Pathogenesis of COPD

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Abstract:
Chronic Obstructive Pulmonary Disease (COPD) is a chronic inflammatory condition of the airways and alveoli leading to airflow limitation, giving rise to chronic cough, sputum production and breathlessness. Classically, tobacco exposure has been commonly linked to causation of COPD. However, many patients develop COPD without any history of tobacco exposure. Exposure to indoor pollution in the form of biomass fuel smoke or occupational exposure to smoke, may also contribute to development of COPD.

It is also known that pulmonary function reaches its peak (FEV1) at about twenty years of age and thereafter there is a gradual physiological decline in lung function. Due to any reason (like abnormal alveolar development in intrauterine life), if this peak lung function is not achieved in early adulthood, then a declining FEV1, at the same physiological rate, may give rise to early COPD symptoms and this process may further be hastened by smoke, tobacco exposure, infection etc. Infections like tuberculosis and systemic syndromes like Rheumatoid arthritis, HIV may also give rise to COPD symptoms.

In the presence(or absence) of genetic and epigenetic factors, due to presence of persistent, noxious stimuli, there can be oxidative stress, protease-antiprotease imbalance, abnormal inflammation with chemical mediators in the lung which lead to chronic airway inflammation with abnormal repair, giving rise to airflow limitation, mucus hyper-secretion, gas exchange abnormalities and gas trapping, that is COPD.

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