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Original Research Article.

Evaluation of the Association between Periodontal Disease and Chronic Obstructive Pulmonary Disease: A Cross Sectional Study

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ABSTRACT

Background: A growing body of evidences proves an association between Periodontitis and various systemic diseases; metastatic infection, inflammation and injury being the connecting link. This cross-sectional study aims to evaluate the periodontal status in Chronic Obstructive Pulmonary Disease (COPD) patients and to determine the association between the two diseases.

Method: Study was conducted on 136 COPD patients and 66 healthy controls. Age, sex, history of smoking, tuberculosis and any allergy were recorded. The number of remaining teeth, Probing Pocket Depth (PPD), Clinical Attachment Level (CAL), Gingival Bleeding Index (Lonox and Kopczyk) and Periodontal Disease Index (Ramfjord's) were recorded. Oral hygiene was assessed using Simplified Oral Hygiene Index (OHI-S by Greene and Vermillion). Pulmonary status (FEV1, FVE, FEV1/FVC ratio) was evaluated using spirometry.

Results: A highly significant ($p<0.001$) difference was found between mean CAL of test and control group and the difference was significant ($p<0.05$) with respect to mean PDI scores. Severity of COPD was found to be significantly correlated with the severity of periodontal disease. A highly significant difference was found in mean CAL of current smoker COPD patients as compared to controls. Subjects with more than 3mm mean CAL were at more risk of developing COPD. Subjects with good oral hygiene were at one third risk of developing COPD which was significant ($p=0.02$).

Conclusion: Poor periodontal health is associated with the presence and severity of airway obstruction in COPD patients. Smoking also seems to accentuate the effect of periodontal disease in these patients.

Key words: Periodontitis, COPD, Smoking, Effect Modifier, Dental Hygiene, Risk Factor(s).

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INTRODUCTION

Periodontitis and COPD are host mediated diseases sharing a common pathogenic pathway. Both are chronic inflammatory diseases caused by anaerobic microorganisms. Inflammatory and infectious burden due to periodontal disease released in circulation recruit inflammatory cells which release various hydrolytic enzymes causing damage to respiratory epithelium. COPD may contribute to poor oral health via direct and indirect mechanisms. It may lead to hypoxic state that affects the entire body including oral mucosa causing xerostomia.[1]

It also leads to compromised dexterity to perform oral hygiene; thereby indirectly compromising oral health.[2] Scannapieco et al (1998) reported an association between poor periodontal health and an increased risk of COPD.[3] There seems to be a paucity of literature regarding the association between periodontal disease and COPD. Hence, this study was conceived to fill in the breach. The present study aimed to evaluate the periodontal status in COPD patients and to determine the possible association between periodontal disease and the presence & severity of airway obstruction.

MATERIALS AND METHODS

The following study was a cross-sectional (observational) study which was conducted on 136 COPD patients who reported to the Department of Medicine, Maharaja Yashwant Rao Hospital, Indore. 66 systemically healthy accompanying persons with normal lung function were taken as controls. The patients who

visited the hospital from September 2010 to December 2010 were included in the study. Sample size was selected based on a time period as suggested by statistician. This study was approved by institutional ethical committee.

Patients diagnosed as having COPD according to Global Initiative for Chronic Obstructive Lung Disease guidelines (GOLD), between 30-70 years of age, with at least 10 natural teeth present and patients willing to take part in the study were included. Patients suffering from any other systemic illness, who had undergone any invasive chest or abdominal surgical procedure during last 12 months, having a history of myocardial infarction or any other cardiac problem, who had undergone dental treatment within last 6 months prior to study and non-ambulatory or severely ill patients were excluded from the study. Informed consent was obtained from all the subjects who were included in the study.

Demographic data i.e. age, sex, socioeconomic status, occupation and oral hygiene habits were noted. History of smoking, tuberculosis and any allergy were also recorded. The number of remaining teeth, Probing Pocket Depth (PPD), Clinical Attachment Level (CAL), Gingival Bleeding Index (Lonox and Kopczyk – binary system of scoring)[4] and Periodontal Disease Index (Ramfjord)[5] were recorded on four sites of six Ramfjord's index teeth. Oral hygiene was assessed using Simplified Oral Hygiene Index (OHI-S by Greene and Vermillion).[5] According to the smoking status, patients were categorized into current, former and non-smokers according to Centers for Disease Control and Prevention (CDC) criteria.[6] All the tests were performed by a single examiner to avoid the inter-examiner variability.

Lung function was evaluated by Spirometry. PC (personal computer) based Medi Soft 13L dry rolling seal spirometer was used. Spirometry was performed by an expert spirometer technician. Diagnosis and severity of COPD were assessed measuring Forced Expiratory Volume in One Second (FEV_1), Forced Vital Capacity (FVC) and $FEV_1/FVC \times 100$ (the ratio of FEV_1 to FVC expressed as a fraction). Classification of patients into different groups (mild, moderate, severe and very severe) was done under the guidance of an expert Chest and TB specialist according to the Gold spirometric criteria.[7]

RESULTS

Univariate analysis of demographic characteristics & periodontal parameters in COPD and control groups has been depicted in table 1. Comparison of periodontal parameters between Test and Control groups has been shown in table 2. Table 3 explains Comparison of periodontal parameters in mild, moderate and severe COPD and control groups. Effect of smoking on periodontal parameters in test and control groups has been demonstrated in table 4. Table 5 depicts the correlation between mean OHI-S score & mean CAL and mean OHI-S score & mean PDI score in COPD and control patients with different smoking behaviours. Odds Ratios for various periodontal parameters and smoking behavior has been demonstrated in table 6.

Table 1: Univariate analysis of demographic characteristics & periodontal parameters in COPD and control groups (Chi-square (χ^2) test)

Parameters	COPD group (n = 136)	Control group (n = 66)	p-value
Age (years) (Mean ± Standard deviation)	49.2 ± 11.7	52.7 ± 9.7	0.27
Gender			
Male	88 (64.7 %)	34 (51.5%)	0.06
Female	48 (35.3 %)	32 (48.5%)	
Smoking status			
Current Smokers	38 (27.9%)	13 (19.7%)	<0.001**
Former smokers	38 (27.9%)	8 (12.0%)	
Non-smokers	60 (44.2%)	45 (70.3%)	
Number of remaining teeth	26.94 ± 6.8	26.68 ± 5.1	0.54
Mean Probing Pocket Depth- PPD (mm)			
< 3.0 mm	134 (98.5%)	66 (100.0%)	0.22
> 3.0 mm	2 (1.5%)	0 (0.0%)	
Mean Clinical Attachment Loss – CAL (mm)			
≤ 3 mm	91(66.9%)	60 (91.0%)	<0.001**
3.0 mm – 4.0 mm	29 (21.3%)	3 (4.5%)	
> 4 mm	16 (11.8%)	3 (4.5%)	
Bleeding on Probing- BOP (% sites)			
< 20 % sites	56 (41.2%)	29 (43.9%)	0.70
> 20 % sites	80 (58.8%)	37 (56.1%)	
Simplified-Oral Hygiene Index (OHI-S) Score			
Good (0 - 1.2)	6 (4.4%)	10 (15.2%)	<0.001**
Fair (1.3 – 3.0)	49 (36.0%)	32 (48.5%)	
Poor (3.1 – 6.0)	81 (59.6%)	24 (36.3%)	
Periodontal Disease Index (PDI) Score			
0 - 2.0	33 (24.3%)	21 (31.8%)	0.32
2.1 – 4.0	73 (53.7%)	35 (53.0%)	
4.1 – 6.0	30 (22.0%)	10 (15.2%)	

** Highly Significant (p < 0.001)

Table 2: Comparison of periodontal parameters between Test and Control groups (Standard unpaired 't' test)

Group	Mean no. of remaining teeth	Mean OHI-S score	Mean CAL (mm)	Mean PDI score
Control (66)	26.68 ± 5.1	2.78 ± 1.3	1.05 ± 0.7	2.54 ± 0.35
Test group (136)	26.94 ± 6.8	2.83 ± 1.2	1.44 ± 0.8	2.66 ± 0.9
t value	0.62	1.79	3.45	2.19
p-value	0.54	0.07	0.0008**	0.03*

*Significant ($p < 0.05$) ** Highly significant ($p < 0.001$)

Table 3: Comparison of periodontal parameters in mild, moderate and severe COPD & control groups (ANOVA test [Analysis of Variance])

Group		Frequency	Mean No. of remaining teeth	Mean OHI-S score	Mean CAL (mm)	Mean PDI score
Control		66 (100.0)	26.68 ± 5.1	2.78 ± 1.3	1.05 ± 0.7	2.54 ± 0.35
Test group	**Mild COPD**	50 (36.8)	26.94 ± 6.8	2.83 ± 0.86	1.44 ± 0.80	2.66 ± 1.26
	Moderate COPD	61 (44.9)	26.15 ± 6.9	3.07 ± 1.16	1.70 ± 0.85	2.90 ± 1.46
	Severe COPD	25 (18.3)	24.28 ± 6.9	3.65 ± 0.95	2.88 ± 1.01	3.92 ± 0.82
F value			2.13	4.1	10.26	7.08
p-value			0.049*	0.008**	0.000**	0.0002**
CD value			1.25	0.22	0.30	0.28

*Significant ($p < 0.05$) ** Highly significant ($p < 0.001$)
CD - Critical Difference

Table 4: Effect of smoking on periodontal parameters in test and control groups (Unpaired 't' test)

Parameters	Smoking behaviour	Control group	Test group	t value	p-value
Mean no. of remaining teeth	**Non-Smokers**	27.4 ± 4.65	27.5±5.4	0.24	0.81
	Former-smokers	22.5±6.14	22.8±7.4	0.05	0.96
	Current smokers	27.5±5.24	26.6±8.1	0.25	0.80
Mean OHI-S score	**Non-Smokers**	2.56±1.24	2.98±1.03	1.62	0.11
	Former-smokers	3.50±0.79	3.61±1.0	0.46	0.65
	Current smokers	2.99±1.39	3.06±1.16	0.39	0.70
Mean CAL (mm)	**Non-Smokers**	0.98±0.29	1.62±0.54	1.59	0.11
	Former-smokers	1.85±0.81	2.67±0.98	1.19	0.24
	Current smokers	0.64±0.36	2.42±1.1	3.83	0.000**
Mean PDI score	**Non-Smokers**	2.33±1.45	2.82±1.2	1.21	0.23
	Former-smokers	3.45±1.29	3.66±1.38	0.28	0.80
	Current smokers	2.59±1.41	3.39±1.34	1.76	0.08

Effect of smoking on periodontal parameters in test and control groups (Applying F test)

Groups	Smoking behavior	Mean no. of remaining teeth	Mean OHI-S score	Mean CAL (mm)	Mean PDI score
Control	**Non-Smokers**	27.4 ± 4.65	2.56±1.24	0.98±0.29	2.33±1.45
	Former-smokers	22.5±6.14	3.50±0.79	1.85±0.81	3.45±1.29
	Current smokers	27.5±5.24	2.99±1.39	0.64±0.36	2.59±1.41
Test group	**Non-Smokers**	27.5±5.4	2.98±1.03	1.62±0.54	2.82±1.2
	Former-smokers	22.8±7.4	3.61±1.0	2.67±0.98	3.66±1.38
	Current smokers	26.6±8.1	3.06±1.16	2.42±1.1	3.39±1.34
F value		4.71	4.50	9.14	5.56
p-value		0.000**	0.001**	0.000**	0.000**
CD value		1.25	0.22	0.30	0.28

** Highly significant ($p < 0.001$)

Table 5: Correlation between mean OHI-S scores & mean CAL and mean OHI-S score & mean PDI score in COPD and control patients with different smoking behaviours

Smoking behaviour	Parameters (mean)	Control group			COPD group		
		Calculated value (r)	Table value (df = n-2) r at 5%	r at 1%	Calculated value (r)	Table value (df = n-2) r at 5%	r at 1%
Non-smokers	OHI-S & CAL	0.69**	0.30	0.39	0.62**	0.26	0.34
	OHI-S & PDI	0.88**	0.30	0.39	0.70**	0.26	0.34
Former smokers	OHI-S & CAL	0.20	0.71	0.83	0.16	0.32	0.41
	OHI-S & PDI	0.48	0.71	0.83	0.32	0.32	0.41
Current smokers	OHI-S & CAL	0.50	0.55	0.68	0.50**	0.32	0.41
	OHI-S & PDI	0.38	0.55	0.68	0.78**	0.32	0.41

* Significant ($p < 0.05$) ** Highly significant ($p < 0.001$)
df - Degree of Freedom

Table 6: Odds Ratios for various periodontal parameters and smoking behaviour

Periodontal parameter		Odds Ratio	Confidence Interval	p-value
Simplified Oral Hygiene Index score	Good	0.29	0.10-0.84	0.02*
	Fair	0.74	0.44-1.21	0.28
	Poor	1.64	0.95-2.82	0.07
Periodontal Disease Index score	0 – 2	0.76	0.41-1.42	0.38
	2.1 – 4	1.01	0.61-1.67	0.96
	4.1 – 6	1.46	0.67-3.16	0.34
Clinical Attachment Loss (mm)	< 2.9 mm	0.74	0.47-1.14	0.17
	3 - 4 mm	4.69	1.38-15.96	0.01*
	> 4.1 mm	2.59	0.73-9.2	0.01*
Smoking behaviour	Non-smoker	0.65	0.4-1.05	0.08
	Former smoker	2.31	1.02-5.23	0.05*
	Current smoker	1.42	0.71-2.84	0.32

*Significant ($p < 0.05$)

DISCUSSION

The present cross-sectional study was conducted on 136 COPD patients and 66 healthy controls to evaluate the periodontal status of COPD patients, to study the association between periodontal disease and COPD and also to ascertain the association between their severities.

There was no statistical difference in age and gender distributions in both COPD and control groups, bias because of these confounding factors were nullified (Table1).

COPD patients were divided into mild, moderate, severe and very severe categories according to GOLD guidelines.[7] Only one patient was suffering from very severe COPD. As the statistical tools could not be applied due to limited data, this patient was included under severe COPD category only. Therefore, in the present study, COPD patients were categorized into three groups i.e. mild, moderate and severe.

Number of Remaining Teeth

Mean number of remaining teeth present in COPD group and control group was 26.94 ± 6.8 and 26.68 ± 5.1 respectively (Table 1). The difference was statistically non-significant at $p = 0.54$. This was in contrast with the findings of Wang et al (2009)[8] who found significantly lesser number of remaining teeth in COPD group as compared to control group (21.5 ± 6.27 and 23.07 ± 5.63 respectively). Difference in the mean number of teeth lost in these two studies can be attributed to a difference in mean age of the patients included in both the studies. Mean age of subjects in the study of Wang et al [8] was 63.94 ± 9.84 years and 63.26 ± 8.98 years in test and control group respectively. Prevalence and severity of periodontal disease increase with age resulting in more tooth loss.[9] Increased tooth loss in Wang's study can be attributed to increased mean age of the patients.

In the present study, there was a statistically significant decrease in the number of remaining teeth with increasing severity of COPD indicating that disease severity of COPD was significantly associated with loss of more number of teeth (due to periodontal disease) (Table 3). As tooth loss can act as an indicator of advanced periodontal disease, this leads to the conclusion that subjects with severe COPD suffer from more advanced periodontal disease and strength of association between periodontal disease and COPD increases with the increasing severity of both diseases.

Holm et al (1994)[10] showed that smoking is a risk factor for tooth loss. In our study, only former smokers showed statistically significant less number of remaining teeth as compared to current smokers (Table 4). Variations in the pack year history, frequency of smoking and age of former smokers could have contributed to greater loss of teeth in former smokers than current smokers or non-smokers. Comparable mean number of remaining teeth were reported in non-smokers, former smokers and current smokers in both COPD and control groups. Scannapieco et al (1998)[3] found that non-smokers with COPD had significantly fewer permanent teeth than non-smokers without COPD. This difference can be

attributed to disparity in grouping of patients depending on their smoking status. Scannapieco et al divided the patients into 2 groups i.e. smokers and non-smokers but in our study, the subjects were grouped into 3 groups i.e. non-smokers, former smokers and current smokers.

Oral Hygiene Index Scores

In the present study, mean OHI-S score of COPD group (2.86 ± 1.2) was greater than that of control group (2.78 ± 1.3), though this difference was statistically non-significant (p = 0.07) (Table 2). Statistically significant results were obtained by Scannapieco et al (1998)[3] and Deo et al (2009)[11] who showed significant differences in mean OHI-S score in COPD and control group. These results reveal that COPD patients have poor oral hygiene than non-COPD patients.

In the present study, with an increase in severity of COPD, there was a statistically highly significant increase in mean OHI-S score. These results indicate that subjects with COPD were not able to maintain their oral hygiene due to compromised dexterity because of increased disease severity. According to Eisner et al (2008),[12] COPD has a major impact on physical health limitation. They stated that COPD was associated with decrement in mean muscle strength of 17% for elbow flexion and 10% for grip strength. This compromised physical strength may lead to decreased dexterity of patients to perform oral hygiene. Due to illness and lack of attention, patients are unable to perform oral hygiene.[2] Negligence of oral hygiene as a result of greater concern to COPD also leads to an increase in the mass and complexity of dental plaque which may foster inter-bacterial interactions between indigenous plaque bacteria and respiratory pathogens such as P. aeruginosa and enteric bacilli.[2] These interactions not only increase the aspiration of respiratory pathogens to lungs but also provide a continuous influx of systemic mediators of inflammation. This leads to damage and alteration of respiratory epithelium and facilitates pathogenic mechanisms resulting in the development of COPD.[2]

Statistically significant differences in mean OHI-S scores were observed when moderate COPD patients were compared to controls but when severe COPD patients were compared to controls, highly significant difference were observed (Table 3). This leads to the inference that oral hygiene status gets more compromised with an increased severity of COPD.

Medications prescribed for patients with COPD can indirectly affect the periodontium and oral cavity. Some inhalational medicaments can reduce pH of the dental plaque. Anticholinergic drugs, adrenergic drugs and antihistaminics can cause xerostomia leading to more plaque formation, increased risk of developing caries, gingivitis and candidiasis.[1] Inhaled and systemic corticosteroids can cause immunosuppression. Long term use of corticosteroids can also lead to myopathy[7] which contributes to muscle weakness and compromised dexterity leading to poor oral hygiene.

In the present study, Odds Ratio (OR) for good oral hygiene (score 0 - 1.2) was 0.29 (Table 6), which indicates that subjects with good oral hygiene were at one third risk of developing COPD which was statistically significant at p = 0.02. The subjects with poor oral hygiene (score 3.1 - 6) were 1.64 times at greater risk of developing COPD which was statistically not significant at p = 0.07. Scannapieco et al (1998)[3] also showed that subjects having median OHI value were 1.3 times more likely to have a chronic respiratory disease relative to those with an OHI of 0 value. They also showed that subjects having maximum OHI values were 4.5 times more likely to have COPD than those with an OHI of 0. They concluded that oral hygiene was found to be significantly associated with COPD.[3] These studies also indicate and enforce the importance of promoting dental care and oral health knowledge in the prevention and treatment of COPD.

Bleeding on Probing (% Sites)

In the present study, the percentage of sites with BOP was calculated. 41.2% of COPD subjects showed BOP in ≤ 20% of sites as compared to 43.9% subjects in control group. In COPD group, 58.8% of subjects showed BOP in > 20% of sites as compared to 56.1% of subjects in control group (Table 1). There was statistically no significant difference in BOP in both test and control groups in the present study at p = 0.70. This was contrary to the study conducted by Deo et al (2009)[11] who demonstrated that 96% of COPD subjects showed gingival bleeding present in >20% of sites as compared to 88% of control subjects (statistically significant at p < 0.05.) Prasanna SJ (2011)[13] also revealed that Gingival Index (GI) and Papilla Bleeding Index (PBI) scores were highly significant in COPD group than controls. Katancik et al (2005)[14] also showed that gingival index (GI) was significantly better in subjects with normal lung function. This difference can be attributed to the differences in OHI-S scores of the subjects in the present study and study by Deo et al (2009).[11] In the present study, mean OHI-S score of COPD group (2.86 ± 1.2) was greater than that of control group (2.78 ± 1.3). However, this difference being statistically non-significant (p = 0.07) could have contributed to non-significance in BOP scores. Deo et al (2009)[11] showed mean OHI-S score of 3.37 ± 0.48 and 2.87 ± 0.64 in COPD and control group respectively (difference being statistically significant at p < 0.05). This could have contributed to difference in statistically significant higher bleeding on probing scores.

Probing Pocket Depth (PPD)

When PPD was compared in test and control group, statistically no significant difference was found (98.5% of COPD subjects showed mean PPD ≤ 3mm as compared to 100% of subjects in control group) (Table 1). This was in accordance with the study conducted by Wang et al (2009)[8] in which mean PPD was 3.12 ± 0.72mm and 3.2 ± 0.76mm at p = 0.146 in test and control group respectively. They also compared percentage of sites having PPD ≥ 4 mm and ≥ 5 mm. There was no statistically significant difference at p = 0.109. Katancik et al (2005)[14] also showed no significant difference between test and control group with respect to PPD at p = 0.13.

Clinical Attachment Loss (CAL)

Significantly more number of COPD subjects suffered from moderate and severe clinical attachment loss (Table 1) As CAL is considered as 'Gold Standard' in assessing periodontal destruction, this points towards more severe periodontal disease in COPD patients. Majority of control subjects had milder CAL scores.

COPD patients had greater mean CAL than the controls (1.44 ± 0.8mm in COPD group and 1.05 ± 0.7mm in control group) (Table 2). This difference was statistically highly significant at p = 0.0008) which indicates a more advanced periodontal disease of periodontal disease in COPD patients. Deo et al (2009)[11], Wang et al (2009)[8] and Katancik et al (2005)[14] also reported statistically higher mean CAL in COPD subjects than control group. Mean

CAL scores in present study were lower than those reported by previous studies by Deo et al (2009)[11] and Wang et al (2009).[8] The presence of more number of smokers and poorer oral hygiene of the subjects included in Deo's study may account for this difference. Disparity in the mean age of the patients included in Wang's study may be responsible for this difference. With an increase in age, cumulative effect of local irritants leads to higher loss of attachment.[9]

In our study, a statistically highly significant increase in CAL with the increasing severity of COPD was observed (Table 3). Similar results were also reported by Deo et al (2009)[11] who stated that lung function appeared to diminish as the amount of attachment loss increased (i.e. > 3.5 mm). Katancik et al (2005)[14] also reported significantly less loss of attachment in the subjects with normal pulmonary function. They also showed that with an increase in severity of COPD from mild to moderate and then to severe stage, there was statistically significant increase in mean LOA (loss of attachment). These findings suggest that severity of COPD is significantly associated with worsening of periodontal health. Immunosuppression caused by the disease per se or pharmacodynamics of the drugs prescribed to COPD patients could have triggered these effects. Corticosteroid use may also lead to osteoporosis[15] which may increase the vulnerability to periodontal disease through modification of alveolar and trabecular bone tissue.[16]

When the effect of smoking on CAL in both test and control groups was compared, only current smokers showed statistically highly significant difference of mean CAL (Table 4). This implies that smoking has an additive effect on severity and progression of periodontal disease in COPD patients. This was in accordance with the findings of Hyman et al (2004)[17] who used Logistic regression models (stratified for smoking status) and showed that significant increase in the risk for COPD occurred only among current smokers with very severe periodontal disease (≥ 4 mm of Loss of Attachment [LOA]). When smoking was considered as an effect modifier, all individuals with mean LOA of more than 4 mm were found to be at an increased risk for COPD. (Confounding is a bias that the investigator hopes to prevent or remove from the effect estimate whereas effect modification is a property of the effect under study. Thus, effect modification is a finding to be reported, rather than bias to be avoided.)[17] Hyman et al (2004)[17] considered smoking as an effect modifier and concluded that much of the observed increase in risk may actually reflect the exposure to smoking. In our study too, former smokers were found to be at higher risk of developing COPD with Odds Ratio of 2.31 ($p = 0.045$) (Table 6).

Garcia et al (2001)[18] analyzed the role of smoking as a confounding factor in the association between periodontitis and risk of COPD. They found that worse periodontal status significantly increased the risk of COPD in subjects who were current smokers but no such association was found in non-smokers.

Comparable mean CAL was reported in non-smokers and former smokers in both COPD and control groups (Table 4). Similar findings were noted by Hyman et al (2004).[17] They stated that smoking is known to be an independent risk factor for both the diseases while periodontal disease appears to be a risk factor only when there is a history of cigarette smoking. The results of these studies were in contrast with the study of Deo et al (2009)[11] who noted that among the non-smokers of both test and control groups, the mean CAL was significantly greater in test group (4.01 ± 0.36) than in control group (3.77 ± 0.44) ($p < 0.05$). This disparity in the results may be due to the difference in categorization of smokers. Deo et al (2009)[11] grouped subjects as smokers and non-smokers whereas in our study, subjects were categorized as non-smokers, former smokers and current smokers.

When the effect of smoking was considered, in both COPD and control groups individually, former smokers in both COPD and control group showed a higher CAL than non-smokers and current smokers. This difference was statistically highly significant. In both test and control groups, current smokers were found to have statistically significant higher mean CAL as compared to non-smokers (Table 4).

In the present study, a highly significant positive correlation was found among non-smokers was found between mean OHI-S score and mean CAL in both test and control groups (Table 5). In former smokers, the correlation between mean OHI-S score and mean CAL was found to be non-significant in both test and control group. In current smokers, statistically highly significant positive correlation was found in COPD group (COPD has important effect on oral hygiene) whereas in control group, correlation between mean OHI-S score and mean CAL was found to be statistically non-significant. These results indicate that in non-smokers, in both test and control group, poor oral hygiene predominantly is responsible for the initiation and progression of periodontal disease. In current smokers of control group and former smokers of both groups, smoking acts as a potential effect modifier for periodontal disease. In former smokers, smoking may have already established its damaging effects on periodontium before these patients had quit smoking. In current smokers, effects of smoking were overshadowed by poor oral hygiene maintenance in COPD patients.

In the present study, Odds Ratio for mean CAL 3 - 4 mm and > 4 mm was 4.69 and 2.59 respectively. This suggests that subjects with mean CAL of more than 3mm showed significantly increased risk (2.59 - 4.69 times) for developing COPD (Table 6). Deo et al (2009)[11] also stated that subjects with mean attachment loss of > 4.5mm were at 1.1 times higher risk for developing COPD. Scannapieco et al (2001)[19] also reported that subjects with mean attachment loss > 3mm had a higher risk of developing COPD than those having mean attachment loss < 3mm with OR = 1.45. All these results indicate that periodontal disease can act as a risk factor for the development of COPD.

Periodontal Disease Index (PDI)

When mean PDI scores of COPD and control groups were compared, statistically significant difference was observed (Table 2). This indicates a higher prevalence of periodontal disease in COPD patients. With the increase in severity of COPD, there was highly significant increase in mean PDI scores (Table 3). Statistically non-significant differences in mean PDI scores were reported in non-smokers, former smokers and current smokers in both test and control groups (Table 4).

A highly significant positive correlation was found between mean OHI-S scores and mean PDI score in non-smokers in both test and control groups. No significant correlation was found between mean OHI-S scores and mean PDI score in former smokers in both COPD and control groups (Table 5). These results indicate

that smoking has a more pronounced effect on periodontal health in former smokers. A highly significant positive correlation was found between mean OHI-S score and mean PDI score in current smokers in COPD group. A statistically non-significant correlation was found between mean OHI-S score and mean PDI score in current smokers in control group (Table 5).

In the present study, Odds ratios (OR) of subjects with mean PDI score of 0 - 2, 2.1- 4 and 4.1 - 6 were 0.76, 1.01 and 1.46 respectively. Patients with mean PDI score 0 - 2 were at 0.76 (p = 0.38) times lower risk for developing COPD than controls. Patients with mean PDI score 2. 1- 4 were at 1.01 (p = 0.92) times higher risk for developing COPD than controls. Patients with mean PDI score 4.1 - 2 were at 1.46 (p = 0.34) times more risk for developing COPD than controls (Table 6). In the present study, with the increase in mean PDI scores there was a corresponding increase in risk of developing COPD though this was found to be statistically non- significant. Garcia et al (2001)[18] also reported that worse periodontal health status was associated with an increased risk of developing COPD with OR of 1.75.

In the present study, there were statistically significant differences in mean CAL and mean PDI scores of test and control groups but comparable scores have been found with respect to OHI and number of remaining teeth. There was a statistically highly significant increase in mean CAL, OHI-S and PDI scores with the corresponding increase in severity of COPD in test as compared to control group. The number of remaining teeth also decreased significantly with the increase in COPD severity. There was a statistically highly significant difference in mean CAL in current smokers in test group as compared to control group. Former smokers were found to have statistically significant lesser number of remaining teeth and higher mean OHI-S, CAL and PDI scores than non-smokers and current smokers in both test and control groups. From the above findings, it can be deciphered that poor periodontal health is associated with the presence and severity of airway obstruction in COPD patients. Smoking also seems to accentuate the effect of periodontal disease in these patients.

SUMMARY AND CONCLUSIONS

The current study provides evidence that COPD patients suffer from poorer periodontal health status. This study also indicates a positive association between the occurrence and severities of periodontal disease and COPD and infers that periodontal disease may act as a risk factor for the development and severity of COPD. Longitudinal epidemiologic studies and randomized controlled interventional trials will be required for validation and establishment of a definitive cause and effect relationship between the two diseases and to confirm the biologic basis of these epidemiologic associations. Although evidence is indirect, our results lend support to the importance of oral health and education in the management of COPD patients. Therefore, promoting oral hygiene for the prevention of initiation and progression of COPD is strongly recommended.

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