Research Article

INTERNATIONAL RESEARCH JOURNAL OF PHARMACY

www.irjponline.com

ISSN 2230-8407 [LINKING]

A CLINICAL REVIEW ON CHRONIC OBSTRUCTIVE PULMONARY DISEASES AFTER COVID 19 PANDEMIC

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How to cite: Sinha Ashutosh Kumar[1]*, Rupajit Bhattacharjee[1], Himshikhar Sarma[1], Chiranjib Bhattacharjee[1], Indranil Ganguly[1], Kuntal Manna[2], Partha Sarthi Datta[3], Joydeb Acharjee[4], Priyanka Majumder[5] A CLINICAL REVIEW ON CHRONIC OBSTRUCTIVE PULMONARY DISEASES AFTER COVID 19 PANDEMIC. International Research Journal of Pharmacy, 2026,17:6:18-28.

Doi:10.32937/2230-8407.37347

ABSTRACT

Introduction: Chronic obstructive pulmonary disease (COPD) is a common, persistent inflammatory illness of the airways characterized by persisting respiratory symptoms and irreversible airflow limitation. Worldwide, COPD is the third leading cause of death, following ischemic heart disease and cerebrovascular illness, due to elevated morbidity and mortality rates.

Aim: Respiratory failure (RF), particularly hypercapnic respiratory failure (HRF), commonly manifests in the majority of people with severe or end-stage chronic obstructive pulmonary disease

(COPD).

Methods: Chronic Obstructive Pulmonary Disease exacerbated by Respiratory Failure (COPD+RF) exacerbates treatment challenges, leading to a diminished prognosis. Although the pathophysiology of RF in COPD remains unclear, abnormal immune activation and systemic inflammation are significant factors.

Results: The systemic immune-inflammation index (SII) is an integrative inflammatory marker calculated from peripheral lymphocyte, neutrophil, and platelet counts. Neutrophils, platelets, and synthesized cytokines are mostly associated with nonspecific immune responses. Conversely, lymphocytes are predominantly associated with immune-related pathways in COPD patients.

Conclusions: Chronic Obstructive Pulmonary Disease (COPD) is a significant airway disorder that necessitates attention and the development of therapeutic formulations for its treatment.

Keywords: Etiology of COPD, Pathophysiology, Risk Factors, Symptoms, COVID-19

INTRODUCTION:

Chronic obstructive pulmonary disease (COPD) is a prevalent and manageable condition marked by progressive airflow restriction and tissue degradation. It is linked to structural alterations in the lungs resulting from chronic inflammation caused by extended exposure to harmful particles or gases, predominantly cigarette smoke. Chronic inflammation results in airway constriction and diminished lung elasticity. The illness frequently manifests with symptoms of cough, dyspnea, and sputum production[1]. Symptoms may vary from asymptomatic to respiratory failure. Chronic Obstructive Pulmonary Disease (COPD) is induced by extended exposure to detrimental particles or gasses. Cigarette smoking is the predominant cause of COPD globally. Additional factors may encompass second-hand smoke, environmental and occupational exposures, and alpha-1 antitrypsin deficiency (AATD) [2]. Chronic Obstructive Pulmonary Disease (COPD) predominantly affects smokers and individuals over the age of 40. The prevalence escalates with age, and it is presently the third most prevalent cause of morbidity and mortality globally. In 2015, the global prevalence of COPD was 174 million, resulting in roughly 3.2 million deaths attributable to the disease. Nonetheless, the

frequency is probably overestimated due to under diagnosis.

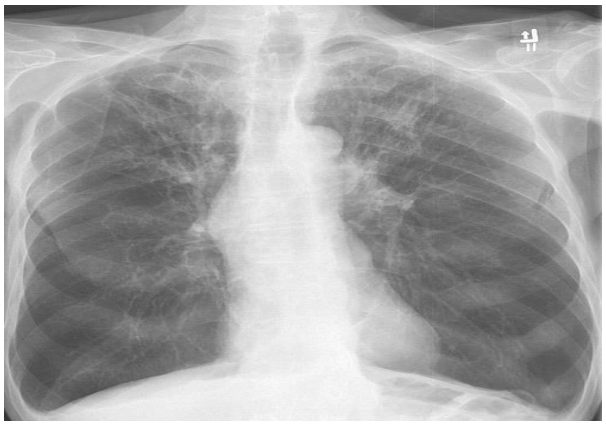

Figure 1: X-ray of the Chest, Chronic Obstructive Pulmonary Disease

MATERIALS AND METHODS:

What are the causes of COPD?

Prolonged exposure to pulmonary irritants such as tobacco smoke or chemicals can harm your lungs and airways [3]. Prolonged exposure can result in chronic obstructive pulmonary disease (COPD), encompassing chronic bronchitis and emphysema [4]. Smoking is the primary cause of COPD; yet, one in four individuals with COPD has never smoked.

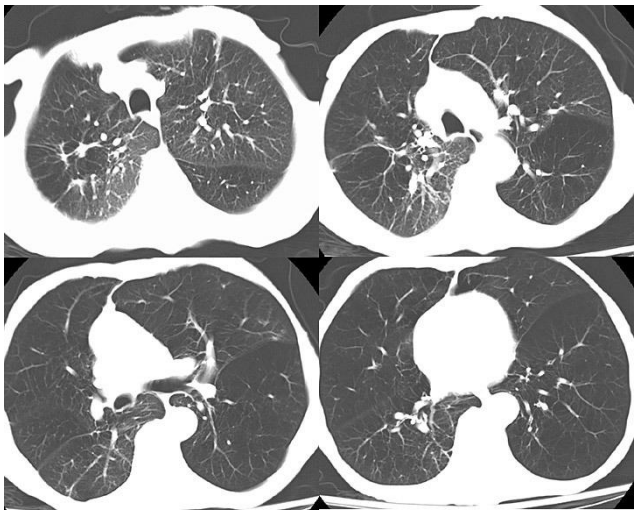

Figure 2: CT scan COPD Left Lung

Risk Factors for COPD Following COVID-19 Situations:COPD is frequently labeled as a "smoker's disease." However, while smoking is a primary risk factor for the development of

COPD, individuals who have never smoked can also be affected by this condition. Additional risk factors may encompass [5]:

• A record of respiratory infections during childhood.

• Smoke exposure from coal or wood burning stove.

• Exposure to environmental tobacco smoke.

• Individuals with a history of asthma.

• Individuals with underdeveloped lung capacity.

• Individuals aged 40 and above may experience a decline in lung function as they grow older.

RESULTS:

Minimize Risk for COPD: If we have concerns regarding the development of COPD, there are measures you can implement to mitigate your risk.

• Eliminate smoking permanently by accessing support, programs, and resources provided by the American Lung Association.

• Minimize exposure to secondhand smoke.

• Ensure you are current with your COVID-19, flu, and pneumonia vaccinations.

• Vaccinations provide essential protection against respiratory viruses.

• When exposed to chemicals, dust, and fumes in the workplace, it is essential to utilize the appropriate protective equipment and participate in safety training [6].

COPD is a progressive disease that may deteriorate over time. If you possess risk factors for COPD or are showing symptoms, it is advisable to consult your healthcare provider without delay. Receiving an early diagnosis of COPD allows for timely initiation of treatment.

Pathological Changes Observed in COPD Proximal Cartilaginous Airways (>2 mm in Diameter)

- An increase in the numbers of macrophages and CD8 T lymphocytes has been observed [7].
- A limited number of neutrophils and eosinophils are present (neutrophil levels rise with advancing disease).

- Submucosal bronchial gland enlargement and goblet cell metaplasia lead to excessive mucus production or chronic bronchitis [8].
- Cellular infiltrates consisting of neutrophils and lymphocytes within bronchial glands.
- Airway epithelial squamous metaplasia, ciliary dysfunction, hypertrophy of smooth muscle, and connective tissue [9].
- Peripheral Airways (Non-Cartilaginous Airways <2 mm Diameter)
- Enhanced populations of macrophages and T lymphocytes (CD8 > CD4).
- An increase in the quantities of B lymphocytes, lymphoid follicles, and fibroblasts has been observed [10].
- Limited presence of neutrophils or eosinophils.
- Early-stage bronchiolitis.
- Luminal and inflammatory exudates.
- Abnormal proliferation of goblet cells and squamous metaplasia in the peripheral airways.
- Peribronchial fibrosis and airway narrowing indicative of advancing disease.

DISCUSSION:

Lung Parenchyma (Respiratory Bronchioles and Alveoli)

a. Atherogenesis: Inflammation exists in the lungs, especially in the tiny airways, of all individuals who smoke. The typical defensive reaction to inhaled toxins is exacerbated in COPD, resulting in tissue damage, dysfunction of the defense mechanisms that mitigate such damage, and disruption of the repair processes [11]. Typically, the inflammatory and structural alterations in the airways escalate with the severity of the disease and last even post smoking cessation [12]. In addition to inflammation, two more mechanisms contribute to the development of COPD: a disparity between proteases and antiproteases, and an imbalance between oxidants and antioxidants (oxidative stress) in the lungs.

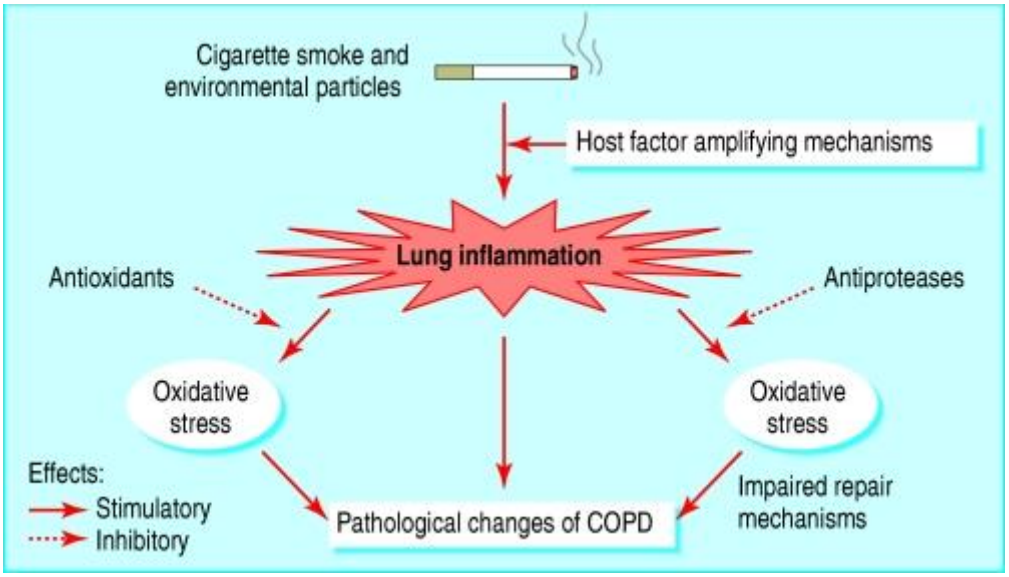

Figure 3: Pathogenesis of COPD

b. Cells Involved in Inflammation: COPD is marked by elevated levels of neutrophils, macrophages, and T lymphocytes, with a predominance of CD8 over CD4, in the pulmonary tissue [13].The severity of inflammation correlates with the level of airflow restriction [14]. These inflammatory cells secrete several cytokines and mediators that contribute to the illness process [15]. This inflammatory pattern significantly differs from that observed in patients with asthma.

c. Mediators of Inflammation: Numerous inflammatory mediators are elevated in COPD, including:

- Leucotriene B4, a chemoattractant for neutrophils and T lymphocytes, generated by macrophages, neutrophils, and epithelial cells [16].
- Chemotactic factors, including the CXC chemokines interleukin 8 and growth-related oncogene α, are synthesized by macrophages and epithelial cells [17].
- These recruit circulating cells and enhance pro-inflammatory responses.
- Pro-inflammatory cytokines including tumor necrosis factor α and interleukins 1β and 6.
- Growth factors, including transforming growth factor β, may induce fibrosis in the airways either directly or by the production of another cytokine, connective tissue growth factor [18].

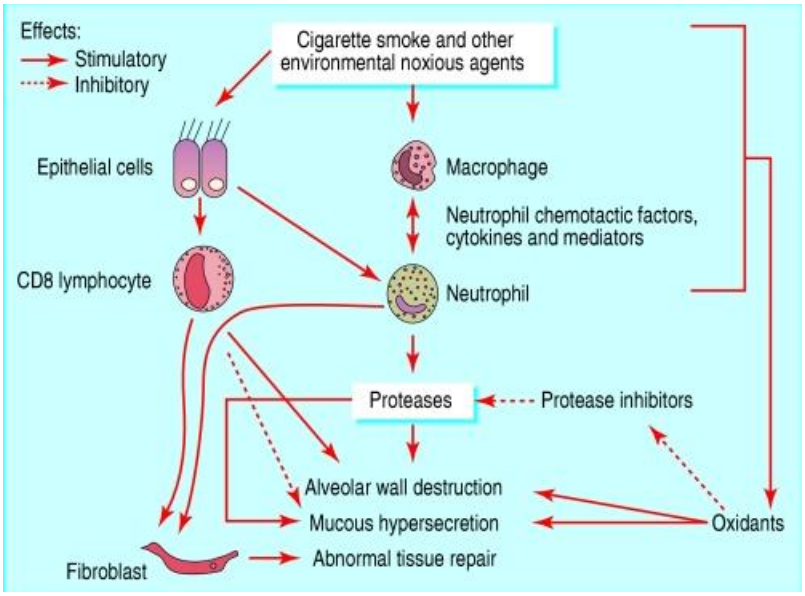

Figure 4: Inflammatory mechanisms in COPD

d. Inflammatory Cells and Mediators in Chronic Obstructive Pulmonary Disease:

- Neutrophils, which secrete proteases, are elevated in the sputum and distal airspaces of smokers; an additional increase is observed in COPD and correlates with disease severity [19].
- Macrophages, responsible for producing inflammatory mediators and proteases, are elevated in quantity within the airways, lung parenchyma, and bronchoalveolar lavage fluid [20].
- T lymphocytes (CD4 and CD8 cells) are elevated in the airways and lung parenchyma, accompanied by an enhanced CD8:CD4 ratio [21].
- The quantities of Th1 and Tc1 cells, which secrete interferon γ, also rise. CD8 lymphocytes can be cytotoxic and induce deterioration of alveolar walls [22].
- B lymphocytes are elevated in the peripheral airways and lymphoid follicles, maybe as a reaction to chronic airway infection [23].

Table 1: Proteases and antiproteases in COPD

Proteases	Antiproteases
Serine proteases	α_1 antitrypsin
Neutrophil elastase	
Cathepsin G	Secretory leucoprotease inhibitor

Protease 3	Elafin
Cysteine proteases	Cystatins
Cathepsins B, K, L, S	
Matrix metalloproteases (MMP-8, MMP-9, MMP-12)	Tissue inhibitors of MMP (TIMP1-4)

e. COPD Systemic Features:

i. Cachexia.

ii. Skeletal muscle degeneration and disuse atrophy.

iii. Elevated risk of cardiovascular disease (correlated with heightened levels of C-reactive protein) [24].

iv. Normochromic normocytic anemia [25].

v. Secondary polycythemia [26].

vi. Osteoporosis.

vii. Depression and anxiety disorders.

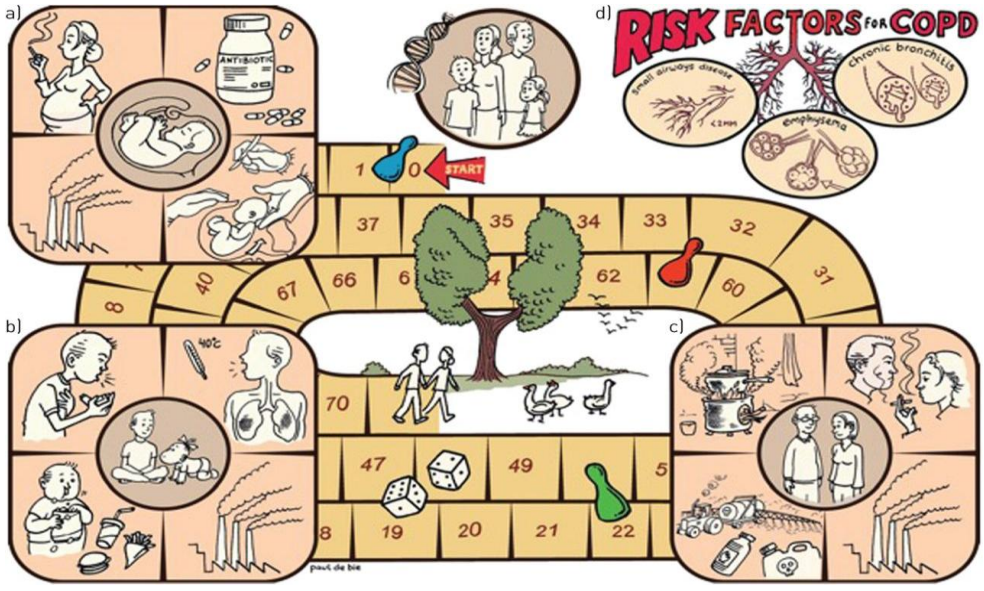

Figure 5: Visual depiction of the risk factors for chronic obstructive pulmonary disease (COPD) across various life stages

CONCLUSION:

COPD has traditionally been viewed as a condition predominantly affecting the elderly, with insufficient focus on its clinical and pathological characteristics in younger populations. Current evidence indicates that early COPD correlates with unfavorable clinical outcomes, highlighting the importance of early detection, diagnosis, and maintenance treatment, in conjunction with smoking cessation and exercise. Regular health exercises, including lung exercises, are essential for protection against dust particles and allergens following the COVID-19 pandemic.

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