Association Between Periodontal Disease and Chronic Obstructive Pulmonary Disease - A Reality or Just a Dogma

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Background: Since last few years, association between chronic obstructive pulmonary disease (COPD) and periodontal disease has been promulgated. The present study was aimed at determining the association between these two diseases.

Methods: This observational study included 500 individuals consisting of 102 patients (case group) having COPD and 399 individuals as controls. Subjects in case group were well functioning and ambulatory patients having COPD as determined by their history and FEV₁ and FVC values (FEV₁ - forced expiratory volume in 1 second, FVC - forced vital capacity). Periodontal status was evaluated by following five indices: Plaque index (PI), Oral hygiene index (OHI), Gingival index (GI), Probing depth (PD) and Clinical attachment level (CAL). Lung function test was performed by all the patients (case group) and were then graded into mild, moderate, severe and very severe.

Results: Preliminary analysis was performed to identify the covariates in this study. Individuals in case group had significantly higher CAL, PD and OHI (p<0.0001) as compared to control group after adjusting for covariates. A significant negative correlation was observed between FEV₁ values and CAL, PD and GI, thus indicating a trend in which severity of lung obstruction increased as these periodontal indices worsened.

Conclusion: Although present study cannot ascertain causal association but provides with substantial evidence that poor periodontal health is associated with obstructive lung disease.

KEY WORDS

Periodontal-systemic disease interactions, periodontitis, cytokines

In the last few years there has been accumulating evidence that suggests an exquisite association between oral infections and systemic diseases. Periodontal medicine is based upon
new data that suggests that periodontal disease may significantly enhance the risk for certain systemic diseases or alter the natural course of systemic conditions. There are many systemic conditions which have been posed in literature to be influenced by periodontal infections.¹

Chronic obstructive pulmonary disease (COPD) is a severe respiratory disease characterized by chronic obstruction to airflow with excess production of sputum resulting from chronic bronchitis and/or emphysema. COPD is ubiquitously present in population and is the fourth leading cause of death in United States.² In 2020 COPD will probably become the third leading cause of death all over the world, following the trend of increasing prevalence of lung cancer. In the past few years association between COPD and periodontal disease is being promulgated by few researchers.

Scannapeico et al³ and Hayes et al⁴ were amongst the first researchers to publish their studies which suggested a potential association between poor oral health and COPD. Periodontal disease and COPD share a similar pathogenic mechanism. Moreover, respiratory infections results from aspiration of oropharyngeal flora into lower respiratory tract. Furthermore, it has been suggested that dental plaque may serve as reservoir for respiratory pathogens.⁵

Potential mechanisms that may account for these associations have been reviewed in past.⁶,⁷ Lower respiratory tract infections, including exacerbation of COPD, depend on the initial colonization of microbial pathogens on oral/pharyngeal surfaces. The pathogens are subsequently shed into the salivary secretions, together with oral bacteria, hydrolytic enzymes, and proinflammatory cytokines. Thus, the contents of this secretion may contaminate and induce alterations of the respiratory epithelium. Therefore, it can be entailed that the chronic inflammatory nature of periodontal disease may indirectly contribute to respiratory inflammation via mediators which are released into the saliva & carried to the respiratory epithelium.

It is therefore possible that periodontal disease activity may contribute to progression of COPD. An association between COPD and oral health was first noted in 1998.³,⁴ Since then, studies have been conducted to reconcile this association. However, there is as yet no direct evidence for a causal relationship between periodontal disease and respiratory disease.⁸

Thus, still the nature of relationship of periodontal disease with COPD remains unclear. In addition, the results published on this specific relationship are very less and those which exist, typically are secondary analysis of previously existing data sets. Azarpazhooh and Leake⁹ in their systematic review suggested that there is a poor evidence of weak association between COPD and periodontal disease. Although causal association between periodontal health status and risk of COPD is biological plausible but remains speculative. Thus, it is evident that, findings of a likely relationship between periodontal disease and COPD require further validation.

In the light of all above mentioned aspects of relationship between COPD and periodontal disease and paucity of sufficient literature in this regard, a systematic study was intended to carry out to evaluate the potential association between COPD and periodontal disease.
MATERIALS AND METHODS

Study Population

A total of 500 individuals were enrolled in this observational study after receiving informed consent. Case group consisted of 102 individuals having COPD as diagnosed by the physician at medicine department of Government Medical College and Hospital, Aurangabad. Individuals enrolled in this particular group were well functioning and ambulatory patients having COPD. Pulmonary/lung function test was done for all the individuals in case group and only those patients who had lung obstruction as evaluated by FEV₁/FVC values (FEV₁ - forced expiratory volume in 1 second, FVC - forced vital capacity) were enrolled in the study. Thus, individuals diagnosed as having COPD were those who had a history of chronic bronchitis and/or emphysema and showed lung obstruction in lung function test. Control group consisted of systemically healthy 399 individuals enrolled from out patient clinic of department of Periodontics.

Ethical Consideration

The protocol was approved by the institutional review boards for human subjects and was further analyzed and approved by Maharashtra University of Health Sciences (MUHS). Written informed consent was obtained from all individuals and were informed of the study objectives and the importance of the findings. Subjects identified as having periodontal disease were informed of their status and referred for treatment at the Dental Hospital.

Case group Criteria for Patient Selection

Inclusion criteria. 1. Patients diagnosed as having chronic obstructive pulmonary disease (COPD) by a physician.
   2. Patient aged 30 years and above.
   3. Presence of at least 20 natural teeth.

Exclusion criteria. 1. Patients having any other systemic disease such as diabetes mellitus, which are known to influence periodontal status.
   2. Patients with a history of periodontal treatment in last six months.
   3. Patients who were unable to perform lung function test.
   4. Patients taking any medication known to influence the periodontal tissues.
   5. Pregnant women and lactating mothers.

Selection Criteria for Control Group

Control group consisted of systemically healthy patients aged 30 years or above.
Method of collection of data. A proforma was designed to record the patient’s demographic details, lifestyle characteristics, medical history, oral hygiene measures, periodontal health status and history of COPD and lung function. Demographic data included age, gender, religion, address, educational status, income and frequency of dental visits.

A thorough medical history of each patient was recorded and any patient found with systemic diseases (other than COPD) known to affect the periodontium was excluded from the study. Lifestyle characteristics examined included history of smoking in cigarette smoked per day and tobacco chewing.

Since smoking status and smoking history are strongly related to both airway and periodontal disease, they may be strong confounders of the association between respiratory obstruction and periodontal disease thereby may variate the inference of the study. Therefore, individuals were stratified according to their smoking status and lifetime cigarette exposure (pack-years). Cigarette smoking status was defined as never smoker, current smoker, or former smoker. A former smoker was defined as not currently smoking, but with a lifetime consumption of greater than 100 cigarettes. A current smoker was defined as currently smoking with a lifetime consumption of greater than 100 cigarettes. Individual was considered as non smoker when he/she had not smoked 100 or more cigarettes in their lifetime.

Evaluation of periodontal status. The subject’s periodontal status was evaluated using the following measures:

1) Oral Hygiene Index (OHI) 10
2) Plaque Index (PI)11
3) Gingival Index (GI) 12
4) Probing Depth (PD)
5) Clinical Attachment Level (CAL)

Oral health examinations of all the individuals were conducted by a single periodontist (Peter). All the teeth in the oral cavity were examined except for third molars. Probing depth and clinical attachment level were measured using Williams graduated periodontal probe at six sites per tooth and thereafter a mean was calculated for the whole oral cavity. At each site PD, recession and CAL were calculated based on the probed distances in millimeters from gingival margin to cemento-enamel junction and the base of sulcus.

Evaluation of obstructive lung disease. A detailed history of duration, exacerbations, hospitalizations, and symptoms of Chronic Obstructive Pulmonary Disease (COPD) were recorded. Lung function test was carried out for each patient in case group based on which patient was classified as having mild, moderate, severe and very severe COPD. Patients had to meet the following criteria at baseline:13 post bronchodilator FEV₁/FVC ratio of less than 0.7 and FEV₁ of less than 80% of predicted value, no primary diagnosis of asthma and no previous lung volume reduction surgery, lung transplantation, or pneumonectomy.

Lung function was assessed by use of spirometry in presence of trained professional under the supervision of a physician (Bardapurkar). Spirometry assesses lung function by measuring the volume of air that the patient can expel from the lungs after a maximal inspiration.
The indices derived from this forced exhaled maneuver have become the most accurate and reliable way of supporting a diagnosis of COPD. It is on the values given by this test that COPD guidelines around the world base the assessment of mild, moderate, severe and very severe disease levels. Patient’s height, weight, gender and age were fed into the patient profile in the Spirometer. Based on the above mentioned data predicted values of the parameters of lung functions are calculated and then compared with the existing data of lung function. Airflow limitation was defined as a reduced FEV\textsubscript{1}/FVC and only those patients showing spirometer values meeting American Thoracic Society (ATS) criteria were considered. Airflow obstruction severity was classified according to guidelines given by Global Initiative for Obstructive Lung Disease (GOLD). Severity of COPD was categorized as mild (FEV\textsubscript{1} ≥ 80% predicted), moderate (50% ≤ FEV\textsubscript{1} < 80% predicted), severe (30% ≤ FEV\textsubscript{1} < 50% predicted) and very severe (FEV\textsubscript{1} < 30% predicted).

**Statistical note.** Descriptive statistics for covariates were obtained with regard to measurement scale. Statistical significance analysis between COPD positive (case) and negative (control) groups was performed on these covariates using Chi-square and Student’s t test depending on the scale. Mean and standard deviation for each periodontal parameter were obtained for both the groups. Covariates differing significantly between groups were used to obtain adjusted mean and standard deviation for each periodontal parameter using analysis of covariance (ANCOVA). The assumptions of ANCOVA were ascertained while estimating adjusted means for each parameter. Further, in case group, the relationship between FEV\textsubscript{1} values, indicating the severity of COPD, and each parameter (covariates adjusted) was determined using Pearson’s correlation coefficient. The statistical significance was evaluated at 5% level.

**RESULTS**

A demographic profile of subjects included in the study was taken along with their lifestyle characteristics (Table-1). The mean age of patients in the case group was 59.48±11.13 years, while in control group it was 49.69±10.16 years. The difference in the mean age was statistically significant (p < 0.0001). The proportion of males in two groups was also significantly different (p = 0.0026). Educational level was not associated with presence or absence of COPD (p = 0.0603). Proportion of tobacco chewing cases insignificantly differed in two groups (p = 0.1167). However, smoking status and pack-year significantly differed in two groups with p-values of 0.0009 and 0.0037 respectively.

Table-2 shows the mean periodontal indices of samples in the study groups. Results show that the periodontal indices (CAL, PD, OHI and GI) were significantly (p<0.0001) worse in case group as compared to control group. Since age, smoking status and pack years were the significant confounding factors in this study (Table-1), the mean periodontal indices were obtained after adjusting for these covariates using ANCOVA. Table-2 shows the adjusted mean levels of periodontal indices and standard deviation for periodontal parameters in both study groups (case and control). Individuals in case group showed significantly worse CAL, PD and OHI (p<0.0001) as compared to control group, whereas GI showed insignificant difference (p = 0.056) between groups. The results for PI remained insignificant (p>0.05) even after adjusting for covariates.

Further, to determine the relationship between FEV\textsubscript{1} values and each periodontal parameter in case group, Pearson’s correlation analysis was performed. Figure-1 shows scatter plot of
periodontal indices (after adjusting for covariates) and FEV$_1$, categorizing samples according to the severity of COPD. Four of the five periodontal parameters (CAL, PD, GI and PI) indicated negative relationship with FEV$_1$, while OHI showed almost no correlation with FEV$_1$. Statistical analysis revealed a significant correlation between three periodontal indices (CAL, PD and GI) and FEV$_1$ with $p < 0.0001$, while PI and OHI showed insignificant relation with FEV$_1$ as indicated by $p$ values of 0.1592 and 0.5879 respectively. Thus it is evident that patients with very severe COPD had higher CAL, PD and GI values, as compared to patients with less severe COPD indicating marked sensitivity of CAL, PD and GI to COPD severity.

DISCUSSION

There has been an arising interest regarding the interaction between periodontal disease and respiratory diseases over the past few years. COPD is a ubiquitous disease and is responsible for a significant number of deaths and considerable suffering in humans. The findings of the present study are in concurrence with previous studies$^{16,17}$ and suggest that patients having obstructive lung disease had worse periodontal health status.

Present study was aimed at determining the association between periodontal disease and COPD. Statistical analysis of demographic characteristics of study population showed that age, smoking status and smoking pack-years were the significant confounders in this study (Table-1). Table-2 shows the comparison of mean periodontal indices between case and control group. Results reveal that periodontal status was poorer in case group as compared to control group. However, as age, smoking status and smoking pack-years were the significant confounding variables in this study mean periodontal indices were adjusted for these covariates and then significance of difference between groups was obtained. Results unveil that individuals in case group had significantly ($p<0.0001$) worse CAL, PD and OHI as compared to control group whereas this difference was not significant for PI and GI. These findings are in commemoration with the findings of previous studies$^{4,16,17}$ which suggest periodontitis as an independent risk factor for COPD. However, the results of the present study are contradictory to the results obtained by Prasanna SJ$^{18}$ to some extent wherein there were associations noted between the GI and chronic respiratory disease.

Interestingly, a trend was noted in which periodontal indices (after adjusting for age, smoking status and smoking pack-years) worsened as the FEV$_1$ values decreased suggesting a negative correlation between periodontal parameters and FEV$_1$ values. This observed correlation was highly significant ($p<0.0001$) for CAL, PD and GI but was insignificant for PI and OHI. Results of this study partially contravene to the inference interpolated by J. Katancik et al$^{17}$ in which GI and LOA (loss of attachment) were significantly associated to lung obstruction but PD showed insignificant association.

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 may result in pulmonary infections. It is of great significance that majority of pulmonary diseases are due to aerobic bacteria that are found in the oral flora in any oral diseases. On the contrary, some of the facultative anaerobes which are responsible for periodontal destruction like Actinobacillus actinomycetemcomitans, Fusobacterium nucleatum, Pseudomonas aeruginosa and Porphyromonas gingivalis also have been isolated from infected lungs.$^{20}$ Furthermore, cytokines released during the progression of
periodontal disease by infected periodontal tissues may alter respiratory epithelium. One mechanism proposed for gross airway epithelial damage observed in COPD involves release of proinflammatory cytokines (IL-8) from respiratory epithelium. This subsequently results in recruitment and infiltration by neutrophils which 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 gingival crevicular fluids which 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. Thus, periodontal disease can probably contribute in exacerbation of COPD.

COPD shares a similar pathogenic mechanism with periodontal disease. In both diseases, a host inflammatory response is mounted in response to chronic challenge by bacteria in periodontal disease and cigarette smoking in COPD. The resulting neutrophil influx leads to release of oxidative and hydrolytic enzymes that causes tissue destruction directly. One of the major complications of COPD is the occurrence of “exacerbations,” or episodes in which there are objective signs of worsening of bronchitis. However, the factors responsible for the initiation of exacerbation are not completely known, although they are thought to be provoked in part by bacterial infection.

Therefore it is possible that accumulation of oral pathogens associated with periodontal disease may increase the risk for serious lower respiratory tract infection in susceptible subjects, including pneumonia or exacerbation and progression of COPD. Thus, it is biologically plausible that periodontal disease and COPD may have an association with each other.

The various epidemiological studies and reviews have evinced the potential role of oral bacteria in respiratory infections. The evidence gathered suggests that poor oral health may serve as a significant risk factor for lower respiratory infection, especially in high-risk groups.

The strength of this study rests in several facts adapted in the methodology of this study. Most importantly, individuals enrolled in case group were those having only COPD and no other systemic disease which influenced periodontal status. It has been concurred that institutionalized patients are more prone to periodontal disease as they are ignorant towards their oral hygiene maintenance. Therefore, only non institutionalized ambulatory individuals whose quality of life was not perturbed were enrolled in this study. Full mouth examination was done and patients having less than 20 teeth were excluded from the study. This criterion eliminated the probability of underestimating the true extent of periodontal disease. Furthermore, most vital strength is the availability of reliable measurement of lung obstruction using spirometry, which is the gold standard for diagnosing COPD.

In conclusion, though present study cannot ascertain causal association but provides substantial evidence that poor periodontal health is associated with obstructive lung disease. In majority of cases COPD is preventable. If periodontitis does enhance the risk of COPD then dentistry especially periodontics has a potentially significant responsibility and an important role
to play. Present study reveals that periodontal indices (CAL, PD and OHI) are poorer in COPD individuals as compared to control group. Results of present study also unveil a trend of increasing values of periodontal indices (CAL, PD, and GI) as the severity of lung obstruction increases. When all of the evidence is considered it can be firmly stated that the association between periodontal disease and COPD is not just a dogma. Thus present study in concurrence with previous studies ideates that improved oral health may help in prevention of progression of obstructive lung disease. Further investigations incorporating randomized controlled interventional studies are needed to validate this reported association.

ACKNOWLEDGEMENTS

The authors report no conflicts of interest related to this study.

No conflict of interest reported

REFERENCES


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Submitted June 05, 2012; accepted for publication December 31, 2012.

**Figure-1:**

*Relationship between periodontal parameters and FEV1 values in case group*
Table 1: Demographic details of study population

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>COPD: Yes</th>
<th>COPD: NO</th>
<th>Overall</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size (N)</td>
<td>102</td>
<td>399</td>
<td>501</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td>Age in years (mean ± SD)</td>
<td>59.48 ± 11.13</td>
<td>49.69 ± 10.16</td>
<td>51.68 ± 11.08</td>
<td>0.0026‡ (S)</td>
</tr>
<tr>
<td>Sex (M)</td>
<td>92 (90.2%)</td>
<td>303 (75.9%)</td>
<td>395 (78.8%)</td>
<td>0.0603‡ (NS)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Illiterate</td>
<td>29</td>
<td>72</td>
<td>101</td>
<td>0.1167‡ (NS)</td>
</tr>
<tr>
<td>Primary</td>
<td>22</td>
<td>88</td>
<td>110</td>
<td>0.3298† (NS)</td>
</tr>
<tr>
<td>High school</td>
<td>34</td>
<td>147</td>
<td>181</td>
<td>0.0009‡ (HS)</td>
</tr>
<tr>
<td>Higher secondary</td>
<td>15</td>
<td>60</td>
<td>75</td>
<td>0.0009‡ (HS)</td>
</tr>
<tr>
<td>Graduate (College)</td>
<td>2</td>
<td>32</td>
<td>34</td>
<td>0.0009‡ (HS)</td>
</tr>
<tr>
<td>Behavioral</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tobacco (Yes)</td>
<td>36 (35.3%)</td>
<td>107 (26.8%)</td>
<td>143 (28.5%)</td>
<td>0.1167‡ (NS)</td>
</tr>
<tr>
<td>Duration (mean ± SD)</td>
<td>13.75 ± 7.41</td>
<td>12.37 ± 7.78</td>
<td>12.72 ± 6.94</td>
<td>0.3298† (NS)</td>
</tr>
<tr>
<td>Never smokers</td>
<td>31 (30.3%)</td>
<td>196 (49.1%)</td>
<td>227 (45.3%)</td>
<td>0.0009‡ (HS)</td>
</tr>
<tr>
<td>Former smokers</td>
<td>55 (53.9%)</td>
<td>139 (34.8%)</td>
<td>194 (38.7%)</td>
<td>0.0009‡ (HS)</td>
</tr>
<tr>
<td>Current smokers</td>
<td>16 (15.7%)</td>
<td>64 (16.1%)</td>
<td>80 (15.9%)</td>
<td>0.0037† (S)</td>
</tr>
<tr>
<td>Pack-years (mean ± SD)</td>
<td>15.88 ± 10.61</td>
<td>11.99 ± 4.58</td>
<td>13.00 ± 6.87</td>
<td>0.0037† (S)</td>
</tr>
</tbody>
</table>

†: t-test for independent samples; ‡: Chi-square test

HS: Highly significant, S: Significant, NS: Not significant
### Table 2

Mean Periodontal Indices (±SD) of study population

<table>
<thead>
<tr>
<th>Periodontal Parameter</th>
<th>COPD: Yes</th>
<th>COPD: NO</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CAL</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>3.85 ± 0.61</td>
<td>2.94 ± 0.35</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Adjusted</td>
<td>3.59 ± 0.59</td>
<td>2.91 ± 0.23</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td><strong>PD</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>3.26 ± 0.50</td>
<td>2.59 ± 0.29</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Adjusted</td>
<td>3.06 ± 0.51</td>
<td>2.60 ± 0.18</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td><strong>OHI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>2.97 ± 0.57</td>
<td>2.28 ± 0.36</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Adjusted</td>
<td>2.72 ± 0.61</td>
<td>2.19 ± 0.28</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td><strong>PI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>2.22 ± 0.31</td>
<td>2.16 ± 0.22</td>
<td>0.0992 (NS)</td>
</tr>
<tr>
<td>Adjusted</td>
<td>2.12 ± 0.31</td>
<td>2.18 ± 0.19</td>
<td>0.056 (NS)</td>
</tr>
<tr>
<td><strong>GI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>2.38 ± 0.31</td>
<td>2.25 ± 0.24</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Adjusted</td>
<td>2.27 ± 0.32</td>
<td>2.26 ± 0.19</td>
<td>0.9180 (NS)</td>
</tr>
</tbody>
</table>

*Mean periodontal indices after adjusting for age, smoking status and pack-years

HS: Highly significant, NS: Not significant

Mean periodontal indices in case and control group before and after adjusting for age, smoking status and smoking pack-years. Statistically significant difference was observed for CAL, PD and OHI between two groups, whereas PI and GI showed insignificant difference after adjusting for covariates

**Abbreviations used:** CAL- Clinical attachment level, PD- Pocket depth, GI- Gingival index, PI- Plaque index, OHI- Oral hygiene index, FEV₁- Forced expiratory volume in 1 second, FVC- Forced volume capacity, COPD- Chronic obstructive pulmonary disease, ANCOVA- Analysis of variance