMC, such desmin, occurs in the pulmonary arteries of people with COPD. Based on the expression pattern of intermediate filaments, it may be possible to discriminate between a synthetic SMC phenotype and the contractile phenotype seen in mature cells. Thus, a fraction of mesenchymal cells with the capacity to generate new cells and take part in ongoing vascular remodeling may be poorly differentiated SMCs and myofibroblasts that are vimentin-positive but desmin-negative [5].

It is unclear where these poorly differentiated cells came from in the pulmonary artery intima. In an idealized scenario, a number of possibilities can be taken into account, including:

(1) Dedifferentiation of mature SMC in the muscular layer and migration toward the intima;

(2) Transdifferentiation from endothelial cells via an endothelial-to-mesenchymal transition process;

(3) Differentiation from resident precursor cells; and

(4) Recruitment and differentiation from circulating bone marrow-derived progenitor cells.

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Conclusion

The so-called endothelial progenitor cells (EPCs), which may aid in vascular re-endothelization and vasculogenesis, are among the progenitor cells that can be produced and mobilized by the bone marrow to regenerate various tissues. EPCs' functional characteristics and characterisation are still up for debate. We have discovered cells with positive immunoreactivity to CD133, a hallmark of progenitor cells coming from the bone marrow, in denuded portions of the endothelium and within the intimal layer of pulmonary arteries from patients with COPD. It's interesting to note that the amount of progenitor cells in the vessel wall was related to the remodeling of the pulmonary arteries as well as the contractile response to hypoxic stimuli, which is related to endothelial function.

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