Original Article

Association between Periodontal Disease and Chronic Obstructive Pulmonary Disease: A Case–Control Study

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Abstract

Background: It has been recognized that oral infections, especially periodontal diseases, may affect the course and pathogenesis of a number of systemic diseases. The present study is done with an aim to determine the association between chronic obstructive pulmonary disease (COPD) and periodontal disease. **Materials and Methods:** The study included 130 individuals consisting of 65 patients (case group) having COPD and 65 individuals as controls. Individuals in the case group were well-functioning and ambulatory patients having COPD as determined by their history and by performing pulmonary function test, who were then graded into mild, moderate, severe, and very severe. Periodontal status was evaluated by the following indices: simplified oral hygiene index (OHI-S), plaque index (PI), gingival index (GI), pocket probing depth (PPD), and clinical attachment level (CAL). **Results:** Individuals in the case group had significantly higher OHI-S, PI, GI, PPD, and CAL ($P < 0.0001$) compared with the control group. A significant positive relationship was observed between COPD subgroup P values and PI, thus indicating a trend in which severity of lung obstruction increased as these periodontal indices worsened. **Conclusion:** The patients with COPD showed poor oral hygiene and higher prevalence of periodontal disease. The lack of awareness and negligence toward oral health care was noted, which increased as the severity of COPD increased. The dental community's awareness of poor health within this population should be elevated. Prevention and treatment of periodontal disease could be included in planned intervention campaigns designed to help patients with COPD.

Keywords: Chronic obstructive pulmonary disease, periodontal diseases, respiratory diseases, systemic diseases

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Introduction

In the past few years, there has been accumulating evidence that suggests an exquisite association between periodontal disease and chronic obstructive pulmonary disease (COPD). Scannapieco *et al*.[1] were among the first researchers to publish their studies suggesting a potential association between poor oral health and COPD. Studies have found that both periodontal disease and COPD have remarkably similar pathogenic mechanism. Both the diseases are chronic inflammatory diseases with common risk factors such as smoking, pathogenic bacteria (*e.g., Porphyromonas gingivalis*), genetic factors, and socioeconomic factors. It has been suggested that dental plaque may serve as a reservoir for respiratory pathogen.[2] Bacteria that colonize the supra- or subgingival dental plaque are shed into the saliva. These are pathogenic bacteria that either can be those associated with periodontal disease (*P. gingivalis, Fusobacterium nucleatum)* or can be respiratory pathogens (*Pseudomonas aeruginosa* and *Klebsiella pneumoniae*). The saliva is aspirated into the lower respiratory tract (bronchus), where an infection can ensue. Cytokines from periodontal tissues can enter the saliva from the gingival crevicular fluid (GCF) or can be aspirated to stimulate local inflammatory process that contributes to the initiation and/or progression of infection in the lung.[3] Hence, it may be possible that periodontal disease activity may contribute to progression of COPD.

The causal association between periodontal health status and risk of COPD is biologically plausible, but remains speculative. In the light of the above-mentioned aspects of the relationship

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between periodontal disease and COPD and the paucity of sufficient literature in this regard, the present study was conducted to determine the association between periodontal disease and COPD.

Materials and Methods

COPD is a chronic respiratory disorder that progresses slowly and is characterized by an obstructive ventilatory pattern, which is rarely reversible, very often related to tobacco smoking and which can lead to chronic respiratory failure. This definition covers, in reality, a number of entities, as follows:[4]

1. Chronic bronchitis, with an obstructive ventilator pattern that is defined by the existence of chronic bronchitis with permanent obstruction of airways (forced expiratory volume in 1 s [FEV1] to forced vital capacity ratio, 70%)
2. Chronic respiratory failure, which is defined by the existence of chronic obstructive bronchitis with hypoxemia
3. Emphysema, which is defined at the anatomical level by destruction of the walls of the alveolar sacs/ducts beyond the terminal bronchiole with an abnormal increase in size of distal airways.

A total of 130 patients both male and female with age and gender matched were divided into two groups: COPD (case group) 65 subjects and non-COPD (control group) 65 subjects. Clinical examination was carried out on patients reporting to the Outpatient Department of Pulmonology, School of Medical Sciences and Research, Sharda University, diagnosed as COPD by a physician. Clinical examination was carried out on patients with non-COPD (control group) reporting to the Outpatient Department of Periodontology, School of Dental Sciences, Sharda University, Greater Noida. The nature of the study was explained to the patients following which verbal and written consent was obtained. Verbal communication was first done to the patient and patient's doubts regarding the study were clarified and after obtaining the verbal consent, written consent was taken for the purpose of record. Ethical committee approval was obtained.

Inclusion criteria for the COPD group (case) included well-functioning and ambulatory patients, patients of COPD as diagnosed by the physician with ≥30 years of age, patients with ≥20 remaining teeth in oral cavity, patients should be willing to give informed consent, and comply with all study requirements. For the non-COPD group (control), criteria included systemically healthy patients as diagnosed by the physician based on their thorough medical history with ≥30 years of age.

Exclusion criteria for the COPD group (case) included patients with inability to perform pulmonary function test, patients with acute exacerbation of COPD with previous lung volume reduction surgery, lung transplantation, or pneumonectomy, history of periodontal treatment in the past 6 months, patients with systemic diseases or conditions that can modify periodontal disease (e.g.,-Types 1 and 2 diabetes mellitus), and patients using any medication known to influence periodontal tissue. Patients consuming nicotine in any other form except cigarettes were excluded. Verbal and written consent was obtained from the above-mentioned patients. Demographic details including age, sex, address, occupation, thorough medical history, dental history, and smoking history were recorded for both the groups. Smokers were stratified according to their smoking status and lifetime cigarette exposure (pack-years). Cigarette smoking status was defined as never smoker, current smoker, and former smoker. As per the Centers for Disease Control and Prevention criteria, current smokers were defined as those who had smoked ≥100 cigarettes over their lifetime and smoked at the time of interview, former smokers as those who had smoked ≥100 cigarettes over their lifetime but were not currently smoking, and nonsmokers as those who had not smoked ≥100 cigarettes in their lifetime.

A detailed medical history of duration, exacerbations, and symptoms of COPD was recorded by the physician to diagnose COPD.

Clinical investigations

The diagnosis of COPD should be considered in anyone over the age of 35–40 years who has shortness of breath, a chronic cough, sputum production, or frequent winter colds and a history of exposure to risk factors for the disease. Chest radiographs were taken as a part of diagnosis. Spirometry is then used to confirm the diagnosis. Screening those without symptoms is not recommended pulmonary function test by use of spirometry for COPD patients based on which patients were classified as shown in Table 1.[5,6]

A detailed present and past dental history was recorded for the patients. Measurement of periodontal parameters was done. Calibration of examiner was done to reduce intraexaminer variability. Simplified oral hygiene index (OHI-S) of Greene and Vermillion was recorded.[7] Only fully erupted permanent teeth were scored. Natural teeth with full-crown restorations and surfaces reduced in height by caries or trauma were not scored; instead, an alternate tooth was then examined.

Plaque index (PI) as described by Löe 1967 was recorded. It assesses only the thickness of plaque at the gingival area of tooth. The evaluation was done on the entire dentition. Mouth mirror, a light source, a dental explorer, and air drying of the teeth and gingiva were used in the scoring of this index.[8]

Gingival index (GI) developed by Loe H and Silness in 1963 was recorded. It assesses the severity of gingivitis and its location in four possible areas by examining only the

Table 1: Classification of severity of airflow limitation in COPD patients

FEV1/FVC	Severity
FEV1 ≥80% predicted	Mild
50%≤ FEV1 <80% predicted	Moderate
30%≤ FEV1 <50% predicted	Severe
FEV1 <30% predicted	Very severe

qualitative changes (i.e., severity of the lesion) of the gingival soft tissue. The severity of gingivitis is scored on all surfaces of all teeth. A probe was used to assess the bleeding potential of the tissue.[8]

Full-mouth pocket probing depth (PPD) was measured from the free gingival margin to the base of the sulcus or periodontal pocket with a graduated Williams' periodontal probe at six sites (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual) per tooth. Clinical attachment level (CAL) defined as the distance from the cementoenamel junction to the bottom of the pocket/sulcus was measured with a graduated Williams' periodontal probe at six sites (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual) per tooth.

Statistical analysis

The data were entered into Microsoft Excel Spreadsheet and then checked for any missing entries. It was analyzed using Statistical Package for the Social Sciences (SPSS) version 21, IBM. This is computer software used for statistical analysis. All the variables were continuous variables which were summarized as mean and standard deviation. Graphs were prepared on Microsoft Excel. Normality of the data was checked by Shapiro–Wilk test. The data were found to be normal. Keeping in view the nature (continuous) and distribution (normal) of the data, inferential statistics were performed using parametric tests of significance.

Inferential statistics were performed using Chi-square test, independent *t*-test, and one-way analysis of variance test. Chi-square test was used to compare categorical data. Independent *t*-test was used to compare two groups. One-way analysis of variance test was used to compare more than two independent means. *Post hoc* pair-wise comparison was done using *post hoc* Bonferroni test. The level of statistical significance was set at 0.05.

Results

There has been a growing interest regarding the interaction between periodontal disease and respiratory diseases over the past years. COPD is a ubiquitous disease and is responsible for a significant number of deaths and considerable suffering in humans. After statistical analysis, the periodontal status of all the groups was compared and following results were obtained.

In the present study, the case group comprised 45 (69.2%) males and 20 (30.8%) females, whereas the control group had 47 (72.3%) males and 18 (27.7%) females, and this difference was not statistically significant ($P = 0.847$, NS). The mean age was higher in the case group (50.31 ± 11.413 years) compared to the control group (41.91 ± 8.089 years), and this difference was statistically significant ($P < 0.0001$S) [Table 2].

When smoking status was compared, in the case group, 40 (61.5%) were past smoker and 25 (38.5%) were current smoker. However, in the control group, 9 (13.8%) were nonsmoker, 27 (41.5%) were past smoker, and 29 (44.6%) were current smoker. There was no statistically significant difference between the smoking statuses of both the groups [Table 2].

The mean OHI-S score, PI score, and GI score were higher in the case group compared to the control group. However, test of significance of the mean values showed that the case group had statistically significant higher values of PI score compared to the control group ($P < 0.0001$). The mean PPD score and CAL score were significantly higher in the case group compared to the control group ($P < 0.0001$) [Table 3].

According to the global initiative for chronic obstructive lung disease (GOLD), COPD group (case) is subdivided into mild, moderate, severe, and very severe. The COPD intragroup comparison was done to evaluate the effect of the periodontal status in the subgroup of COPD group (case) patients. The mean score of OHI-S, PI, GI, PPD, and CAL was higher in the moderate and severe COPD group patients compared to the mild COPD group patients; however, this difference was not statistically significant [Table 4].

Discussion

There has been a growing interest regarding the interaction between periodontal disease and respiratory diseases over the past years. COPD is a ubiquitous disease and is responsible for a significant number of deaths and considerable suffering in humans.

Several mechanisms have been proposed for the association between these two highly common diseases. Dental plaque provides a reservoir for respiratory pathogen colonization that can be shed into saliva. Contamination of the distal portions of the respiratory tree by saliva containing such organisms

Table 2: Demographic details of case and control groups

Demographic details	Case (COPD)	Control	*P*
Count (%)	69.2% (male), 30.8% (female)	72.3% (male), 27.7% (female)	0.847 (NS)
Age (years)	50.31±11.413	41.91±41.91	<0.0001 (S)
Smoking (%)	61.5% (S), 38.5% (P)	13.8% (N), 41.5% (S), 44.6% (P)	0.003 (NS)

COPD: Chronic obstructive pulmonary disease, S: Significant, NS: Not significant

Table 3: Comparison of periodontal parameters of case and control groups

Periodontal parameters	Case	Control	*P*
OHI-S	2.571±0.4741	2.334±0.7322	0.030 (S)
PI	2.10±0.774	1.36±0.447	<0.0001 (S)
GI	1.703±0.5668	1.402±0.3529	<0.0001 (S)
PPD (mm)	2.454±0.6913	2.078±0.4446	<0.0001 (S)
CAL (mm)	3.048±0.8584	2.552±0.6955	<0.0001 (S)

OHI-S: Simplified oral hygiene index, PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment level

Table 4: Intragroup comparison of periodontal parameters in mm

COPD	OHI-S	PI	GI	PPD (mm)	CAL (mm)
Mild	2.438±0.6333	1.98±0.691	1.656±0.5929	2.500±0.7742	3.016±0.8009
Moderate	2.758±0.8732	2.06±0.838	1.723±0.5101	2.308±0.5727	2.927±0.8743
Severe	2.486±486	2.10±0.561	1.843±0.6997	2.786±0.6336	3.643±0.9378
P	0.244 (NS)	0.031 (S)	0.719 (NS)	0.235 (NS)	0.141 (NS)

OHI-S: Simplified oral hygiene index, PI: Plaque index, GI: Gingival index, PPD: Probing pocket depth, CAL: Clinical attachment level, COPD: Chronic obstructive pulmonary disease, S: Significant, NS: Not significant

may result in pulmonary infections. It is of great significance that the majority of pulmonary diseases are attributable to aerobic bacteria that are found in the oral flora in any oral diseases.[9] On the contrary, some of the facultative anaerobes that are responsible for periodontal breakdown, such as *Aggregatibacter actinomycetemcomitans*, *F. nucleatum, P. aeruginosa*, and *P. gingivalis*, also have been isolated from the infected lungs.[9]

Another mechanism proposed for gross airway epithelial damage observed in COPD involves release of proinflammatory cytokines (interleukin-8) from respiratory epithelium.[10] This subsequently results in recruitment and infiltration by neutrophils that then release proteolytic enzymes and toxic oxygen radicals. It is conceivable that oral bacteria in secretions in contact with respiratory epithelial surfaces may adhere to the mucosal surface. These bound oral bacteria may stimulate cytokine production by mucosal epithelium. It is also possible that cytokines originating from the oral tissues (for example, from the GCFs that exit the gingival sulcus to be mixed with whole saliva) may contaminate the distal respiratory epithelium to stimulate respiratory epithelial cells. The stimulated respiratory cells may then release other cytokines that recruit inflammatory cells (neutrophils) to the site.

These inflammatory cells may release hydrolytic enzymes and other modifying molecules, resulting in damaged epithelium that may be more susceptible to colonization by respiratory pathogens.

The findings of the present study are in concurrence with previous studies[11,12] and suggest that patients having obstructive lung disease had worse periodontal health status.

The present study is a case–control study, with the aim to determine the association between periodontal disease and COPD. A total of 130 patients were included in the study with equal distribution in each group. The control group comprised 65 systemically healthy patients, whereas the case group included 65 patients suffering from COPD. Detailed history of both case and control groups was recorded and lung function was analyzed by spirometry in the case group. Patients in the case group were further subdivided into mild, moderate, and severe on the basis of GOLD standard of severity and spirometry.

In the present study, the age of the study population for both case and control groups ranged from (30 years to <60) years with equal proportion of males and females in both the groups. The mean age of patients in the case group was significantly higher than that of the control group with no significant difference in the smoking status of both groups. Thus, age is a significant confounder in the study which might influence the results of study also. Similar results with age and smoking status as confounding factors were also obtained in a study conducted by Peter *et al*.[12] The potential drawback of the study lies in the fact that the extent of effect of age on the results of the study was not estimated.

Both the groups were further evaluated for clinical periodontal parameters such as OHI-S, PI, GI, probing pocket depth, and CAL. OHI-S given by Greene and Vermilion[7] has its advantage to evaluate an individual's level of oral cleanliness with a high level of reproducibility. This index was used to assess the oral health status in case and control groups in our study. Keeping in view its advantage to detect even small deposits of plaque, PI[8] was used in our study. Gingival status was assessed using GI by Löe and Silness (1963).[8]

Periodontal pockets are known to be reservoirs for microorganisms. It has been reported that periodontal pathogens have been isolated from infected lungs[13] and presence of periodontal pockets in controls may complicate the systemic conditions; therefore, the assessment of periodontal damage is a mandatory component in periodontal examination, and for this, PPD and CAL were measured using Williams' periodontal probe at six sites of all teeth for both the groups. Williams' periodontal probe is a manual probe which has been for long compared to automated probes. Gibbs *et al*.[14] stated that force controlled automated probes provide better results over manual probes. However, van de Velden and Vries[15] stated that standardized probing force does not lead to more reproducible pocket depth measurements. Mayfield *et al*.[16] later observed better intra- and interindividual reproducibility using a manual probe in comparison to automated force controlled probes. However, they even pointed out that these results may have been influenced by lack of familiarity with automated probes.

In our study, significantly higher mean OHI-S score was found in the case group (2.571 ± 0.7322) compared to the control group (2.334 ± 0.4741). These results are in accordance with other study by Sharma and Shamsuddin H (2001).[11]

In our study, significantly higher mean PI score was found in the case group (2.10 ± 0.774) compared to the control group (1.36 ± 0.447). These results are in accordance with other

studies by Peter *et al.* (2013)[12] and Prasanna SJ (2011).[17] It has been suggested that the supragingival plaque accumulation favors respiratory pathogen colonization and make susceptible patients more prone to a greater risk of developing respiratory disease.

In our study, significantly higher scores of mean GI were found in the case group (1.703 ± 0.5668) compared to the control group (1.402 ± 0.3529) and this difference was found to be statistically significant. This observation was substantiated by Katancik *et al*. (2005)[18] and Sharma and Shamsuddin[11] who also found significant positive relationship between GI scores and respiratory diseases. However, Scannapieco and Ho[19] reported no significant relationship between gingival bleeding and respiratory disease. The possible reason for significant positive relationship between GI score and COPD patients could be due to lack of dental hygiene and health awareness in this population owing to their low economic status.

The mean PPD values for case and control groups were 2.454 ± 0.6913 mm and 2.078 ± 0.4446 mm, respectively. The mean CAL values for case and control groups were 3.048 ± 0.8584 mm and 2.552 ± 0.6955 mm, respectively, and this difference was found to be statistically significant. Similar observation was reported by Sharma N and Shamsuddin H (2011)[11] and Zeng *et al*.[20] Both the above-mentioned studies document that poorer periodontal status was seen in patients with COPD when compared to the control group. Similarly, Ghani and Bhattacharya[21] suggested an association between chronic periodontitis and COPD. They observed that patients with more clinical attachment loss had a higher prevalence of diminished lung function. Positive correlation of our study results is also in synergy with the study done by Deo *et al*.,[22] which also proved that more severe the mean attachment loss, greater is the association with COPD.

In the present study, the case group was further divided into mild, moderate, and severe on the basis of GOLD spirometry guidelines. All periodontal parameters including OHI-S, PI, GI, PPD, and CAL have been observed to increase as severity of lung function impairment increases, i.e., FEV, percentage decreases. Furthermore, patients with severe impairment also had higher mean PPD and CAL values as compared to mild and moderate COPD patients. Similar observation was also made by Scannapieco and Ho[19] in their cross-sectional study for the evaluation of potential association between COPD and periodontal disease and concluded that lung function appeared to be diminished with increase in periodontal attachment loss.

Peter *et al*.[12] in an observational study aimed at the association between periodontal disease and COPD that the severity of lung obstruction increased as the value of PPD and CAL increased. Si *et al*.[23] in their study in Chinese population found a strong association between periodontitis and COPD and observed that PPD and CAL values significantly increased with increasing severity of COPD. The results are similar to our study where higher mean values of OHI S, PI, GI, PPD, and CAL are seen in patients with severe lung function impairment in comparison to COPD patients who are categorized as mild and moderate. Additional investigation incorporating randomized controlled intervention studies is needed to validate this reported association.

The strength of this study rests in several facts adapted in the methodology of this study. Most importantly, individuals enrolled in the case group were those having only COPD and no other systemic disease that influenced periodontal status. It has been concurred that institutionalized patients are more prone to periodontal disease because they are ignorant regarding their oral hygiene maintenance.

Therefore, only noninstitutionalized ambulatory individuals whose quality of life was not disturbed are enrolled in this study. Full-mouth examination was done, and patients having <20 teeth were excluded from the study. This criterion eliminated the probability of underestimating the true extent of periodontal disease. Furthermore, the most vital strength is the availability of reliable measurement of lung obstruction using spirometry, which is the gold standard for diagnosing COPD.

However, this study also has some limitations. First, since case control is not the best design of observational study, evidence from these are less accurate. Second, the relationship between cause and effect cannot be inferred from this study because of its observational and retrospective study design. Third, this study cannot completely exclude the possibility of residual confounding by other healthy lifestyle variables. Fourth, in the present study, the mean age was higher in case group compared to the control group years and this difference was statistically significant; this may be the reason for higher periodontal parameter as periodontal disease is more prevalent in older age group. Finally, this study is not an intervention study, so no evidence suggests that poor oral health characterized by inadequate hygiene resulting in the formation of dental plaque or periodontal disease is associated with serious pulmonary disease. Poor oral health may work in concert with other factor such as continued smoking, environmental pollutants, viral infections, and allergy to promote the progression and exacerbation of COPD. Hence, need for rigorous plaque control and treatment of oral infections, particularly in this risk group, is highly justified.

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Conflicts of interest

There are no conflicts of interest.

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