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Research Article

Association between Chronic Obstructive Pulmonary Disease and Periodontal Disease

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Abstract

Introduction: A relationship between poor periodontal health and respiratory disease has been suggested by various studies available in the literature. The present study was conducted to evaluate potential association between periodontal disease and respiratory diseases and clinically coordinate the severity of periodontal disease with chronic obstructive pulmonary disease (COPD).

Materials and Methods: 100 patients of COPD (test group) and 100 Patients without COPD (Control group) were engaged for this present study. Patients with COPD were categorized into mild, moderate and severe category on the basis of Spirometry. Periodontal health was assessed by measuring probing pocket depth (PPD), Clinical Attachment Loss (CAL) and Oral Hygiene Index (OHI).

Results: COPD patients had a higher mean periodontal index (OHI, PPD and CAL) than those without COPD. A distinct trend of an increase in the periodontal parameters (OHI, PPD and CAL), was noticed with increase in the severity of the COPD (according to GOLD criteria). The association was found to be statistically significant. The mean score of FEV1/FVC and severity of FEV1/FVC was significantly more among COPD group in comparison to the non-COPD group. There was a significant difference in mean FEV1 between mild, moderate and severe COPD groups and also significant difference between moderate group and severe group.

Conclusion: Based on the results of the present study, it might be concluded that the risk for COPD emerged to be significantly increased when attachment loss was found to be severe. It is believed that oral interventions improve oral hygiene status and might be shown to lower the severity of lung infection in susceptible populations.

Keywords: *Periodontal Disease; Chronic Obstructive Pulmonary Disease; Lung Function; Clinical Attachment Loss, Risk Factor*

Introduction

The relationship between periodontal health or disease and systemic health or disease gave rise to a new discipline in Periodontology termed "Periodontal Medicine," which was proposed by William Ray C, and Steven Offenbacher [1] in 1996 World workshop in Periodontics. Periodontitis is defined as an inflammatory disease of supporting tissues of teeth caused by specific microorganisms or groups of specifics microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession or both [2].

Dental plaque serves as a pool for respiratory pathogens, especially in high-risk patients with poor oral hygiene. During periodontitis, oral bacteria may continuously stimulate periodontal tissues to release the inflammatory mediators such as cytokines, and these cytokines from the gingival crevicular epithelium (GCF) [3] enter the whole saliva which may contaminate distal respiratory epithelium and there is a recruitment of inflammatory cells (e.g. neutrophils) to the site. Hydrolytic enzymes are released from inflammatory cells and causes damage of epithelium, which make the epithelium more susceptible to colonization and infection by respiratory pathogens. Proteins inside the suction may devastate macromolecules on mucosal surface to uncover the receptors that allow attachment and colonization of respiratory pathogens conjointly devastate the defensive secretory molecules such as mucins, which clear the microbes from the mucosal surface [4]. There is a prove that hereditary character of respiratory pathogens separate the recouped from broncho-alveolar lavage fluid of elderly individuals, who are hospitalized is same as confines from their dental plaques [5].

Several studies provide evidence that oral cavity may influence the progression and/or initiation of lung diseases such as chronic obstructive pulmonary disease (COPD). Chronic Obstructive Pulmonary Disease has been defined by the Global Initiative for Chronic Obstructive Pulmonary Disease (COPD) as a disease state characterized by airflow limitation that is not fully reversible [6]. It includes two important disorders Chronic Bronchitis and Emphysema. Chronic Bronchitis is an inflammatory condition associated with excessive production of tracheobronchial mucous production sufficient to cause the cough with expectoration for at least 3 months of the year for two or three excessive years. Emphysema is the destruction of the air spaces distal to the terminal bronchiole with the destruction of alveola septa. COPD is characterized by chronic obstruction to the airflow due to chronic bronchitis and or emphysema [7].

In the early 20th century, "focal infection" theory came into existence and various studies explored a possible role for PD as a risk factor for systemic diseases over the past two decades [8], including cardiovascular diseases [9], diabetes [10], adverse pregnancy outcome [11], osteoporosis [12], rheumatoid arthritis [13], and Chronic obstructive pulmonary disease (COPD) [14,15]. According to the Global Burden of disease (GBD) study 2000, COPD was responsible for an estimated 2.75 million deaths worldwide. In the USA, COPD is currently the fourth leading cause of death by 2020 (US Department of health and human services 2003) [16]. A relationship between poor periodontal health and respiratory disease, especially in high-risk individuals, with the history of prolonged cigarette smoking, has been demonstrated by various recent microbiologic and epidemiologic studies [17].

Pathophysiological link between these two chronic diseases was first put forward in the 1990s. Neutrophil phenotype and behaviour is considered to be as a shared final pathway in the pathological process [18]. The neutrophils confined from patients with COPD have a more noteworthy damaging potential [19], more noteworthy unconstrained attachment to epithelial cells beneath the flow [20], and distinctive and more convoluted transient flow than those from suitable controls [21], which may account for their destructive potential. Bronchial tissue from patients with COPD have more numbers of neutrophils [22] similar to the airway secretions in chronic bronchitis patients [23]. However, fluorodeoxyglucose positron emission tomography scanning has shown that the activated neutrophils and neutrophils elastase enzymes co-localize with areas and severity of emphysema [24,25].

Alpha Antitrypsin (AAT) was along these lines appeared to hinder neutrophil elastase, a protein presents in high concentrations in neutrophil azurophilic granules and a powerful degrader of elastin, collagen and laminin. The anti-proteases alpha antitrypsin, serine protease inhibitor gene (SERPINA 3) and secretory leukocyte protease inhibitor (SLP1) can inhibit Neutrophil elastase which in turn inhibits TIMP-4 which is an inhibitor of Matrix Metalloproteinase (MMP). MMP in turn inactivates antiproteases which ultimately lead to tissue destruction [18]. Cigarette smoke contains higher amounts of oxidants and free radicals which are primary part of the oxidative stress endured by host cells [26]. In addition to these exogenous sources, activated inflammatory cells such as macrophages and neutrophils generate reactive oxygen species (ROS) in response to environmental insults, bacteria and their products [27].

ROS formation occurs in every human cell as a bi-product of physiological metabolism. Neutrophils and ROS can be generated as part of the respiratory burst. When there is an excess amount of ROS released, it may result in oxidative stress causes tissue damage. Oxidative stress is primary factor in COPD and periodontitis and also associated in the pathophysiology of both [21]. Ideally in chronic periodontitis, unstimulated neutrophils had higher spontaneous release of ROS which might be determined by chemiluminescence in control individuals [28]. Also, superoxide might have a greater chemo attractant production [29] and hydrogen peroxide may channelise the expression of attachment molecules [30] and have greater inflammatory cell infiltrate. Significantly, results from three preliminary intervention trials [31-33] demonstrated that attention to oral hygiene, either by the use of mechanical cleansing and/or oral antiseptic rinses, such as Povidone Iodine or chlorhexidine gluconate, significantly reduced the rate of lower respiratory tract infection in institutionalized patients.

Material and Methods

Source of data

The study was comparative cross-sectional study. It was conducted in the Outpatient department, Civil Hospital, Sirsa and Department of Periodontics, JCD dental college, Sirsa. An ethical clearance was obtained before conducting the study from the institutional ethical committee**.** The protocol was explained to the pa-

tients and they were requested to sign a consent form. The Study group comprised of 200 Subjects, within the age group of 20 - 45 years of age, reporting to the Outpatient Department, Civil Hospital, Sirsa and Department of Periodontics, JCD dental college, Sirsa.

Sampling

In this cross-sectional study, 100 patients were selected on purposive selection criteria from the Outpatient Department, Civil Hospital and Department of Periodontics, JCD dental college, Sirsa. The patients were in the age group 20 - 45 years, of which100 patients belonged to group A (Test group) and 100 belonged to group B (Control group). Both the groups comprised age- and sex-matched individuals. Group-A comprised 100 patients diagnosed as COPD. Group B- comprised100patients diagnosed as Non-COPD.

Inclusion criteria

1. Patient should be between 20 - 45 years.
2. Patient should have at least 6 natural teeth.
3. Patient who underwent pulmonary function test (PFT).
4. Patients with non-COPD should be relatives of the COPD patients.

Exclusion criteria

1. Patient with compromised medical conditions like bleeding disorders, malignancy etc.
2. Pregnancy and lactating women.
3. Physically or mentally challenged patients.

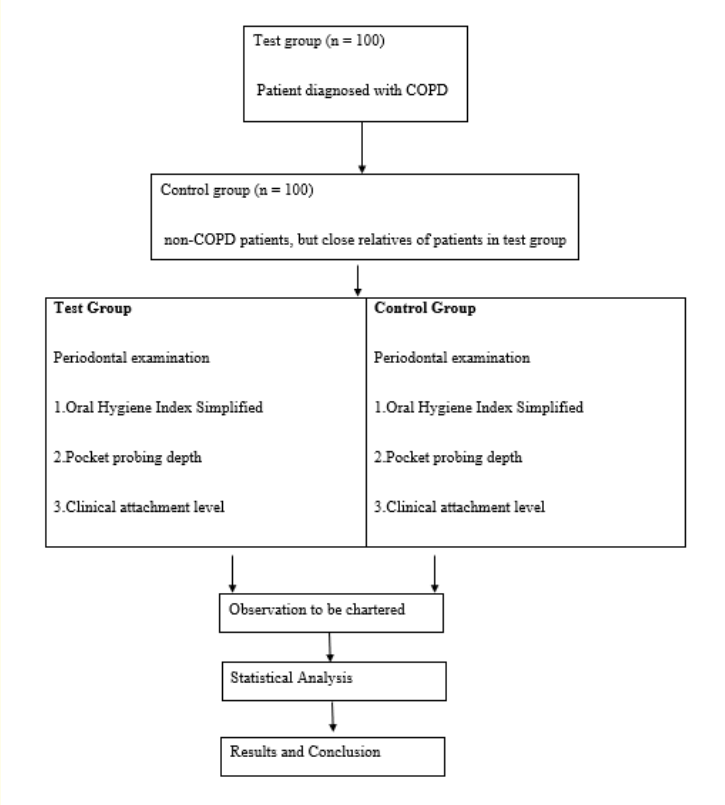


Figure 1: *Study design.*

Methodology

Clinical examination

The subjects were subjected for an initial examination consisting of

a. Subjects were diagnosed for the presence or absence of COPD using pulmonary function tests. If test came positive then the gold criteria was used for categorising COPD severity.

COPD diagnosis

- It was done with the help of pulmonary function test using Spirometer.
- Forced Expiratory Volume (FEV1) is reduced.

GOLD criteria for COPD severity

- Stage I: Mild COPD FEV1/FVC<0.70 FEV_1≥ 80% normal
- Stage II: Moderate COPD FEV1/FVC<0.70 FEV_1 50-79% normal
- Stage III: Severe COPD FEV1/FVC<0.70 FEV_1 30-49% normal
- Stage IV: Very Severe COPD FEV1/FVC<0.70 FEV_1 <30% normal, or <50% normal with chronic respiratory failure present

b. Both COPD and non-COPD subjects were subjected to complete history and periodontal examination.

c. Complete history was taken including age, sex, and smoking status. BMI was also being calculated. Periodontal examination with following clinical parameters:

- Oral Hygiene Index (OHI-S)
- Probing pocket Depth (PPD)
- Clinical Attachment Loss (CAL).

All the clinical parameters were recorded to the nearest millimeter using UNC - 15 probe at baseline. For diagnosing severity of Periodontitis, we used Armitage classification of periodontitis and the classification of periodontitis used in US Third National Health and Nutrition Examination Survey in which participants were divided into 3 groups:

1. Mild periodontitis:

- ≥ 1 teeth with ≥ 3 mm PD (CAL: 1 - 2 mm),
- ≤ 30% of teeth with ≥ 4 mm PD (CAL: 1 - 2 mm) and
- No teeth with ≥ 5 mm PD.

2. Moderate periodontitis:

- ≥ 1 teeth with ≥ 5 mm PD (CAL: 3 - 4 mm) or
- 30 - 60% teeth with ≥ 4 mm PD (CAL: 3 - 4 mm).

3. Severe periodontitis:

- ≥ 60% of teeth with ≥ 4 mm PD (CAL: ≥ 5 mm) or
- ≥ 30% of teeth with ≥ 5 mm PD (CAL: ≥ 5 mm).

Then various periodontal parameters and indices were used for assessing the periodontal health status in COPD and non-COPD subjects. Data obtained from this study was subjected to statistical analysis for the possible outcome.

Results and Statistical Analysis

The software used for the statistical analysis were SPSS (statistical package for social sciences) version 21.0 and Epi-info version 3.0. The values were represented in Number (n), Percentage (%), and Mean (υ). The statistical tests used were:

Unpaired or Independent T-test is used for comparison of mean value between 2 groups.

ANOVA (Analysis of Variance) test for comparison of difference between mean values of more than 2 groups.

Chi square test is used to investigate whether distributions of categorical variables differ from one another.

The p-value was taken significant when less than 0.05 ($p < 0.05$) and confidence interval of 95% was taken.

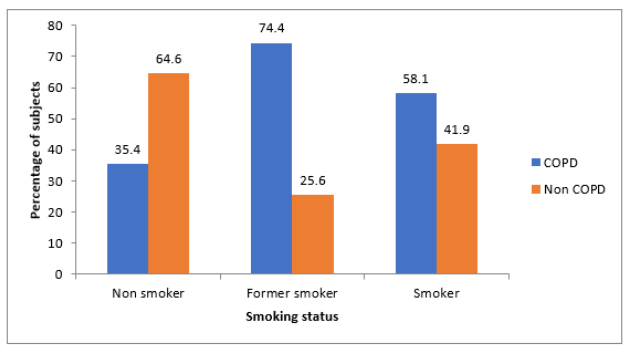
Smoking status	COPD N (%)	Non COPD N (%)	p-value
Non smoker	35 (35.4%)	64 (64.6%)	0.000*
Former smoker	29 (74.4%)	10 (25.6%)	
Smoker	36 (58.1%)	26 (41.9%)	
Total	100 (50%)	100 (50%)	

Table 1: *Comparison of smoking status among COPD and non-COPD group.*

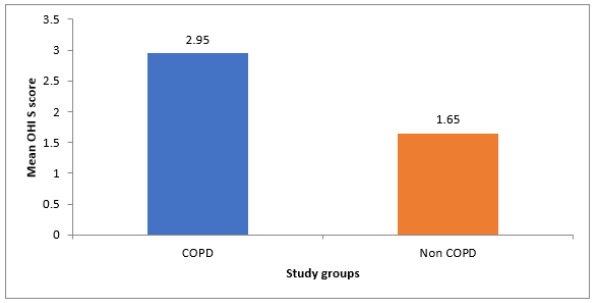
The distribution of smoking status was compared between COPD and Non-COPD groups using the Chi-Square Test. There was a significant difference in the distribution of smoking status between COPD and Non-COPD groups. Former and current smokers were significantly more among COPD group and non-smokers were significantly more Non-COPD group. This implies that COPD is found to be more evident in smokers which are a known fact.

Study groups	Oral Hygiene Index score (Mean ± SD)	p-value
COPD	2.95 ± 1.37	0.000*
Non COPD	1.65 ± 0.91	

Table 2: *Comparative assessment of mean Oral Hygiene Index score among COPD and Non-COPD groups. Test applied: Independent t test, *indicates statistically significant difference.*



Graph 1: *Comparison of smoking status among COPD and Non-COPD group.*

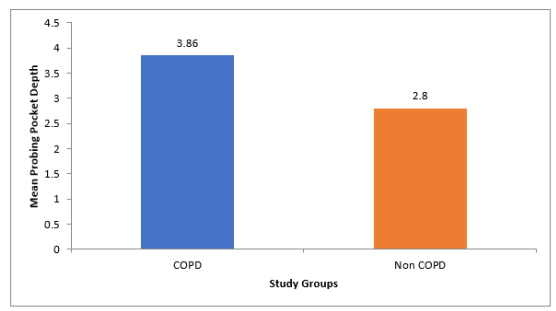


Graph 2: *Comparative assessment of mean Oral Hygiene Index score among COPD and Non-COPD groups.*

The Mean OHI value were compared between COPD and Non-COPD groups using the Unpaired or independent t-test. There was a very high significant difference in mean OHI value between COPD and Non-COPD groups. Mean OHI value were significantly more among COPD group in comparison to the Non-COPD group. This also proved that the frequency of tooth brushing was less among the patients with COPD. Also, these patients had a negligent attitude and poor oral hygiene apart from poor health status.

Study groups	Probing Pocket Depth (Mean ± SD)	p-value
COPD	3.86 ± 1.092	0.000*
Non COPD	2.80 ± 0.81	

Table 3: *Comparative assessment of mean probing pocket depth among COPD and Non-COPD groups. Test applied: Independent t test, *indicates statistically significant difference.*

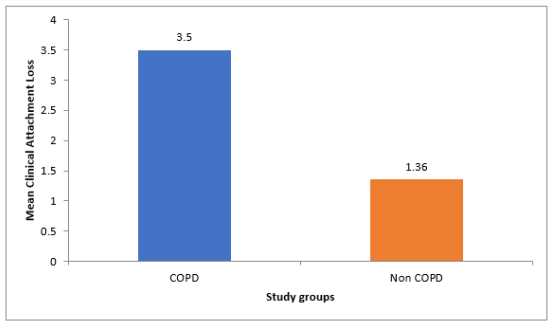


Graph 3: *Comparative assessment of mean Probing Pocket Depth among COPD and Non-COPD groups.*

The Mean PPD value were compared between COPD and Non-COPD groups using the Unpaired or independent t-test. There was a very high significant difference in mean PPD value between COPD and Non-COPD groups. Mean PPD value were significantly more among COPD group as compared to the Non-COPD group.

Study groups	Clinical Attachment Loss (Mean ± SD)	p-value
COPD	3.50 ± 1.68	0.000*
Non COPD	1.36 ± 1.58	

Table 4: *Comparative assessment of mean clinical attachment loss among COPD and Non-COPD groups.*
*Test applied: Independent t test, *indicates statistically significant difference.*

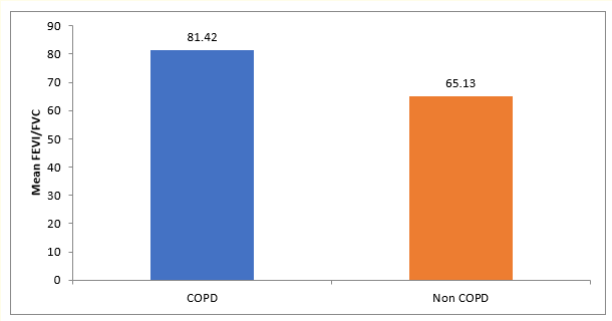


Graph 4: *Comparative assessment of mean Clinical Attachment Loss among COPD and Non-COPD groups.*

The Mean CAL value were compared between COPD and Non-COPD groups using the Unpaired or independent t-test. There was a very high significant difference in Mean CAL value between COPD and Non-COPD groups. Mean CAL value were significantly more among COPD group in comparison to the Non-COPD group.

Study groups	FEVI / FVC (Mean ± SD)	p-value
COPD	81.42 ± 15.31	0.000*
Non COPD	65.13 ± 25.64	

Table 5: *Comparative assessment of mean FEVI/FVC score among COPD and Non-COPD groups.*
*Test applied: Independent t test, *indicates statistically significant difference.*

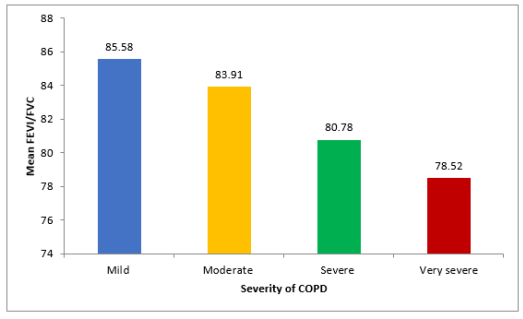


Graph 5: *Comparative assessment of mean FEVI/FVC score among COPD and Non-COPD groups.*

The Mean FEV1/FVC% value was compared between COPD and Non-COPD groups using the Unpaired or independent t-test. There was a very high significant difference in mean value between COPD and Non-COPD groups. Mean FEV1/FVC value was significantly more among COPD group in comparison to the Non-COPD group.

Severity of COPD	FEVI / FVC (Mean ± SD)	p-value
Mild	85.58 ± 17.78	0.520
Moderate	83.91 ± 14.21	
Severe	80.78 ± 16.34	
Very severe	78.52 ± 14.79	

Table 6: *Comparative assessment of mean FEVI/FVC score according to severity of COPD.*
Test applied: One way ANOVA.

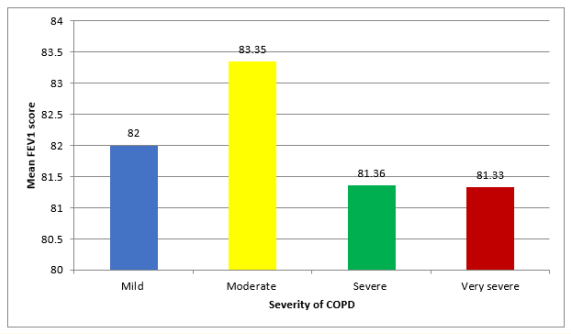


Graph 6: *Comparative assessment of mean FEVI/FVC score according to severity of COPD.*

The intergroup comparison of mean FEV1/FVC score was done among patients with mild, moderate and severe COPD using the One-way ANOVA test. There was a significant difference in mean FEV1/FVC score between mild group and moderate and severe COPD groups and also between moderate group and severe group.

Severity of COPD	FEV1 (Mean ± SD)	p-value
Mild	82.00 ± 20.09	0.964
Moderate	83.35 ± 15.83	
Severe	81.36 ± 17.97	
Very severe	81.33 ± 17.83	

Table 7: *Comparative assessment of mean FEV1 score according to severity of COPD. Test applied: One way ANOVA.*



Graph 7: *Comparative assessment of mean FEV1 score according to severity of COPD.*

The mean FEV1 was compared among patients with mild, moderate and severe COPD using one-way ANOVA test. There was a significant difference in mean FEV1 between mild, moderate and severe COPD groups and also between moderate group and severe group.

Discussion

Research on the relationship between periodontitis and systemic diseases has been given increasing attention over recent decades and established that periodontal infection might be a possible risk factor for various systemic conditions including cardiovascular diseases, diabetes, respiratory diseases, adverse pregnancy outcomes and osteoporosis. An association between COPD and oral health was first noted in community-dwelling population in an analysis of 23,808 individuals in the National Health and Nutrition Examination Survey I (NHANES I) data [34]. The study of Hayes., *et al.* [35] also found that periodontitis measured as alveolar bone loss is an independent risk factor for COPD in adult males enrolled in the VA Normative Aging Study.

Out of the various systemic diseases, an area of particular interest is the link between periodontitis and COPD (Chronic Obstructive Pulmonary Disease). COPD is a chronic respiratory disease with systemic component, affecting a significant segment of the population and is considered as the sixth cause of mortality [16]. Better understanding of this correlation will help both the dental and medical professionals to determine the best approach to patient care.

The findings of the present analysis, together with other recently published studies [Fatemi., *et al.* (2009) [36], Wang., *et al.* (2009) [37], Deo., *et al.* (2009) [17], Kalpak., *et al.* (2013) [38], Benazir., *et al.* (2015) [39]] support an association between poor periodontal health and COPD and suggest that patients having obstructive lung disease had worse periodontal health status. Our findings suggest chronic periodontitis as a potential risk factor for COPD. A significantly higher mean OHI, PI, PPD and CAL was found in individuals with COPD (test group) compared to non-COPD (control group). Similar findings were shown by Nikhil., *et al.* (2011) [40] and Benazir., *et al.* (2015) [39].

Oral Hygiene Index (OHI) scores were found to be more in COPD group than in Non-COPD group (2.95 ± 1.37 and 1.65 ± 0.91 respectively). Furthermore, mean OHI scores were significantly more among COPD group in comparison to the Non-COPD group and difference was found to be statistically significant ($p \leq 0.000$). The result of present study was consistent with the studies of Scannapieco., *et al.* (1998) [34], Deo., *et al.* (2009) [17], Nikhil., *et al.* (2011) [40], Kalpak., *et al.* (2013) [38], Benazir., *et al.* (2014) [39]. This could be explained because of inadequate maintenance of oral hygiene in COPD patients as most of the patients with COPD had never visited a dentist due to high cost of dental treatment and lack of dental health awareness among the poor.

Hyman (2004) [41], Katancik., *et al.* (2005) [42] found that the association of periodontal health status as measured by probing depth to obstructive disease was not statistically significant. Scannapieco., *et al.* (1998) [34] found no associations between the gingivitis or periodontal indices and chronic respiratory disease which may be due to the fact that they used to quantitate periodontal

diseases might be insensitive. The NHANES data combined influenza, pneumonia, and acute bronchitis, however the etiologies of these diseases are totally distinguished from each other. Also, acute disease sample may have precluded detection of statistical significance.

In the present study, the assessment of periodontal destruction was made by measuring loss of attachment on all teeth except third molars. The measurements were done to the nearest millimeter using UNC -15 Periodontal Probe (Hu-Friedy). The clinical attachment loss was defined both clinically and quantitatively as the distance in millimeters from the cemento-enamel junction (CEJ) to the bottom of pocket. The mean CAL of COPD subjects was 3.50 ± 1.68 compared to Non-COPD patients 1.36 ± 1.58 which was statistically significant ($p \leq 0.000$). One possible explanation could be because majority of COPD subjects in our study were smokers and thus may have demonstrated more loss of attachment at the time of examination when compared to controls (subjects without COPD). Similar results were observed in studies conducted by Scannapieco., *et al.* (2001) [4], Fatemi., *et al.* (2009) [36], Deo., *et al.* (2009) [17], Nikhil., *et al.* (2011) [40], Yan., *et al.* (2012) [43], Kalpak (2013) [38] who found that there is significant difference in CAL in patients from COPD and Non-COPD group.

In contrast to our study, Katancik., *et al.* (2005) [42] found that among the current smokers in healthy subjects mean loss of attachment was 3.33 ± 0.25 compared to 2.87 ± 0.46 in current smokers from COPD group which was not statistically significant ($p = 0.195$). However, in our present study, there was a significant difference in the distribution of smoking status between COPD and Non-COPD groups. Former (74.4%) and current smokers (58.1%) were significantly more among COPD group and non-smokers were significantly more Non-COPD group. An association between periodontal health and obstructive disease severity was seen primarily among former smokers. Among former smokers, all periodontal measures were associated with pulmonary disease status.

A distinct trend was noted whereby an increase in the periodontal parameters (OHI, PPD and CAL), was noticed with increase in the severity of the COPD (according to GOLD criteria). In the present study, there was a significant decrease of mean FEV1/FVC score and mean FEV1/FVC score according to the mild, moderate, severe and very severe COPD subjects. It is noteworthy here that correlation exists between the severity of periodontal destruction and mean FEV1/FVC score. There is an inverse relation between periodontal disease and FEV1/ FVC score (mild, moderate, severe and very severe COPD) and also FEV1 score (mild, moderate, severe and very severe COPD) as the subjects with more periodontal destruction had a higher prevalence of diminished lung function and lung volumes based on the mean FEV1/FVC score. Similar results were shown by Scannapieco and Ho (2001) [4], Benazir (2013) [39], Deo., *et al.* (2009) [17].

Results unveil those individuals in the test group had significantly ($P < 0.000$) worse CAL, PPD and OHI compared with the control group. These findings agree with the findings of previous studies [Hayes., *et al.* (1998) [35], Scannapieco., *et al.* (2001) [4] that suggest periodontitis as an independent risk factor for COPD. Dental plaque provides a pool for respiratory pathogen colonization that can be shed into saliva. Periodontitis may be linked to COPD through bacterial species, either by colonization of respiratory pathogens in dental plaque [37], or by periodontal pathogens and their endotoxins that may enhance airway inflammation and exacerbations. Brook's study stated that there is an increase in antibody levels against *Fusobacterium nucleatum* and *Prevotella intermedia* in the sputum of patients with an acute exacerbation of chronic bronchitis [44]. Pavord., *et al.* stated that the coexistence of multiple inflammatory stimuli to the airway might be a primary link leading to the development of more severe airway disease [45].

In both diseases, a host inflammatory response in terms of chronic challenge by microbial species in periodontitis and cigarette smoking in COPD. The neutrophil influx leads to release of oxidative and hydrolytic enzymes that causes tissue destruction directly. Considering the pathogenesis of the periodontal disease and COPD, it seems possible that the inflammatory process underneath the periodontal disease may modify the respiratory epithelium and increase the risk of COPD.

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 unknown, although they are thought to be aggravated by bacterial infection. Therefore, there is an increase in the possibility of deposition of periodontal pathogens associated with periodontitis may increase the risk of lower respiratory tract infection in susceptible individuals, including pneumonia or exacerbation and progression of COPD [14,20].

The majority of pulmonary infections and emphysema cases are the result of aerobic and facultative anaerobic bacteria found in the

oral cavity including various Streptococcus species, various gram-negative bacilli (e.g. *Enterobacteriaceae* such as *Escherichia coli, Klebsiella pneumoniae, Serratia* species and *Enterobacter species*, *Pseudomonas aeruginosa)* and *Staphylococcus aureus* [14]. High salivary concentrations of *P. gingivalis* were reported to enhance the risk of developing respiratory diseases [21]. On other aspect, some of the facultative anaerobes are responsible for periodontal breakdown, such as *Aggregatibacter actinomycetemcomitans, Fusobacterium nucleatum, Pseudomonas aeruginosa*, and *Porphyromonas gingivalis* have been isolated from infected lungs which causes periodontitis and respiratory infection both. Thus, it is biologically accepted statement that periodontal disease and COPD may have a close association with each other.

The association between periodontitis and COPD may have been confounded by various shared factors such as smoking, age, gender, BMI. Smoking is the leading risk factor for periodontitis, emphysema, chronic bronchitis, and lung infections. On one side, smoking is involved in the pathogenesis and progression of periodontal diseases [22]. The peripheral vasoconstrictive effect of tobacco smoke and nicotine reduces the delivery of oxygen and nutrients to the gingival tissue and alters gingival inflammatory response [23]. Cigarette smoking can also impair immunologic function. On the other side, smoking affects respiratory defenses, promotes chronic lung diseases resulting in respiratory infections, damages the airway epithelium, and has effects on lung clearance by suppressing coughing and the protective waving action of cilia in the airways. Cigarette smoking is known to hamper lung clearance by directly suppressing coughing, compromising the protective muco-ciliary action in the airways, and phagocyte activity. Here, there is more damage to the large airways and to the airway epithelium causing high levels of sputum production in patients, especially in the mornings (known as chronic bronchitis) [24]. The mechanism behind this increased risk is poorly understood, but it has been suggested that tobacco use suppresses the production of protective IgG2 antibodies and blocks Phagocytosis and killing of bacteria by neutrophils.

As our study was cross-sectional and non-interventional with no microbiological evaluation, it limits determining a causal relationship between periodontitis and the COPD. The microbiological evaluation could have been done by collecting sputum samples and dental plaque of the subjects with COPD to find out any periodontal pathogen in the lung fluid and quantify the microbial load in both the lung fluid and dental plaque samples. It could have strengthened the study because the increased bacterial load is associated with the initiation of exacerbation that is thought to be provoked in part by bacterial infection. Another limitation of the study is that quantitative changes of specific salivary biomarkers such as CRP could have been done which could have significance in the early diagnosis and management of both oral and systemic diseases. Gan., *et al.* (2004) [25] confirmed a significant increase in CRP levels in COPD patients compared with controls indicating a persistent systemic inflammation in subjects with COPD.

The strength of this study based on several factors. Most importantly, individuals enrolled in the test group were those having only COPD and no other systemic disease that influenced periodontal status. Full-mouth examination was done, and patients who underwent Pulmonary Function Test (PFT) were included in the study. These criteria eliminated the probability of underestimating the true extent of periodontal disease. Furthermore, the most vital strength is the diagnosing of COPD which was measured by using Spirometry and is gold standard for diagnosing COPD.

The available evidence for COPD and its relationship in periodontitis is at an immature level. Several clinical trials demonstrated that improved oral hygiene, either by the use of mechanical or chemical agents (e.g. Povidone Iodine, chlorhexidine gluconate), significantly reduces the rate of lower respiratory tract infection in institutionalized subjects [DeRiso., *et al.* (1996) [26], Yoneyama., *et al.* (1996) [27], Fourrier., *et al.* (2000) [28] Furthermore, it needs to be investigated through clinical trials, whether maintenance therapy of periodontitis might have a positive effect on functioning of lungs.

Multi-center randomized studies are needed to be done in different areas providing different environment to the patients to avoid the residual confounding by the other factors like environmental pollutants. This can help to clearly establish the association between COPD and periodontitis. Additional longitudinal epidemiologic studies are required to validate the reported association between periodontal disease and COPD. Future studies on the relationship of periodontal and systemic diseases ought to include a separate analysis for never smokers and should be conducted on a variety of populations to validate the observed associations. Studies of the association between periodontal disease and exacerbations of COPD would be valuable.

Conclusion

Although the causal relationship cannot be established between both the diseases because of the observational design of the study, but due to the absence of any other risk factor and any other systemic disease in the patients, the study provides substantial evidence that poor periodontal health is associated with obstructive lung disease. Chronic obstructive pulmonary disease (COPD) and periodontitis frequently shared a common feature of abnormal neutrophil responses, but this may reflect common risk factors of disease (smoking, age and socio-economic status) rather than shared pathophysiology. Improving oral habits appeared to be associated with a reduction in COPD exacerbations.

Evaluation and education may improve the oral hygiene in COPD as in these cases the burden of periodontitis is at peak level. Altered neutrophil functions are implicated in both disease processes. Further investigations with longitudinal data are required to address the relation between periodontitis and COPD. Professional and patient level interventions to improve oral health status may prove to lower the severity of lung infection in susceptible populations.

To conclude, the causal relation of these two inflammatory diseases once identified, the treatment protocol could be of an interdisciplinary approach in an effort to prevent the progression of both the diseases.

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