

## ORIGINAL ARTICLE

# CORRELATION BETWEEN RESPIRATORY HEALTH AND PERIODONTAL DISEASES IN CHHATISGARH POPULATION- A PROSPECTIVE CROSS-SECTIONAL STUDY

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### ABSTRACT:


**Background:** Oral infection, especially periodontitis, may affect the course and pathogenesis of a number of systemic diseases. Present study was aimed to evaluate the potential association between periodontal health and respiratory infections. **Materials and Methods:** A cross-sectional, study was conducted among 100 adult patients (20–60 years of age) in which 50 patients belonged to group A (test group) and 50 belonged to group B (control group). Gingival Index (GI) was used to assess the gingival status and Periodontal indices such as Russell's Periodontal Index (PI) and Periodontal Index for Risk of Infectiousness (PIRI), which were assessed in all 100 patients. **Results:** The scores of GI, PI and PIRI were highly significant in COPD group than in non-COPD group. **Conclusion:** There is positive association between respiratory infections and periodontal diseases. Periodontal status may serve as a useful risk marker to identify persons at higher risk for COPD. Further longitudinal studies are required to establish a positive correlation between periodontal health and development of COPD.

**Key words:** COPD, Gingival health, Periodontitis.

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### INTRODUCTION:

Periodontal disease (PD) is a chronic inflammatory disorder that leads to the destruction of the structures that give support to the teeth, as well as the alveolar bone, and subsequently to the loss of teeth due to bacterial infection. PD increases the risk for systemic diseases, and specifically respiratory infections. There is

evidence for an association between oral health and systemic and/or chronic diseases, particularly cardiovascular diseases, diabetes, osteoporosis, but also respiratory diseases such as COPD or pneumonia.<sup>1-3</sup>

The relationship between oral and respiratory health is not well understood, although the oral cavity and the lower airways are a continuum and exposures are

similar, and understanding the relationship might provide novel opportunities for interventions.<sup>4,5</sup>

Periodontal disease and chronic obstructive pulmonary disease (COPD) are characterized by chronic inflammation. The two conditions have a number of features in common such as a chronic trajectory, progressive and irreversible tissue destruction and gradual loss of normal organ function. Both conditions are strongly associated with tobacco smoking.<sup>6</sup>

It is beyond all doubt that smoking is an important risk factor for development of both periodontal disease and COPD and that the retrospectively reported association between the two conditions most likely reflects exposure to tobacco smoke. It is, however, not clear whether the susceptibility to smoke-induced tissue destruction is a general characteristic within an individual or if different tissues within one and the same person react differently to the harmful effects of smoking.<sup>6</sup> Till date there are sparse studies in which possible interrelationship between these two conditions has been explored and the results are inconclusive.

The present study was aimed to investigate the inter-relationship between respiratory infections and Periodontitis.

**MATERIAL AND METHODS**

A cross-sectional, study was conducted among 100 adult patients (20–60 years of age) of Chhatisgarh,

India reported January 1<sup>st</sup> to December 31<sup>st</sup> 2015. The protocol was approved by the Institutional Ethics Committee and verbal consent from subjects involved in the study was taken.

100 patients were selected on purposive selection criteria from the Outpatient Department of General Medicine. The patients were in the age range 20–60 years, of whom 50 patients belonged to group A (test group) and 50 belonged to group B (control group). Both the groups comprised age- and sex-matched individuals. Group A comprised 50 patients diagnosed as COPD. Group B comprised 50 patients without disease. Edentulous patients, patients who had undergone periodontal therapy for last 3 months or on medications known to influence the periodontal tissues for last 6–8 weeks were excluded from study. All detailed case history was taken and examined by physician for all patients. After confirmation of COPD as diagnosis by Physician, these patients were taken for the study. Lifestyle characteristics examined included history of smoking and alcohol consumption. A thorough medical history of each patient was recorded.

All 100 patients of groups A and B were examined for gingival and periodontal status by recording the Gingival Index (GI) (Loe and sillness), Periodontal Index (PI) (Russell’s), Periodontal Index for Risk of Infectiousness (PIRI). Results obtained were tabulated and statistically analyzed using SPSS software.

**RESULTS:**

**Table 1:** Age distribution of the study population

Age (in years)	Non COPD (n=50)	COPD (n=50)	t value	p
20-30	1	0	-10.062	0.023
30-40	6	2		
40-50	16	5		
>50	27	43		
<b>Total</b>	50	50		

**Table 2:** Comparison of mean gingival index between COPD and non COPD groups

Group (n=100)	Mean±S.D.	t value	p value
Non COPD (n=50)	1.48 ± 0.22	-7.02	<0.0001
COPD(n=50)	2.16 ± 0.54		

**Table 3:** Comparison of Russell's index between COPD and non COPD groups

Group (n=100)	Mean±S.D.	t value	p value
Non COPD (n=50)	3.68 ± 0.82	-16.42	<0.0001
COPD(n=50)	5.16 ± 0.64		

**Table 4:** Comparison of periodontal index for risk of infectiousness between COPD and non COPD groups

Group (n=100)	Mean±S.D.	t value	p value
Non COPD (n=50)	2.18 ± 1.82	-12.82	<0.0001
COPD(n=50)	6.16 ± 2.04		

**Table 5:** Cox Proportional-hazards Regression Analysis Measured Hazard Ratio of Periodontal Diseases among COPD Patients by Different Treatments

Treatment	Rate	Crude HR (95% CI)	Adjusted HR (95% CI)
Non steroids	42.8	1 (reference)	1 (reference)
Systemic steroids	46.2	1.17 (1.08-1.26)	1.36 (1.28-1.44)
Inhaled steroid	40.2	1.08 (1.04-1.12)	1.28 (1.22-1.34)

Table 1 showed that COPD is observed significantly observed in higher number in older age individuals above 50 years than younger individuals.

Table 2 showed that gingival index is significantly high in COPD subjects than non COPD subjects.

Table 3 revealed that Russell's index for periodontal health status is significantly high in COPD subjects than non COPD subjects.

Table 4 revealed that periodontal index for risk of infectiousness (PIRI) is significantly high in COPD subjects than non COPD subjects.

Table 5 indicated that patients on steroid therapy for COPD are at higher risk to develop periodontal diseases.

**DISCUSSION:**

It is a well known fact that oral health plays a major role in overall health and well-being of an individual. Thus, it was hypothesized that periodontitis and systemic diseases could be traced back to the beginning of recorded history and medicine. Certain systemic diseases, such as osteoporosis, diabetes and immune disorders, may increase the risk for periodontal disease. Literature regarding the association between periodontitis and respiratory disorders is scarce.<sup>7</sup>

Present study demonstrates that there is an increased risk of periodontal diseases in patients with respiratory infections compared with people in the general population.

Evaluation of the gingival status parameters revealed that GI scores had highly significant correlation in the COPD group. (Table 2)

Evaluation of the periodontal status parameters revealed that RI and PIRI scores had highly significant correlation in the COPD group. These scores were highly significant in the COPD group compared to non-COPD group. (Table 3 and 4)

A distinctive direct correlation was observed between COPD and periodontal diseases whereby more the severity of periodontal disease, greater was the association with COPD. These results are in agreement to Scannapieco and Genco<sup>8</sup> who suggested that poor oral hygiene and periodontal attachment loss is an independent risk factor for COPD. Scannapieco and Ho<sup>9</sup> and Xiaojing et al<sup>10</sup> studies also showed that more severe the mean attachment loss, greater is the association with COPD. However, contradictory results have been obtained by Scannapieco<sup>7</sup> who suggested that there is no association between the PI and chronic respiratory diseases.

Several microbiological studies have revealed that the same microorganisms are observed in oral cavity as well as in pulmonary infections.<sup>9</sup> Periodontal status may serve as a useful risk marker to identify persons at higher risk for COPD.<sup>11</sup> Further longitudinal studies are required to establish a positive correlation between periodontal health and development of COPD

This study has also revealed that patients with COPD treated with inhaled or systemic corticosteroids are at higher risk of having periodontal diseases than those who do not receive corticosteroid treatment. (Table 5) The effects of COPD medications, particularly corticosteroids, on periodontal diseases should also be emphasized. Corticosteroids can cause a decrease in the bone mineral density, and systemic bone loss may have an impact on the onset and progression of periodontal diseases. Komerik et al<sup>12</sup> reported that patients who inhaled corticosteroid treatments could experience impaired bone metabolism that may lead to a marked decrease in bone mineral density.

Smoking induces chronic airflow limitation in some smokers who possess a specific susceptibility to tobacco smoke whereas smoking seems to cause periodontal disease in a larger portion of the smokers. Although pathologic processes in the mouth cavity weakly co-varied with pathological processes in the lungs (emphysema and impaired diffusion capacity), we conclude that the susceptibility to the harmful effects of smoking is not a general characteristic within an individual smoker as the association between chronic airflow limitation and periodontitis tangibly varied among smokers.<sup>13</sup>

A number of potential mechanisms have been proposed to explain the biological plausibility for an association between periodontal disease and lung function. The oral cavity provides an optimal environment for bacterial growth because of its humidity and temperature. 1 mm<sup>3</sup> of plaque contains >10<sup>6</sup> bacteria with 300 different anaerobic and facultative anaerobic species. The most common route by which the oral cavity may influence pulmonary function is the aspiration of saliva harbouring oral bacteria into the lung. Aspiration of small amounts of saliva during sleep is quite common, even in healthy subjects. Subjects with severe periodontal disease harbour elevated levels of periodontal pathogens in saliva compared with healthy subjects. The amount of pathogens reflects pathogen burden in periodontal pockets. When

aspirated, oral pathogens cause local damage to the small airways.<sup>14-17</sup>

Furthermore, oral bacteria stimulate the inflamed periodontal tissues to release cytokines via the sulcus fluid into the saliva. In subjects with periodontal disease salivary biomarker load is higher than in healthy subjects. Aspirated cytokines may promote the adhesion and growth of respiratory pathogens in the lower airways. Subsequently, the respiratory epithelium may release cytokines and attract neutrophils, which in turn infiltrate airway parenchyma and release proteolytic enzymes and toxic oxygen radicals which damage the epithelium. As a consequence, the resultant inflamed mucosal epithelium may be particularly prone to infection.<sup>17</sup> Further insight into this might provide important opportunities for intervention that have been very little explored until now.

#### CONCLUSION:

There is definitive positive correlation between respiratory infections and periodontal diseases. Periodontal status may serve as a useful risk marker to identify persons at higher risk for COPD. Further longitudinal studies are required to establish a positive correlation between periodontal health and development of COPD.

#### REFERENCES:

1. Russell AL. Epidemiology of periodontal disease. *Int Dent J.* 1967;17:282–96.
2. Armitage GC. Periodontal diagnoses and classification of periodontal diseases. *Periodontol 2000* 2004; 34:9–21
3. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. *Lancet* 2005; 366:1809–1820.
4. Garcia RI, Henshaw MM, Krall EA. Relationship between periodontal disease and systemic health. *Periodontol 2000.* 2001;25:21–36.
5. Salmon A, Xiaoze H. The impact of periodontal infection on systemic disease. *Med Sci Monit.* 2003;9:291–9.
6. Bergström J, Cederlund K, Dahlén B, Lantz A-S, Skedinger M, Palmberg L, et al. (2013) Dental Health in Smokers with and without COPD. *PLoS ONE* 8(3): e59492.
7. Scannapieco FA, Papandonatos GD, Dunford RG. Associations between oral conditions and respiratory disease in a national sample survey population. *Ann Periodontol* 1998;3: 251–256.
8. Scannapieco FA, Genco RJ. Association of Periodontal infections with atherosclerotic and pulmonary diseases. *J Periodont Res.* 1999;34:340–5.

9. Scannapieco FA, Ho AW. Potential association between chronic respiratory disease and periodontal disease: Analysis of National Health and Nutrition Examination Survey III. *J Periodontol.* 2001;72:50–6.
10. Li X, Kolltveit KM, Tronstad L, Olsen I. Systemic diseases caused by oral infection. *Clin Microbiol Rev.* 2000;13:547–58.
11. Prasanna SJ. Causal relationship between periodontitis and chronic obstructive pulmonary disease. *J Indian Soc Periodontol.* 2011 Oct-Dec; 15(4): 359–365.
12. Komerik N, Akkaya A, Yildiz M, Buyukkaplan US, Kuru L. Oral health in patients on inhaled corticosteroid treatment. *Oral Dis* 2005; 11:303–308.
13. Jones PW, Quirk FH, Baveystock CM, Littlejohns P. A self-complete measure of health status for chronic airflow limitation. The St. George's Respiratory Questionnaire. *Am Rev Respir Dis* 1992;145: 1321–1327.
14. Scannapieco FA, Bush RB, Paju S. Associations between periodontal disease and risk for nosocomial bacterial pneumonia and chronic obstructive pulmonary disease. A systematic review. *Ann Periodontol* 2003;8: 54–69.
15. Slots J, Rams TE, Listgarten MA. Yeasts, enteric rods and pseudomonads in the subgingival flora of severe adult periodontitis. *Oral Microbiol Immunol* 1988; 3: 47–52.
16. Sinclair DG, Evans TW. Nosocomial pneumonia in the intensive care unit. *Br J Hosp Med* 1994; 51:177–180.
17. Pesci A, Balbi B, Majori M, et al. Inflammatory cells and mediators in bronchial lavage of patients with chronic obstructive pulmonary disease. *Eur Respir J* 1998; 12: 380–386.



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