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Chronic Pulmonary Obstructive Disease and its Pathophysiology

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Opinion Article

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DESCRITPION

Chronic Obstructive Pulmonary Disease (COPD), otherwise known as pulmonary emphysema and chronic bronchitis, is triggered by the exposure of the airways to potentially harmful gases or particles. The inhalation of these injurious gases consequently lead to the constant inflamed state of the airways themselves which are ultimately highlighted by clear obstruction of airflow and hyperinflation--unwanted expansion of the lung. On a purely mechanical scale, the effects of COPD on the respiratory system can be revealed through the understanding of pressure-volume (P-V) relationships. Airway blockages are categorized into three groups: (1) tissue abnormality around the airways, (2) airway wall thickening, (3) partial obstruction of lumina; in this case, the bronchi located in the lungs. The first group mentioned involves airway constriction due to the rapid loss of radial traction while the second group is caused by muscle hypertrophy or edema. Finally, the third group is a direct result of an accumulation of mucous that lead to the formation of semisolid plugs around the airways. Both emphysema and chronic bronchitis are associated with COPD and are therefore characterized by the three groups mentioned above.

Pathology

In patients with COPD, there are clear pathological changes not only in small airways, but in larger ones as well. These changes are usually located around the pulmonary and parenchyma vasculature regions and are exemplary of continuous damage and repair of the airways themselves. The consequential inflammatory response triggered may be an internally genetic factor, or may be attributed to the harmful effects of cigarette smoke. Although the existence of cigarette smoke is an alarming indicator in the manifestation of COPD, there have been confirmed

instances where patients develop COPD without cigarette smoke exposure, which ultimately leads to the fundamental cause of this respiratory disease. As oxidative stress and imbalance grows between proteases and antiproteases, the inflammatory response of the disease is greatly amplified. Both these proteases serve to protect the connective tissue in the system from deteriorating and tension between them triggers the inflammatory state. More specifically, the inflammatory response is enhanced by the CD8+ cytotoxic Tc1 lymphocytes and inflammatory mediators–inflammatory cells. An explanation for these occurrences is embedded in the oxidative DNA damage of lung epithelial barrier cells. In detail, the damaging inhaled gases induce damage upon the DNA of the LEBCs and these mutations are highlighted at the microsatellite DNA level of the LEBCs themselves. The newly mutated LEBCs then relay information to the lymph nodes by way of dendritic cell recognition. Once the T lymphocytes receive this information, a CD8+ cytotoxic T-lymphocyte massive production results. Finally, these CD8+ T lymphocytes will release granzymes and perforin which will attract the mutated LEBCs, resulting in cellular destruction.

Pathophysiological characteristics

As mentioned, COPD is characterized by two symptoms: Hyperinflation and Airflow Obstruction. Hyperinflation is usually described by the loss of the elastic recoil present within parts of the respiratory system. This symptom is seen regularly in COPD patients stricken with emphysema and the body consequently tries to keep the airways open and the air trapped during premature closure. Hyperinflation undoubtedly has unwanted effects on not only the main respiratory system consisting of the lungs, but also the diaphragm. It tends to affect the function of the diaphragm to regulate breaths by increasing the work of breathing. Hyperinflation affects the diaphragm in two ways. First, the diaphragm is flattened which will lead to a decrease of the apposition zone between the abdominal wall and the diaphragm itself. Second, the muscle fibers of the diaphragm become shorter and are therefore unable to generate enough inspiratory pressures that will override trans-pulmonary pressures. Given these two effects, there will be an increase in the proportion of the type I fibers (fatigue resistant and slow twitch) and an increase in mitochondrial concentration and the efficiency of the electron transport chain. This occurrence will then lead to further impaired respiratory muscle function as the diaphragm becomes weaker.

Airflow during exhalation is defined as the balance between the airway resistance that limits flow and the elastic recoil of the lungs the promote flow. Hence, the factors that result in the blockages in the lumen along with the increased resistance are the presence of secretions, the hypertrophy of submucosal glands, and the increased tone of bronchial smooth muscle. It is the great difference resulting between the intraluminal pressure and the surrounding pressure that ultimately causes the airflow obstruction. For example, in emphysema, there is an obvious loss of elastic recoil on the walls of small airways due to the reduced numbers of elastic tissue in the pulmonary parenchyma. In addition, the lack of cartilage along the wall of these airways leads to even less elastic recoil. Pathological aspects of COPD, the dynamic and static representations of the lung in COPD vary as well. As mentioned several times, the loss of elastic recoil in the bronchi of the lungs will result in an abnormal pressure difference between the inside of the alveoli and the lung surface. This difference in pressure was defined as transpulmonary pressure. Thus, patients with emphysema have high compliance in their lungs which expands largely in comparison to a normal lung with low compliance, an event known as hyperinflation.