Pathogenesis of COPD

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Abstract:

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.

Biography:

Dr. Gargi Maitra, completed her MBBS from North Bengal University, stood 1st class 1st in her MBBS. She completed her MD in Pulmonary Medicine followed by Indian Diploma in Critical Care Medicine. She has worked as an Associate Editor for 'Bronchoscopy in ICU, A Practical guide' book. She has keen interest in critical care and interventional bronchoscopic procedures and has been actively involved in conducting and participating in various conferences at national level on Pulmonology and Bronchoscopy. She has presented cases in Lung India journal. She is an active member of Indian Chest Society (ICS), Indian Society of Critical Care Medicine (ISCCM), American College of Chest Physician (ACCP) and European Respiratory Society (ERS). Certain genetic factors like alpha-1 antitrypsin deficiency, single genes like matrix metalloproteinase 12, glutathione S transferase have all been linked to declining lung function giving rise to COPD as well.

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.

Recent Publications:

1. Alvar Agusti, James C. Hogg, 2019. Update on the Pathogenesis of Chronic Obstructive Pulmonary Disease. N Engl J Med:381;1248-56.
2. Global Strategy for the Diagnosis, Management and Prevention of Chronic Obstructive Pulmonary Disease, 2020. Global Initiative for Chronic Obstructive Pulmonary Disease; 8-13
3. Abraham SC, Wilentz RE, Yeo CJ, Sohn TA, Cameron JL, Boitnott JK, Hruban RH (2003) Pancreaticoduodenectomy (Whipple resections) in patients without malignancy: are they all 'chronic pancreatitis'? Am J Surg Pathol 27:110-120
4. Abramson MA, Jazag A, van der Zee JA, Whang EE (2007) ThMahadevane molecular biology of pancreatic cancer. Gastrointestinal Cancer Res: GCR 1:S7-S12
5. Aguirre AJ et al. (2003) Activated Kras and Ink4a/Arf deficiency cooperate to produce metastatic pancreatic ductal adenocarcinoma. Genes Dev 17:3112-3126. doi:10.1101/gad.1158703

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