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Original Research Article

A Clinical Study to Evaluate the Differences in Systemic Inflammatory Markers between COPD and Post Tubercular Obstructive Airway Disease

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Abstract:

Aim: This study aims to evaluate the difference in systemic inflammatory markers between COPD and post-tubercular obstructive airway disease.

Materials and Methods: This study was conducted at SRN Hospital, affiliated with MLN Medical College in Allahabad, and received ethical approval from the institutional ethics committee. The primary aim was to evaluate and compare the difference in systemic inflammatory markers between COPD and post-tubercular obstructive airway disease between two patient groups: those with chronic obstructive pulmonary disease (COPD) and those with post-tubercular obstructive airway disease (PTOAD). The methodology included a review of medical histories, physical exams, pulmonary function tests, alongside blood sample analyses for inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha). Additional evaluations of total lymphocyte count (TLC) and erythrocyte sedimentation rate (ESR) provided a comprehensive view of participants' inflammatory status.

Statistical Analysis and Results: Total 36 patients were diagnosed with Chronic Obstructive Pulmonary Disease (COPD), comprising 25 males (69.44%) and 11 females (30.55%). The remaining 32 participants had Post-Tuberculosis Obstructive Airway Disease (Post-TB OAD), with 19 males (59.37%) and 13 females (40.62%). Statistical analysis revealed no significant differences in the gender distribution between the two groups. The researchers measured C-reactive protein (CRP) levels as a marker of systemic inflammation. In the COPD cohort, 12 had elevated CRP levels and 24 were negative, whereas the Post-TB OAD group had 5 positive and 27 negative. No significant difference in CRP positivity rates was found ($z=1.6833$, $p=0.092$). They also evaluated the erythrocyte sedimentation rate (ESR), finding a mean ESR of 18.69 ± 7.02 mm/h in COPD and 19.75 ± 11.78 mm/h in Post-TB OAD. Again, no significant difference was noted ($t=0.454$, $p=0.65$). The total leukocyte count (TLC) showed a mean of 9358.33 ± 4305.50 cells/mm³ in COPD and 9315.62 ± 3498.48 cells/mm³ in Post-TB OAD, with no significant difference ($t=0.044$, $p=0.96$).

Conclusion: The findings showed no significant differences in serum parameters, including Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP), and Total Leukocyte Count (TLC), indicating a similar inflammatory response in both groups.

Keywords: Chronic Obstructive Pulmonary Disease (COPD), Post-Tubercular Obstructive Airway Disease, Inflammatory Markers.

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Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a complex and progressive respiratory condition that is characterised by an obstruction of airflow, leading to increasingly difficult breathing. This multifaceted disease includes two main components: chronic bronchitis and emphysema. Chronic bronchitis involves persistent inflammation of the airways, which results in the production of excess mucus and narrowing of the bronchial tubes. Emphysema, on the other hand, leads to the destruction of the air sacs (alveoli) in the lungs, significantly reducing their elasticity and impairing gas exchange.[1,2] The primary cause of COPD is prolonged exposure to harmful irritants, with cigarette smoking being the leading contributor, responsible for the majority of cases. Other risk factors include long-term exposure to air pollution, occupational dust, and chemicals, as well as frequent respiratory infections in childhood. A less common but noteworthy risk factor is a genetic

condition known as Alpha-1 antitrypsin deficiency, which can predispose individuals to lung damage. The symptoms of COPD typically develop gradually and can worsen over time.[3,4] Common manifestations include shortness of breath, especially during physical activities, a persistent cough that produces mucus, wheezing, a sensation of chest tightness, and an overwhelming sense of fatigue. These symptoms can significantly impact an individual's quality of life and may lead to increased isolation and emotional distress. Management of COPD is multifaceted and primarily focuses on alleviating symptoms and improving pulmonary function.[5,6] The most crucial step in treatment is smoking cessation, as quitting is known to significantly slow disease progression and improve outcomes. Inhaled medications, such as bronchodilators and corticosteroids, are commonly prescribed to help open the airways and reduce inflammation. Additionally, patients may benefit from pulmonary rehabilitation programs, which include supervised exercise, education on disease management, and nutritional support. For individuals with severe COPD, supplemental oxygen therapy may be necessary to ensure adequate oxygenation, especially during exertion or at rest. Post-Tubercular Obstructive Airway Disease (PT-OAD) represents another serious chronic respiratory condition that can develop in individuals who have been successfully treated for pulmonary tuberculosis.[7] PT-OAD is characterized by permanent airflow limitation and structural lung damage resulting from chronic inflammation, airway remodelling, and the development of fibrosis. The symptoms largely mirror those of COPD, including shortness of breath and a chronic cough. Research indicates that both COPD and PT-OAD are associated with heightened levels of pro-inflammatory cytokines, such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6).[8,9] These cytokines are often found in elevated concentrations in patients with PT-OAD, signifying ongoing inflammatory processes. Furthermore, acute-phase reactants such as C-reactive protein (CRP) and fibrinogen frequently rise during acute exacerbations in individuals with a history of tuberculosis, indicating a heightened inflammatory state. Novel indices derived from blood counts, like the Systemic Immune-Inflammation Index (SII) and the Neutrophil-to-Lymphocyte Ratio (NLR), have emerged as valuable tools in evaluating the severity of post-tubercular obstructive changes. These biomarkers provide important insights into the systemic inflammatory processes at play and can aid in assessing disease progression and treatment responses. Overall, the intertwined nature of these conditions highlights the necessity for comprehensive management strategies that address both lung health and the underlying inflammatory mechanisms.[10,11] This study aims to evaluate the difference in systemic inflammatory markers between COPD and post-tubercular obstructive airway disease.

Materials and Methods

This study was planned, abstracted and carried out at Department of Critical Care of SRN Hospital, which is affiliated with MLN Medical College, located in Allahabad. The investigation received ethical approval from the institutional ethics committee, ensuring that all procedures adhered to ethical standards for research involving human subjects. The participants in this study were adults aged between 40 and 75 years old, who presented to the outpatient department with symptoms of chronic breathlessness, a persistent cough, sputum production, and wheezing, which are common indicators of obstructive airway conditions. Stringent inclusion criteria were established to ensure that only individuals displaying confirmed airway obstruction were enrolled, as demonstrated by post-bronchodilator spirometry results. For the COPD cohort, patients typically had a history of significant exposure to risk factors, notably heavy smoking or exposure to biomass fuels, which are known to contribute to the development of the disease. In contrast, participants classified as PTOAD had a documented history of treated pulmonary tuberculosis, with the subsequent development of residual obstruction affecting their airways. Prior to enrollment, all participants were required to provide informed consent, affirming their understanding of the study procedures and their willingness to participate. The study also defined specific exclusion criteria to maintain the integrity of the research findings. Individuals who presented with active infections, including ongoing pulmonary tuberculosis, or those experiencing recent exacerbations of their COPD or PTOAD were excluded. Other respiratory conditions, such as asthma or bronchiectasis, as well as systemic inflammatory diseases and active malignancies, were also not considered for participation to isolate the effects of the primary diseases under investigation. The methodology employed in this study involved a comprehensive review of each participant's medical history, coupled with thorough physical examinations and pulmonary function tests. Based on their individual medical histories and spirometry results, patients were categorized into either the COPD or PTOAD group. To assess systemic inflammation, blood samples were meticulously collected from all participants. These samples were analyzed for various inflammatory markers, including C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha), utilizing standard laboratory assays tailored to accurately quantify these substances. An additional aim of the

study was to compare the levels of these inflammatory markers between the two patient groups, seeking to uncover any significant differences that could yield insights into the nature and extent of systemic inflammation present in each condition. Furthermore, other haematological parameters such as total lymphocyte count (TLC) and erythrocyte sedimentation rate (ESR) were also assessed in the enrolled patients to provide a more comprehensive evaluation of their inflammatory status and overall health profile. Through this detailed approach, the study sought to contribute valuable knowledge to the understanding of COPD and PTOAD and their associated inflammatory profiles.

Statistical Analysis: In this study, we conducted all statistical analyses using SPSS version 26.0 software. To assess the significance of our findings, we employed the chi-square test, a robust statistical method that is especially useful for comparing the differences in proportions among different groups. This approach allowed us to rigorously evaluate the relationships within our data, ensuring that our conclusions were well-supported by the evidence.

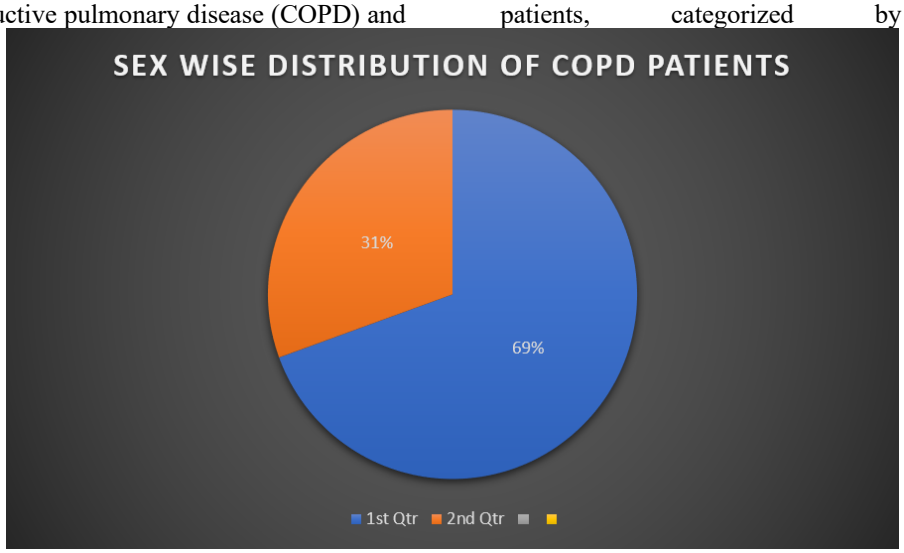
Results

The study involved a comprehensive analysis of a cohort consisting of a total of 68 patients who were enrolled within the timeframe of October 2018 to September 2019. To ensure that the sample was uniform and representative, the researchers meticulously adhered to established inclusion and exclusion criteria. Among the participants, 36 individuals were diagnosed with Chronic Obstructive Pulmonary Disease (COPD). A detailed demographic breakdown of this subgroup showed that 25 males were included, which constituted approximately 69.44% of the COPD patients, while the female population accounted for 11 participants, representing 30.55%. This differential distribution of sex provided insight into the demographics of COPD patients within this sample. Conversely, the remaining 32 participants were diagnosed with Post-Tuberculosis Obstructive Airway Disease (Post-TB OAD). In this group, the male representation consisted of 19 individuals (59.37%), and there were 13 females (40.62%). To evaluate the potential for biases in sex distribution, statistical analyses were performed, comparing the male-to-female ratios between the two diagnostic categories. The results indicated no significant differences in these ratios, suggesting that the gender distribution was relatively balanced across both the COPD and Post-TB OAD patient groups. To explore the aspect of systemic inflammation, the researchers specifically measured the levels of C-reactive protein (CRP), which is an invaluable biomarker commonly associated with inflammatory processes within the body. Within the COPD cohort, 12 individuals demonstrated elevated CRP levels, while 24 were classified as CRP negative. On the other hand, the Post-TB OAD group showed that only 5 patients had positive CRP results, whereas a notable 27 tested negative. The comparison of CRP positivity rates between the COPD and Post-TB OAD groups yielded no statistically significant difference ($z=1.6833$, $p=0.092$). This result implies that the underlying systemic inflammation, as evidenced by CRP levels, was quite similar in both groups of patients. Following the CRP analyses, the erythrocyte sedimentation rate (ESR)—another key marker for inflammation—was evaluated. The COPD group presented a mean ESR value of 18.69 ± 7.02 mm/h, suggesting a mild degree of inflammation. In comparison, the Post-TB OAD cohort exhibited a slightly elevated mean ESR of 19.75 ± 11.78 mm/h. However, statistical comparisons of the mean ESR values indicated no significant difference ($t=0.454$, $p=0.65$), further cementing the conclusion that systemic inflammation levels, as measured by ESR, did not vary significantly between the two patient categories. In addition to CRP and ESR, the total leukocyte count (TLC) was also assessed to obtain insights into the overall white blood cell response in both COPD and Post-TB OAD groups. The COPD patients showed a mean TLC of 9358.33 ± 4305.50 cells/mm^3, while the Post-TB OAD group exhibited a mean TLC of 9315.62 ± 3498.48 cells/mm^3. Subsequent statistical analysis of the TLC values revealed no statistically significant difference ($t=0.044$, $p=0.96$), which further supports the notion that systemic inflammation levels, as reflected through various parameters including CRP, ESR, and TLC, were comparable between the two conditions. In conclusion, the findings from this rigorous study strongly indicate that there are no significant differences in systemic inflammation levels between patients diagnosed with COPD and those suffering from Post-TB OAD. The results underscore a need for further research into the inflammatory processes underlying these respiratory conditions to deepen our understanding of their pathophysiology and explore potential therapeutic avenues.

Table 1: Sex wise distribution of patients

	COPD	Percentage	Post-TBOAD	Percentage
Male	25	69.44	19	**59.37**
Female	11	30.55	13	**40.62**
Total	**36**	**-**	**32**	**-**

Graph 1 & 2: Analysis of the distribution of chronic obstructive pulmonary disease (COPD) and post-tuberculosis obstructive airway disease (OAD) patients, categorized by gender



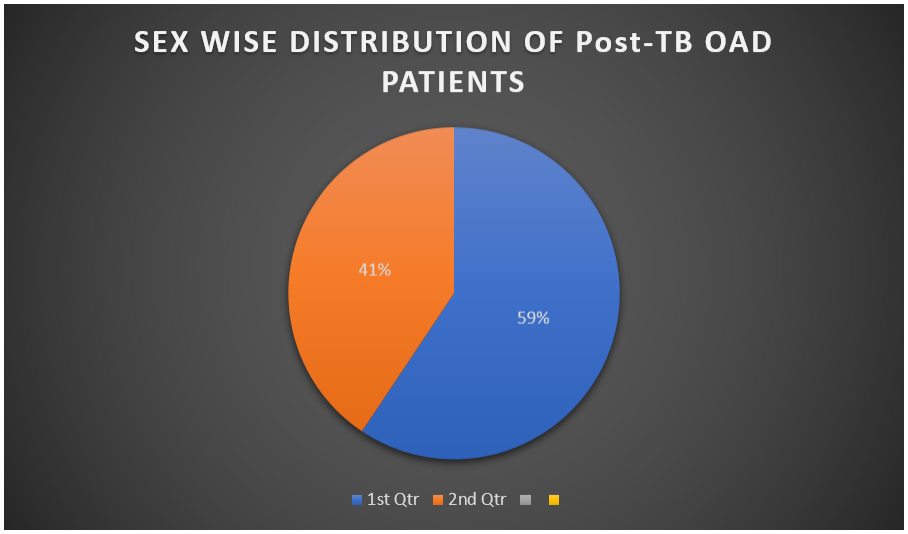


Table 2: Comparison of CRP parameter of COPD & Post-TBOAD patients

Patients characteristics	CRP		
	COPD (no. of patients)	Post-TBOAD (no. of patients)	Statistical analysis (z-test)
Total	36	32	z=1.6833,p=0.092
Positive	12	5	
Negative	24	27	
Percentage	33.33%	15.00%	

Table 3: Comparison of ESR parameter of COPD & Post-TBOAD patients

S. NO.	ESR(mm/hr)		
	COPD (Mean±SD)	Post-TBOAD (Mean±SD)	Statistical analysis (unpaired t-test)
1	18.69±7.02	19.75±11.78	t=0.454,p=0.65

Table 4: Comparison of TLC parameter of COPD & Post-TBOAD patients

S. NO.	TLC(/mm^3)		
	COPD (Mean±SD)	Post-TBOAD (Mean±SD)	Statistical analysis (unpaired t-test)
1	9358.33±4305.50	9315.62±3498.48	t=0.044,p=0.96

Discussion

Riley CM et al reported in their study that chronic Obstructive Pulmonary Disease (COPD) is a progressive and debilitating respiratory condition that profoundly affects airflow, resulting in significant challenges when breathing. This disease is primarily characterized by two key components: emphysema and chronic bronchitis. Emphysema is marked by the destruction of the lung's elastic air sacs, known as alveoli. This damage hinders the lungs' ability to transfer oxygen into the bloodstream and remove carbon dioxide effectively, leading to symptoms such as shortness of breath, chronic cough, and the production of sputum. In chronic bronchitis, there is persistent inflammation of the bronchial tubes, which carry air to and from the lungs.[12,13] Rabe KF et al reviewed in their study that this inflammation results in mucus overproduction, which clogs the airways and further narrows them, creating breathing difficulties. Together, these components contribute to irreversible damage to lung tissue that typically deteriorates progressively over time. While there is currently no cure for COPD, a variety of treatment strategies have been developed that can significantly enhance patients' quality of life and slow the disease's progression.[14,15] Debrus C et al reported in their study that a crucial intervention for anyone suffering from COPD is smoking cessation, as ongoing tobacco use is the primary cause of exacerbations and the overall advancement of the disease. Smoking cessation not only helps halt further lung damage but can also lead to improvements in pulmonary function over time. Pharmacological treatments play a critical role in managing COPD. These often include inhaled bronchodilators, which work to relax the muscles surrounding the airways, thereby improving airflow and reducing shortness of breath.[16,17] Tárrega J et al reported in their study that inhaled corticosteroids are also commonly prescribed to mitigate inflammation in the airways, helping to decrease the frequency and severity of exacerbations. Beyond medication, patients often find significant benefit in participating in pulmonary rehabilitation programs. These comprehensive programs consist of tailored treatments that incorporate physical exercise, breathing techniques, and nutritional counselling, all aimed at enhancing pulmonary function and promoting overall wellness. For those with advanced COPD, oxygen therapy becomes a vital component of their treatment regimen, improving oxygen delivery to the body and relieving breathlessness during daily activities. Preventive strategies are equally important in the management of COPD. For instance, it is highly recommended that patients receive annual vaccinations for influenza and pneumococcal pneumonia. These vaccinations are essential for safeguarding lung health, as respiratory infections can exacerbate existing COPD and lead to severe complications, including hospitalisations. Another significant condition related to lung health is Post-Tubercular Obstructive Airway Disease (PTOAD).[18] Lodha R et al reviewed in their study that this chronic respiratory disorder can persist long after a patient has clinically recovered from pulmonary tuberculosis. Although the causative agent, Mycobacterium tuberculosis, may have been eradicated, the aftermath can include lingering immune responses and chronic inflammation, which collectively contribute to lasting damage to lung tissue. Research indicates that patients with PTOAD frequently exhibit elevated levels of pro-inflammatory cytokines, including interleukin-6 (IL-6), interferon-gamma (IFN-γ), and soluble interleukin-2 receptor (sIL-2R).[19] Reséndiz-Hernández JM et al showed in their study that these biomarkers have been associated with an accelerated decline in pulmonary function and a higher frequency of exacerbations. Furthermore, composite haematological indices such as the Systemic Inflammation Response Index (SIRI) and the Systemic Immune-Information Index (SII) have emerged as valuable tools for differentiating PTOAD from COPD that arises primarily from smoking.[20] Sinden NJ et al reported in their study that while both PTOAD and smoking-induced COPD display elevated levels of C-Reactive Protein (CRP) and an increased Erythrocyte Sedimentation Rate (ESR) during periods of exacerbation, it is noteworthy that stable patients with PTOAD often show consistently higher baseline CRP levels compared to their COPD counterparts. This observation suggests that chronic low-grade systemic inflammation is a pivotal factor in PTOAD, potentially leading to structural alterations in the lungs. Such changes may include bronchiectasis (characterised by abnormal dilation of the airways) and cavitation (the formation of hollow spaces within lung tissue), ultimately exacerbating respiratory function decline and worsening health outcomes. Understanding the complex mechanisms underlying both COPD and PTOAD underscores the importance of comprehensive management strategies and highlights the need for ongoing research into effective treatments for these debilitating conditions.[21,22]

Conclusion

The study to assess the differences in systemic inflammatory markers between patients diagnosed with Chronic Obstructive Pulmonary Disease (COPD) and those suffering from Post-Tubercular Obstructive Airway Disease. Through thorough analysis, the findings revealed that there were no statistically significant differences in several important serum parameters measured. Notably, the

serum Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP), and Total Leukocyte Count (TLC) exhibited comparable levels across both patient groups, suggesting a similar inflammatory response in these conditions.

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