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Research Article

A cross-sectional study to study relationship between periodontitis and chronic obstructive pulmonary diseases (COPD)

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Abstract

Background: Recent studies have suggested increased prevalence of periodontal diseases in chronic obstructive pulmonary disease (COPD) patients. This study was conducted to assess the association between periodontitis and chronic obstructive pulmonary Diseases.

Materials and Methods: The patients were in the age group of 30–60 years, of whom 60 patients belonged to group C (COPD group) and 60 age- and gender-matched group NC (Non-COPD group). Patients with COPD were classified as per GOLD classification into mild, moderate, severe and very severe category. Periodontal health was assessed by measuring plaque index (PI), oral hygiene index (OHI), probing pocket depth (PPD), clinical attachment loss (CAL) and gingival index (GI).

Results: Periodontal indices (PI, GI, OHI, CAL and PPD) were significantly ($P < 0.001$) worse in the COPD group compared with the non-COPD group. However, there was no significant difference in the mean BPI value of the two groups. Also, lung functions (FEV1/FVC %) appeared to worsen linearly as the amount of attachment loss increased.

Conclusion: Our study provides the substantial evidence that poor periodontal health is common and worse in patients with COPD.

Keywords: Periodontal disease, chronic obstructive pulmonary disease, lung function, attachment loss

1. Introduction

Chronic obstructive pulmonary disease (COPD) is a syndrome of progressive airflow limitation caused by chronic inflammation of the airways and lung parenchyma. COPD, one of the most common and costly respiratory diseases, is a major cause of morbidity and mortality worldwide. It has been found that COPD affects a large number of 174.5 million population across the globe and responsible for over 3 million deaths each year [1, 2].

COPD commonly appears to be associated with other comorbidities that are thought to share an underlying inflammatory process, systemic or organ-specific, including a chronic inflammatory oral disease such as periodontal disease [2]. Shi et al conducted a meta-analysis finding that COPD is related with several symptoms of periodontal disease including, lower number of remaining teeth, deeper periodontal pockets, more gingival bleeding and inflammation, and worse oral hygiene [3, 3].

The association between periodontitis and COPD has been increasingly recognized over the last two decades. As reviewed by Azarpazhooh & Leake, four cross-sectional studies suggested an association between poor oral health (including alveolar bone loss, periodontal attachment loss, oral hygiene index, and oral plaque colonization) and chronic pulmonary disease [4].

There was an agreed upon association of periodontitis with COPD even after adjustment for smoking status [5]. Katancik conducted a study among elderly patients with finding that the periodontal indices among previously active smokers had association with pulmonary diseases [6]. Also, has been reported that the present smokers having ≥4 mm loss of attachment had a higher risk for COPD [7].

The measures showing the poor level of periodontal health, such as the plaque index, alveolar bone loss, and dental care, were having high COPD risk as has been reported recently [8, 9]. One of the meta-analysis of fourteen observational stud-

ies has demonstrated that periodontal disease is a major and sole risk factor for the occurrence of COPD [10]. Many current observational studies have shown a positive connection between periodontitis and aggravation of COPD [11, 12].

An important arena of research is the possible connection of chronic obstructive pulmonary disease (COPD) with 'chronic periodontitis' [13]. The mechanism involved includes the oral elements getting aspirated into trachea-bronchial tree, overflowing of cells produced during the inflammation and cell mediators into the circulation together with either oral cavity or lung-derived blood circulating bacteria which stimulates an acute-phase reaction. Moreover, cytokines and other mediators or substances like oxygen-free radicals that may be released by polymorphs at distant sites also have an essential role [14].

The information available support the periodontitis and COPD relationship, increasing the likelihood that dealing one disorder can affect the other regarding its progression and severity. The resemblances in both processes (e.g. chronic neutrophilic inflammation, neutrophil dysfunction, and loss of connective tissue) propose a common pathophysiology and favor the suggestion of association [15].

There is limited data on the association of COPD and periodontal disease from India. This cross-sectional observational study was done to study the relationship between periodontal disease and COPD.

2. Materials and Methods

2.1. Sample Population

In this cross-sectional study, 120 patients were selected on the basis of the following inclusion and exclusion criteria from the periodontics department and department of medicine, Baghi hospital. An ethical clearance was obtained before conducting the study from the institutional ethical committee. The informed written consent was taken from the patients.

The patients were in the age group of 45–55 years, of whom 45 patients belonged to group A (COPD group) and 45 belonged to group B (Non-COPD) which were age- and gender-matched.

2.2. Inclusion Criteria

- Confirmed diagnosis of COPD on clinical criteria and spirometery
- Dentate subjects with teeth ≥16
- Age 45-55 years.

2.3. Exclusion criteria

- If patients had undergone periodontal therapy for last 6 months.
- Subjects with any other systemic diseases which can influence the probability of having periodontal diseases.
- Patients on medications (antibiotics) known to influence the periodontal tissues for last 4 weeks.

2.4. Clinical examination

Clinical history and physical and periodontal examination were done for all the subjects. Pulmonary function test (PFT) using Spirometer was done to diagnose the presence or absence of COPD. Information of lung function in the test group was estimated by calculating the ratio of forced expiratory volume after 1 second (FEV1)/Forced Vital Capacity (FVC) × 100 by trained respiratory technicians. Patients with COPD as per GOLD classification into mild (FEV1≥ 80% predicted), moderate (50% ≤ FEV1 < 80% predicted), severe (30% ≤ FEV1 < 50% predicted) and very severe category (FEV1 < 30% predicted) on the basis of spirometry.

Periodontal examination was done with the following clinical parameters: Plaque index (PI), Gingival index (GI), Probing pocket depth (PPD), Clinical attachment loss (CAL) and Oral hygiene index (OHI).

2.5. Statistical analysis

The software used for the statistical analysis were SPSS (statistical package for social sciences) version 25.0. The categorical variables are presented as absolute numbers and percentage. Mean and standard deviation was calculated for the continuous variables. The Shapiro-Wilk (SW) test was used for checking the normality distribution.

Mann-whitney U test was used for comparison of mean value between 2 groups and kruskal-wallis test for comparison of difference between mean values of more than 2 groups. The p-value of less than 0.05 was taken as significant.

3. Results

The study subjects were comparable with respect to the baseline demographic variables like age, gender, marital status and oral hygiene practices. (Table 1) The subjects with COPD had significantly higher Pocket probing depth and Clinical attachment loss score among the COPD group compared to the non-COPD group though no difference could be seen in the Gingival, Plaque and Oral hygiene indices. (Table 2)

Table 1: Basic demographic data of the two groups.

		COPD	Non-COPD	Total
Gender	Male	45 (75.0%)	50 (83.3%)	95 (79.2%)
	Female	15 (25.0%)	10 (16.7%)	25 (20.8%)
Marital status	Married	51(85.0%)	53 (88.3%)	104 (86.7%)
	Unmarried/ divorced/ widowed	9 (15.0%)	7 (11.7%)	16 (13.3%)
Smoking status	Non-smoker	0	43	43
		0.0%	71.7%	35.8%
	Ex-Smoker	13	14	27
		21.7%	23.3%	22.5%
	Current Smoker	47	3	50
		78.3%	5.0%	41.7%
Oral hygiene practices	Toothbrush	54 (90.0%)	52 (86.7%)	106 (88.3%)
	Other aids	6 (10.0%)	8 (13.3%)	14 (11.7%)
	Use of inter-dental aids	2 (3.3%)	3 (5.0%)	5 (4.2%)
Age (Mean±SD)		53.25±6.59	52.17±7.09	51.78±7.07

Table 2: Comparison of the periodontal parameters between the two groups (Mean ± SD).

	COPD (Mean±SD)	Non-COPD (Mean±SD)	p-value
Pocket probing depth	4.01±1.25	2.86±0.71	< 0.001*
Clinical attachment loss	3.77±1.92	1.18±0.89	< 0.001*
Gingival index	1.78±25.76	1.67±7.50	0.358
Plaque index	1.98±0.53	1.82±0.51	0.203
Oral hygiene index	2.07±1.28	1.98±0.93	0.301

* Significant at 0.05 level

A negative association of CAL and Pocket depth was found with increase in the FEV1/ FVC % showing a progressive increase in periodontal disease with decreasing lung function. (Table 3) The CAL and Pocket depth was also having higher severity among patients with severe COPD. (Table 4)

Table 3: Relationship between periodontal disease indices (CAL) and respiratory parameters (FEV1/FVC Ratio).

		FEV1/ FVC %
PPD	Pearson Correlation	-0.615
	p-value	< 0.001*
CAL	Pearson Correlation	-0.780
	p-value	< 0.001*
Gingival index	Pearson Correlation	-0.273
	p-value	0.105
PI	Pearson Correlation	-0.209
	p-value	0.113
OHI	Pearson Correlation	-0.263
	p-value	0.147

* Significant at 0.05 level

Table 4: Comparison of the periodontal parameters among COPD patients.

		Mean±SD	p-value	Post-hoc comparisons
Pocket probing depth	Mild (1)	2.46±0.36	< 0.001*	4 > 3 > 2 > 1
	Moderate (2)	3.55±0.62		
	Severe (3)	3.99±0.95		
	Very severe (4)	5.15±1.15		
Clinical attachment loss	Mild (1)	1.12±0.25	< 0.001*	4 > 3 > 2 > 1
	Moderate (2)	3.18±0.28		
	Severe (3)	4.85±0.48		
	Very severe (4)	5.91±0.79		
Gingival index	Mild (1)	1.78±0.49	0.211	N/A
	Moderate (2)	1.88±0.58		
	Severe (3)	1.95±0.42		
	Very severe (4)	2.01±0.60		
Plaque index	Mild (1)	1.86±0.12	0.196	N/A
	Moderate (2)	1.73±0.23		
	Severe (3)	1.90±0.42		
	Very severe (4)	1.88±0.62		
Oral hygiene index	Mild (1)	1.82±0.47	0.185	N/A
	Moderate (2)	1.93±0.94		
	Severe (3)	2.03±0.98		
	Very severe (4)	2.19±1.18		

Table 5: Binary logistic regression analysis for the predictors for COPD in the two groups.

	Adjusted Odds ratio (95.0% C.I.)	p-value
Pocket probing depth	4.912 (3.455-6.917)	0.001*
Clinical attachment loss	4.444 (2.389-6.443)	0.004*
Gingival index	1.198 (0.266-2.231)	0.119
Plaque index	1.080 (0.604-2.082)	0.124
Oral hygiene index	1.047 (0.550- 2.024)	0.121

(Adjusted for age, gender, BMI, marital status, smoking and oral hygiene practices)
* Significant at 0.05 level

The binary logistic regression analysis showed that both adjusted and unadjusted odds ratio showed that periodontal indices (CAL and PPD) were significantly (P < 0.05) worse in the COPD group compared with the non-COPD group.

4. Discussion

Out of the various systemic diseases, an area of particular interest is the link between periodontitis and COPD. An association between COPD and oral health was first noted in the community dwelling population after an analysis of 23,808 individuals in the National Health and Nutrition Examination Survey I (NHANES I) data [16]. Our findings suggest chronic periodontitis as a potential association for COPD.

A significantly higher mean OHI, PI, BPI, PPD and CAL were found in individuals with COPD group compared to non-COPD group. We age and gender matched subjects in our study. By doing this, COPD and without COPD participants were identical with respect to age and gender, we could compensate for the confounding effect of these two variables. A distinct trend of an increase in the periodontal parameters (PI, OHI, PPD and CAL), was noticed with increase in the severity of the COPD.

Not much difference could be found in the mean BPI value between COPD and non-COPD groups which might be due to the residual confounding effect of tobacco smoking on the bleeding of the gingiva. Bleeding on probing the gingiva is affected by chronic, and dose dependent suppressive effect of the tobacco smoke [17]. The result of present study agreed with the result of studies conducted by Wang et al, Yan et al, Prasanna et al and Lopex et al found a positive association of the periodontal parameters with the COPD [18-21].

Shen et al found that periodontal treatment is beneficial in decreasing the frequency and intensity of the respiratory events among COPD patients [22]. One of the clinical trials had also postulated that periodontal treatment is effective in decreasing the frequency of exacerbation and improvement in the lung function among COPD sufferers [23]. Our analysis indicated that there is an inverse relation between CAL (periodontal disease) and FEV1/ FVC% (COPD) and also between CAL and FEV1 (COPD) as the subjects with more periodontal attachment loss had a higher prevalence of diminished lung function and lung volumes.

As our study was a cross-sectional study, casual association is difficult to determine but it is still evident that association of severity of lung function deterioration and periodontal disease is linear. Bomble et al found that only mean OHI was significantly associated with COPD, and no association was found with mean PPD and mean CAL after adjusting for smoking. However, a study conducted by Peter et al. had significantly worse PD, CAL, and OHI in case group when compared to control group when adjusted for smoking. Lung function declined rapidly among patients with increase in the mean CAL and PPD levels after adjusting for smoking [24-26].

In the study conducted by Mojon, he could not find any causal association between oral hygiene and respiratory infection (COPD) [27]. The common pathophysiological processes may play a critical role in these results. The COPD patients tend to have higher levels of circulation of inflammatory cytokines and destructive mediators including C-reactive protein (CRP), interleukin (IL)-8, tumor necrosis factor (TNF-?), and matrix metalloproteinase (MMP).

Similarly, the periodontal disease has the similar pathophysiological process and these similar inflammatory cytokines and destructive mediators were also more among the periodontitis patients, leading to the loss of ligamentous support and alveolar bone of the teeth. Neutrophils have been found to be dysfunctional among COPD patients making the host less capable of dealing efficiently with bacteria in the periodontium, abnormal periodontal tissue-inflammatory reaction and higher intensity of the local inflammation [3].

5. Conclusion

The present study cannot ascertain causal association but provides substantial evidence that poor periodontal health is associated with obstructive lung disease. Professional and patient level interventions to improve oral health status may prove to lower the severity of lung infection in susceptible populations. Also, timely screening for periodontal diseases among COPD patients could be worthwhile in promoting oral hygiene.

To summarize, 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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