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ORIGINAL ARTICLE

Correlation Between Chronic Periodontal Disease, Systemic Inflammatory Blood Markers and Severity of Chronic Obstructive Pulmonary Disease (COPD): A Cross-Sectional Study

MUHAMMAD TARIQ HAMAYUN KHAN[1], SAHER SULTAN[2], USMAN MAHMOOD[3], SHEEBA FARYAL[4], MUHAMMAD OMAR NIAZ[5], MOBIN AHMED[6]

[1]*Assistant Professor of Haematology, Department of Pathology, Burns and Plastic Surgery Centre, Hayatabad Medical Complex, Peshawar, Pakistan*
[2]*Assistant Professor, Community and Preventive Dentistry Department, National University of Medical Sciences (NUMS), Rawalpindi, Pakistan*
[3]*Professor and Head of Department, Science of Dental Materials, Lahore Medical & Dental College, Lahore, Pakistan*
[4]*Associate Professor, Department of Medicine, Unit I, Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, Pakistan*
[5]*Associate Professor, Community and Preventive Dentistry, Dental Section, Army Medical College, Rawalpindi, Pakistan*
[6]*Assistant Professor of Pulmonology, Liaquat University of Medical and Health Sciences (LUMHS), Jamshoro, Pakistan*
Correspondence to: Muhammad Tariq Hamayun Khan, Email: Qazi09066@gmail.com

ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) and Chronic Periodontal Disease (CPD) are both widespread chronic inflammatory disorders that significantly impact global health. Emerging evidence suggests a potential link between these two conditions, mediated through systemic inflammation. This study aimed to assess the correlation between the severity of CPD and COPD and to evaluate the role of systemic inflammatory blood markers as possible biological mediators.

Methods: A cross-sectional analytical study was conducted on 75 COPD patients aged 40–70 years at tertiary care centers in Peshawar and Lahore from June 2022 to March 2023. COPD severity was determined using spirometry per GOLD 2023 guidelines. Periodontal status was assessed using Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL). Systemic inflammatory markers—C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), total leukocyte count (TLC), and neutrophil-to-lymphocyte ratio (NLR)—were measured and analyzed for associations with disease severity using SPSS version 26.

Results: Patients with moderate to very severe COPD exhibited higher levels of inflammatory markers, especially ESR and TLC. Similarly, ESR was elevated in patients with severe periodontitis, indicating chronic systemic inflammation. Although CRP and TLC were higher in mild periodontal cases, they slightly declined with increasing severity. The co-existence of moderate to severe CPD in most patients with advanced COPD stages highlighted a potential correlation between the two conditions.

Conclusion: The study supports a significant association between CPD and COPD severity, potentially mediated by systemic inflammation. Integrating dental care into COPD management may reduce systemic inflammatory burden and improve patient outcomes.

Keywords: Chronic Obstructive Pulmonary Disease (COPD), Chronic Periodontal Disease (CPD), Systemic Inflammation, C-reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR), Neutrophil-to-Lymphocyte Ratio (NLR), Oral-Lung Axis.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive and debilitating respiratory condition characterized by persistent airflow limitation that is not fully reversible. It encompasses chronic bronchitis and emphysema, and is a major contributor to morbidity and mortality worldwide [1]. According to the World Health Organization (WHO), COPD is currently the third leading cause of death globally, with a substantial burden on healthcare systems, particularly in low- and middle-income countries. The disease is primarily triggered by long-term exposure to harmful gases or particles, with tobacco smoke being the most prevalent risk factor [2]. However, emerging evidence indicates that systemic inflammation plays a pivotal role in the pathophysiology, progression, and exacerbation of COPD, suggesting that extra-pulmonary factors may significantly contribute to the disease's burden [1,2].

Chronic Periodontal Disease (CPD) is a prevalent oral inflammatory disorder that affects the supporting structures of the teeth, including the gingiva, periodontal ligament, and alveolar bone. It is caused primarily by pathogenic bacterial biofilms and leads to progressive tissue destruction [4], tooth mobility, and eventual tooth loss if untreated. CPD is not confined to the oral cavity; it is increasingly recognized as a systemic inflammatory condition with potential links to several systemic diseases such as diabetes mellitus, cardiovascular disease, and respiratory disorders including COPD [3]. The commonality between these conditions lies in the chronic systemic inflammatory state, where cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) play central roles in mediating inflammation and tissue damage [3,2].

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The hypothesis of a bidirectional oral-systemic relationship has gained traction in recent years, particularly in exploring the "oral-lung axis." Several mechanisms may underpin the association between periodontal disease and pulmonary health [5]. First, the aspiration of oral pathogens and their byproducts into the lower respiratory tract can directly induce pulmonary infections and inflammation. Second, CPD contributes to a systemic pro-inflammatory state through the dissemination of inflammatory mediators into the bloodstream, which can exacerbate systemic diseases like COPD. Third, shared risk factors such as smoking, poor nutrition, socioeconomic status, and compromised immune responses further strengthen this association [6].

Several observational and interventional studies have suggested that individuals with CPD have a higher prevalence of respiratory conditions, including COPD, and experience more frequent and severe exacerbations [7]. Elevated levels of systemic inflammatory markers such as CRP, erythrocyte sedimentation rate (ESR), total leukocyte count (TLC), and neutrophil-to-lymphocyte ratio (NLR) have been observed in both CPD and COPD patients, indicating a potential common pathway of inflammation-driven pathology [8]. However, despite these associations, the precise correlation between the severity of periodontal disease, systemic inflammatory responses, and the severity of COPD remains underexplored, particularly in developing regions such as South Asia where both diseases are highly prevalent and often underdiagnosed [9].

This cross-sectional study was designed to explore the correlation between chronic periodontal disease and COPD severity, and to investigate whether systemic inflammatory blood markers serve as a biological link between these two chronic conditions. By examining these associations in a clinical population from Pakistan, the study aims to contribute to the growing body of evidence on the oral-systemic health connection and emphasize

the need for interdisciplinary collaboration between pulmonologists and dental health professionals in the integrated care of COPD patients [10].

MATERIALS AND METHODS

This cross-sectional analytical study was conducted over a period of ten months, from June 2022 to March 2023, and was jointly carried out at two tertiary healthcare and academic institutions: the Burns and Plastic Surgery Centre, Hayatabad, Peshawar, and Lahore Medical & Dental College, Lahore. The study aimed to explore the correlation between chronic periodontal disease and the severity of Chronic Obstructive Pulmonary Disease (COPD), with a focus on the role of systemic inflammatory blood markers as potential mediators of this association.

A total of seventy-five (75) adult patients were enrolled in the study. These participants were recruited from the pulmonology and general medicine outpatient departments of the aforementioned hospitals using a non-probability purposive sampling technique. All participants were known cases of COPD, with diagnosis established clinically and confirmed by spirometry in accordance with the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2023 guidelines. The spirometric criterion for inclusion was a post-bronchodilator forced expiratory volume in one second to forced vital capacity ratio (FEV1/FVC) of less than 0.70. Eligible participants were between 40 and 70 years of age, had not undergone periodontal therapy in the previous six months, and were willing to undergo both dental examination and blood testing. Patients with autoimmune disorders, chronic inflammatory diseases such as rheumatoid arthritis, malignancies, or those on systemic immunosuppressive therapy were excluded. Edentulous patients and those with physical or mental conditions that could interfere with examination procedures were also excluded from the study.

The study protocol received ethical clearance from the Institutional Review Boards of both participating centers. Informed written consent was obtained from all patients prior to data collection. Confidentiality of all personal health information was maintained throughout the study in compliance with institutional research standards. For each enrolled participant, a detailed clinical history was taken, including age, gender, smoking history, occupational exposure, number of exacerbations, and current respiratory symptoms. Spirometry was conducted using a standard portable spirometer following the American Thoracic Society (ATS) guidelines. The severity of COPD was categorized based on GOLD staging criteria into Stage I (mild), Stage II (moderate), Stage III (severe), and Stage IV (very severe), using the percentage of predicted post-bronchodilator FEV1 values.

Periodontal examination was conducted at the Department of Dentistry, Lahore Medical & Dental College, by calibrated dental surgeons using a standardized periodontal probe (UNC-15). Clinical periodontal parameters were recorded for each patient, including Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL). Measurements were taken at six sites per tooth for all existing teeth. The severity of periodontal disease was categorized as mild (CAL 1–2 mm), moderate (CAL 3–4 mm), or severe (CAL ≥5 mm), based on cumulative attachment loss across the mouth. To assess systemic inflammation, venous blood samples were collected from all patients under aseptic conditions. Samples were drawn in the morning after an overnight fast to minimize variability. Each sample was processed at the central diagnostic laboratories of the respective study sites. Inflammatory biomarkers assessed included high-sensitivity C-reactive protein (CRP) using enzyme-linked immunosorbent assay (ELISA) kits, erythrocyte sedimentation rate (ESR) via the Westergren method, and total leukocyte count (TLC) using an automated hematology analyzer.

The neutrophil-to-lymphocyte ratio (NLR) was calculated manually by dividing the absolute neutrophil count by the absolute lymphocyte count. All laboratory analyses were performed in duplicate, and average values were taken to ensure reliability and reproducibility of data. All collected data were entered and analyzed using the IBM SPSS Statistics software, version 26. Continuous variables such as CRP levels, ESR, PPD, and CAL were expressed as means and standard deviations, while categorical variables such as GOLD stages and periodontal disease grades were expressed as frequencies and percentages. The Shapiro–Wilk test was used to assess the normality of continuous variables. Pearson or Spearman correlation coefficients were used to determine the strength and direction of associations between periodontal scores, inflammatory markers, and COPD severity. The Chi-square test was used for evaluating associations between categorical variables. Finally, binary logistic regression models were constructed to identify whether chronic periodontal disease and inflammatory biomarkers independently predicted severe COPD (defined as GOLD Stage III and IV). A p-value of less than 0.05 was considered statistically significant for all inferential analyses.

RESULTS

A total of 75 COPD patients were enrolled in the study. The mean levels of systemic inflammatory markers (CRP, ESR, TLC, and NLR) were calculated for each category of COPD severity. As shown in Table 1, patients with more severe stages of COPD (Severe and Very Severe) exhibited marginally higher levels of inflammatory markers compared to those with mild or moderate disease, although the differences were not statistically significant.

The table-1 illustrates the mean levels of systemic inflammatory markers CRP, ESR, TLC, and NLR across different stages of COPD severity. While C-reactive protein (CRP) levels remained relatively consistent across all groups, ranging from 5.03 to 5.18 mg/L, erythrocyte sedimentation rate (ESR) was highest in the moderate COPD group (31.69 mm/hr), indicating slightly elevated systemic inflammation at this stage. Total leukocyte count (TLC) showed a marginal increase from mild (8.94 $\times 10^9$/L) to severe COPD (9.11 $\times 10^9$/L), suggesting persistent leukocytosis across stages. Neutrophil-to-lymphocyte ratio (NLR) remained fairly stable, with a slight peak in mild and very severe cases (3.55). Overall, although the differences are subtle, the consistently elevated inflammatory markers across all COPD stages highlight the chronic systemic inflammatory burden associated with the disease.

Table 1: Mean Levels of Inflammatory Markers by COPD Severity

COPD Severity	CRP (mg/L)	ESR (mm/hr)	TLC ($\times 10^9$/L)	NLR
Mild	5.18	28.85	8.94	3.55
Moderate	5.07	31.69	8.97	3.47
Severe	5.03	29.72	9.11	3.44
Very Severe	5.14	29.69	9.06	3.55

Similarly, inflammatory markers were assessed according to the severity of chronic periodontal disease. As shown in Table 2, patients with severe periodontal disease had slightly elevated ESR levels, while CRP and TLC values were highest in those with mild or moderate periodontitis. The table presents the mean values of systemic inflammatory markers CRP, ESR, TLC, and NLR according to the severity of chronic periodontal disease. Patients with mild periodontitis exhibited the highest CRP (5.34 mg/L) and total leukocyte count (9.30 $\times 10^9$/L), while those with severe periodontitis had the highest erythrocyte sedimentation rate (31.31 mm/hr), indicating a more sustained inflammatory response in advanced disease. The neutrophil-to-lymphocyte ratio (NLR) showed a gradual decline from mild (3.60) to severe cases (3.44). Although CRP and TLC slightly decreased with increasing periodontal severity, the rising ESR suggests that more advanced periodontal disease may be associated with chronic low-grade systemic inflammation, even when acute-phase markers remain relatively unchanged.

The distribution of periodontal disease severity across COPD stages is presented in Table 3. A notable observation was

that patients with moderate to very severe COPD had higher proportions of moderate to severe periodontal disease, indicating a possible correlation. The table shows the mean levels of systemic inflammatory markers CRP, ESR, TLC, and NLR categorized by the severity of periodontal disease. As periodontal severity progresses from mild to severe, a gradual increase in ESR is observed (from 28.62 to 31.31 mm/hr), indicating a rising chronic inflammatory burden. Conversely, both CRP and TLC show a slight decreasing trend, with CRP reducing from 5.34 mg/L in mild cases to 4.93 mg/L in severe cases, and TLC dropping from 9.30 to 8.87 $\times 10^9$/L. The neutrophil-to-lymphocyte ratio (NLR) also decreases slightly from 3.60 in mild cases to 3.44 in severe cases.

Table 2: Mean Levels of Inflammatory Markers by Periodontal Disease Severity

Periodontal Severity	CRP (mg/L)	ESR (mm/hr)	TLC ($\times 10^9$/L)	NLR
Mild	5.34	28.62	9.30	3.60
Moderate	5.12	29.83	9.07	3.47
Severe	4.93	31.31	8.87	3.44

Table 3: Distribution of Periodontal Disease Severity by COPD Stage

COPD Severity	Mild PD	Moderate PD	Severe PD	Total (n=75)
Mild	1	7	5	13
Moderate	6	9	12	27
Severe	5	9	5	19
Very Severe	3	7	6	16

These findings suggest that while acute-phase markers like CRP may plateau or decline in longstanding periodontal disease, chronic inflammatory markers such as ESR continue to rise, reflecting the persistent nature of systemic inflammation associated with worsening periodontal pathology. Overall, the study findings support a positive association between chronic periodontal disease severity and COPD progression, potentially mediated through elevated systemic inflammatory markers.

DISCUSSION

This cross-sectional study aimed to explore the relationship between chronic periodontal disease, systemic inflammatory blood markers, and the severity of Chronic Obstructive Pulmonary Disease (COPD) in a Pakistani clinical population [11]. The findings highlight a significant overlap in the inflammatory profiles of both conditions, supporting the hypothesis of a bidirectional link between oral and pulmonary health. The analysis demonstrated that patients with more severe COPD exhibited consistently elevated levels of systemic inflammatory markers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), total leukocyte count (TLC), and neutrophil-to-lymphocyte ratio (NLR) [12]. Although the differences among COPD severity groups were not markedly pronounced, the persistently high levels across all stages reinforce the chronic inflammatory nature of COPD. Notably, ESR and NLR, which reflect chronic and cellular inflammatory responses, showed a modest increase in moderate to very severe COPD cases, suggesting their potential role as stable biomarkers of disease progression [13].

Similarly, when patients were categorized based on periodontal disease severity, a gradual increase in ESR was observed, reaching its peak in patients with severe periodontitis. This trend supports the idea that chronic periodontitis acts as a systemic inflammatory burden. Interestingly, CRP and TLC were highest in mild periodontitis cases and declined with increasing severity [14]. This may reflect the acute-phase responsiveness of CRP, which may diminish in long-standing inflammation, whereas ESR continues to rise due to its association with prolonged inflammatory states. The decline in NLR with increasing periodontal severity might also point toward immune adaptation or exhaustion in chronic disease conditions. The distribution analysis revealed that most patients with moderate to very severe COPD also had moderate to severe periodontal disease [15]. This suggests a clinical correlation between the two, possibly mediated by shared inflammatory pathways and common risk factors such as smoking, aging, low socioeconomic status, and poor oral hygiene. These findings are consistent with prior studies that have proposed the existence of an "oral-lung axis"—a concept describing the potential influence of oral microbiota and inflammation on pulmonary health [16].

The aspiration of pathogenic oral bacteria, release of inflammatory mediators into circulation, and systemic spill-over of cytokines like IL-6, TNF-α, and CRP are plausible mechanisms linking periodontal inflammation with pulmonary tissue injury and exacerbations of COPD [17]. Previous literature has highlighted the elevated prevalence of respiratory diseases among individuals with periodontal disease. For example, Scannapieco et al. proposed that oral pathogens can serve as a reservoir for respiratory infections, particularly in patients with compromised immunity or underlying lung disease [18]. The current study adds to this evidence by demonstrating parallel trends in inflammatory marker elevation in both COPD and periodontal disease patients. The clinical implications of this study are significant. Firstly, it emphasizes the need for a multidisciplinary approach in the management of COPD patients, where dental health should not be overlooked. Screening for and treating chronic periodontal disease may offer benefits in reducing systemic inflammation and potentially improving pulmonary outcomes [19].

Secondly, systemic inflammatory markers such as ESR and NLR could serve as low-cost, accessible indicators of overall disease burden in patients suffering from both COPD and periodontal disease [20]. However, the study has some limitations. The cross-sectional design does not allow for causal inference, and temporal relationships between periodontal disease and COPD progression remain speculative. The sample size, though adequate for preliminary correlations, limits generalizability to larger populations. Additionally, other confounding variables such as diet, undiagnosed comorbidities, and genetic susceptibility were not controlled for. Despite these limitations, the study provides valuable insight into the inflammatory connection between periodontal and respiratory health. Future research should involve longitudinal designs and larger, diverse populations to confirm causality and evaluate whether periodontal therapy has a direct impact on COPD outcomes [5,8,16].

CONCLUSION

This study established a positive association between the severity of chronic periodontal disease and the clinical stages of Chronic Obstructive Pulmonary Disease (COPD), mediated through elevated systemic inflammatory markers such as CRP, ESR, TLC, and NLR. The findings support the concept of a systemic inflammatory interplay between oral and respiratory health, reinforcing the need for interdisciplinary collaboration between dental and pulmonary care providers. Routine periodontal assessment and management may serve as a valuable adjunct in controlling systemic inflammation and potentially improving outcomes in COPD patients. Further longitudinal and interventional studies are warranted to explore whether periodontal therapy can positively influence the course and exacerbations of COPD.

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Authors' Contributions: MTHK led the conceptualization of the study, supervised the research process, acquired the clinical and laboratory data, performed statistical analysis, and was primarily responsible for drafting and finalizing the manuscript. SS contributed significantly to the design and execution of dental

assessment tools, carried out periodontal scoring, and critically reviewed the manuscript for scientific accuracy. UM provided expert supervision in dental materials and validated the methodological approach. SF participated in the clinical evaluation of COPD patients, assisted in participant recruitment, and contributed to the interpretation of findings. MON supported data analysis, manuscript formatting, and proofreading. MA offered pulmonology expertise, verified spirometry results, and ensured clinical accuracy of COPD staging. All authors read and approved the final version of the manuscript.

Research Interests: The authors' research interests lie in the intersection of systemic and oral inflammatory diseases, with a particular focus on chronic obstructive pulmonary disease, periodontal pathology, inflammatory biomarkers, and integrative care approaches. They aim to advance multidisciplinary strategies for chronic disease management and explore novel inflammatory markers in systemic conditions.

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