Association Between Respiratory Disease in Hospitalized Patients and Periodontal Disease: A Cross-Sectional Study

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Background: Recent research indicated that periodontal infection may worsen systemic diseases, including pulmonary disease. Respiratory infections, such as pneumonia and the exacerbation of chronic obstructive pulmonary disease, involve the aspiration of bacteria from the oropharynx into the lower respiratory tract.

Methods: A group of 100 cases (hospitalized patients with respiratory disease) and a group of 100 age-, sex-, and race-matched outpatient controls (systemically healthy patients from the outpatient clinic, Department of Periodontics, Government Dental College and Hospital, Calicut, Kerala, India) were selected for the study. Standardized measures of oral health that were performed and compared included the gingival index (GI), plaque index (PI), and simplified oral hygiene index (OHI). Data regarding probing depths and clinical attachment levels (CALs) were recorded at four sites per tooth and compared statistically. The χ^2 and Student *t* tests were used for statistical analyses.

Results: The comparison of study-population demographics on the basis of age, sex, education, and income showed no significant differences between groups. Patients with respiratory disease had significantly greater poor periodontal health (OHI and PI), gingival inflammation (GI), deeper pockets, and CALs compared to controls. In the case group, patients with a low income were 4.4 times more prone to periodontal disease compared to high-income patients. Smokers had significantly higher CALs compared to non-smokers in the control group.

Conclusion: The findings of the present analysis support an association between respiratory and periodontal disease. *J Periodontol 2011;82:1155-1160.*

KEY WORDS

Cross-sectional studies; lung diseases, obstructive pulmonary disease; oral health; periodontal diseases/complications; pneumonia; risk factors.

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Periodontal medicine, as a new branch or arm of periodontology, has lent an impetus to coalesce medicine and dentistry. Research established that periodontal infection is a probable risk factor for various systemic diseases, including pulmonary disease.[1-13] Respiratory infections, such as pneumonia and the exacerbation of chronic obstructive pulmonary disease (COPD), involve the aspiration of bacteria from the oropharynx into the lower respiratory tract. The failure of the host defense systems to eliminate pathogens from the mucosal surface results in their proliferation, which sometimes is followed by infection and tissue destruction.[14-18] It was suggested that dental plaque may serve as a reservoir for respiratory pathogens, especially in high-risk patients with poor oral hygiene.[15] Institutionalized patients appear to be more prone to oral colonization by these bacteria than ambulatory, non-institutionalized patients.[5,19,20]

Poor oral health (e.g., periodontal disease) was also associated with COPD.[1,2,21] Patients with COPD suffer from periodic acute exacerbations or a worsening of lung function, which are partially due to infection, typically by bacteria such as *Streptococcus pneumonia*, *Haemophilus influenzae*, and *Moraxella catarrhalis*[22] or rhinovirus.[23] A lower respiratory infection begins by contamination of the lower airway epithelium

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by microorganisms contained in aerosolized droplets or by aspiration of oral secretions containing microorganisms, but all individuals do not have an equal risk for a lower respiratory infection.[24] Community-acquired pneumonias are associated with conditions that increase the propensity for aspiration (alterations of consciousness because of stroke, seizures, dementia, and alcohol abuse). In these neurologic conditions, the existing synchronized swallowing and breathing mechanism is often disrupted. Recurrent pneumonia suggests the presence of specific predisposing factors, such as a congenital defects in the host defense, cystic fibrosis, acquired immunodeficiency, and other immunodeficiency syndromes or COPD.[17] Significantly, results from three preliminary intervention trials[12,13,25] demonstrated that attention to oral hygiene, either by the use of mechanical cleansing and/or oral antiseptic rinses, such as betadine or chlorhexidine gluconate, significantly reduced the rate of lower respiratory tract infection in institutionalized patients.

Pulmonary diseases are highly prevalent in our society. The associated pain and suffering is high; because of their chronic nature, the economic cost to the society and the burden on the health care system is enormous. COPD is severely disabling and debilitating, requiring lengthy periods of institutionalization and usually culminating in death. There is good evidence that improved oral hygiene and frequent professional oral health care reduces the progression or occurrence of respiratory diseases.[6] If periodontitis enhances the risk of respiratory disease, dentistry in general and, in particular, periodontology have significant roles to play in overall prevention. Hence, this study was planned and performed with an aim to substantiate the association between respiratory and periodontal disease in Indian subcontinent populations.

MATERIALS AND METHODS

Sample Population

A group of 100 cases (hospitalized patients with respiratory disease at the Institute of Chest Disease, Government Medical College and Hospital, Calicut, Kerala, India) was selected. Similarly, 100 age-, sex-, and race-matched controls (new patients at the outpatient clinic, Department of Periodontics, Government Dental College and Hospital, Calicut, Kerala, India) were also selected. After obtaining institutional and ethics board approval, written consent of patients was also provided. The hospital records pertaining to each patient during the study period in the hospital were screened. The inclusion and the exclusion criteria were set, and systematic random sampling was used to select cases and controls. The study period was from April 2004 to November 2004.

Inclusion criteria. 1) Potential cases were patients who were hospitalized for >3 days and diagnosed with acute respiratory disease (i.e., pneumonia, acute bronchitis, or a lung abscess) or an exacerbation of chronic respiratory disease (i.e., COPD, which included chronic bronchitis and emphysema); 2) Patients with no past or present history of respiratory disease were considered potential controls; and 3) Patients were aged 20 to 60 years old and had ≥20 natural teeth.

Exclusion criteria. 1) Patients who had a history of systemic diseases (e.g., diabetes mellitus) other than respiratory disease; 2) Patients who were under any medication that was known to influence periodontal tissues; 3) Patients with a history of any periodontal treatment in the past 6 months; and 4) Patients hospitalized in the Intensive Care Unit.

Information regarding a patient's age, sex, socioeconomic status (SES), and lifestyle was considered in this study. SES variables considered included education (categorized as illiterate, primary school, middle school, high school, and college) and monthly household income (categorized as <50, 50 to 100, and >100 US dollars [US$]). Lifestyle characteristics examined included a history of smoking and past dental treatment. All patients who had a history of smoking were heavy smokers (>20 cigarettes/day), and there were no former smokers.

Clinical Parameters

The following standardized measures of oral health were performed: the gingival index (GI) of Löe and Silness,[26] the plaque index (PI) of Silness and Löe,[27] and the simplified oral hygiene index (OHI) of Greene and Vermillion.[28] The clinical attachment level (CAL) was obtained by subtracting the distance from the free gingival margin (FGM) to the cement-enamel junction as a reference point of each tooth from the distance from the FGM to the bottom of the sulcus. Measurements regarding probing depth (PD) and CAL were recorded at four sites per tooth on the disto-facial, facial, mesio-facial, and lingual surface, which were averaged for all sites within the patient and then across groups. An oral examination was done under proper illumination with the case-group patient sitting erect on a bed and with a mouth mirror and William's graduated probe.‡ All measurements were performed by a single calibrated examiner (NS).

Statistical Analyses

Data collected were entered into a master sheet and later transferred into computer spreadsheets. The Student t and χ^2 tests and contingency-table analysis were used to assess imbalances in diseased (cases) and non-diseased (controls) patients with respect to

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study-population demographics and periodontal parameters. A comparison of periodontal parameters between groups was adjusted for smoking. The association between income and periodontal health was also assessed. The level of significance was determined at $P < 0.0012$.

RESULTS

None of the demographic variables were statistically different between groups (Table 1). In the case group, 72% of patients had COPD (48 males and 24 females), 17% of patients had pneumonia (nine males and eight females), and 11% of patients had a lung abscess (eight males and three females). Mean values of the GI, PI, and OHI for patients with respiratory disease were significantly higher than for the control group: GI (P = 4.46E-27), PI (P = 1.83E-27), and OHI (P = 4.04E-30). Deeper PDs and higher CALs were associated with respiratory disease: PD (P = 9.27E-12) and CAL (P = 1.91E-17) (Table 2).

The mean CAL for both groups was computed and dichotomized as patients who had CALs <3.0 mm compared to patients who had CALs >3.0 mm. Patients with and without respiratory disease were divided into two groups on the basis of their monthly income (i.e., <$50 US dollars and >$50 US dollars). Contingency tables were prepared to find odds ratios. The mean CAL value was found to be statistically higher in the high income group, which predicted that a low SES and clinical attachment level were highly dependent in patients with respiratory disease. By calculating the odds ratio, patients with respiratory disease in the low income group were ≈4.4 times more likely to have poor periodontal health compared to the high-income group. No such association was observed in the control group (Table 3). In the non-respiratory disease group, the mean CAL value for smokers was significantly higher compared to non-smokers, whereas the mean CAL values were statistically indifferent within the case group (Table 4).

DISCUSSION

A lower respiratory infection begins by the contamination of the lower airway epithelium by microorganisms contained in aerosolized droplets or by aspiration of oral secretions containing microorganisms.[29] Oral bacteria, poor oral hygiene, and periodontitis seem to influence the incidence of pulmonary infections, especially nosocomial pneumonia episodes in high-risk patients.[30] Only some studies attempted to test this hypothesis.[2,12] These preliminary findings were encouraging and collectively support the hypothesis but clearly indicated the need for further corroboration.

The authors in the present study found significantly higher scores of the mean GI in the case group contrary to the findings in the study by Scannapieco and Ho[21] who found no significant relationship between gingival bleeding alone and respiratory disease. Significantly higher values of the mean PI in the respiratory disease group were also observed by Scannapieco et al.[5] and Russell et al.[3] Significantly greater mean OHI, mean PD, and mean CAL values were also associated with respiratory disease as demonstrated by Scannapieco et al.,[1] Garcia et al.,[31] and Hayes et al.[2]

These results that were substantiated by several studies[3,5,25,32-34] suggested that supragingival plaque accumulation and/or periodontal pockets favor respiratory pathogen colonization and make susceptible patients prone to a greater risk of acquiring a respiratory disease. Patients with periodontal disease and elevated levels of proteolytic bacteria such as *Porphyromonas gingivalis* and spirochetes produce enzymes (such as proteases). These enzymes and elevated levels of various hydrolytic salivary enzymes that are due to an increased dental-plaque load destroy protective domains of host secretory components (e.g., mucins) and, thus, diminish the non-specific host defense against respiratory pathogens in high-risk patients.[17] High salivary concentrations of *P. gingivalis* enhance the risk for respiratory disease with an odds

Table 1.

Study-Population Demographics

Variable	Cases (n)	Controls (n)
Age groups		
20 to 30 years	5	7
31 to 40 years	13	20
41 to 50 years	30	28
51 to 60 years	52	45
Total	100	100
Sex		
Males	65	58
Females	35	42
Education		
A (nil)	25	9
B (grade 5)	12	3
C (grade 8)	26	40
D (grade 12)	33	41
E (college)	4	7
Monthly income (US$)		
<50	74	77
50 to 100	8	12
>100	18	11
Smoking		
Smokers*	37	17
Non-smokers†	63	83

* All subjects were heavy smokers (>20 cigarettes/day).
† There were no former smokers.

Table 2.

Comparison of Periodontal Parameters Between Groups

Parameter	Cases	Controls
GI*		
Mean	1.787	1.219
SD	0.404	0.632
PI*		
Mean	1.825	1.273
SD	0.386	0.197
OHI*		
Mean	3.943	2.817
SD	0.636	0.531
PD*		
Mean (mm)	2.227	1.689
SD	0.608	0.425
CAL*		
Mean (mm)	3.135	2.436
SD	0.533	0.522

* Significant at $P < 0.0012$.

Table 3.

Association of Income and CAL

Patients	CAL >3.0 mm	CAL <3.0 mm	Odds Ratio
With respiratory disease (n)			
<US$50	20	57	4.433
>US$50	14	9	
Without respiratory disease (n)			
<US$50	12	62	0.674
>US$50	3	23	

Table 4.

CAL Comparison of Smokers and Non-Smokers

Patients	Subjects (n)	CAL (mean)	CAL (SD)	*P*
With respiratory disease				
Non-smokers	63	3.116	0.613	0.7018
Smokers	37	3.160	0.494	
Without respiratory disease				
Non-smoker	83	2.450	0.513	0.0011
Smokers	17	3.151	0.319	

ratio of 4.2.[35] *Staphylococcus aureus* can colonize in the mouths of debilitated or institutionalized persons. *S. aureus* in the saliva enhances the risk for aspiration pneumonia by 7.4-fold.[36] Hence, poor oral health may work in concert with other factors (such as continued smoking, environmental pollutants, viral infections, allergy, and/or genetic factors) to promote the progression and/or exacerbation of respiratory disease.[21]

The present study may have been limited in several ways. The association between periodontitis and respiratory diseases may have been confounded by shared risk factors especially smoking, which is the leading risk factor for periodontitis , emphysema, chronic bronchitis, and lung infections. Tobacco smoking suppresses the production of protective immunoglobulin G2 antibodies and blocks phagocytosis and the killing of bacteria by neutrophils.[37] Tobacco smoking also paralyzes the ciliary action and hampers lung clearance, enhancing the risk for respiratory disease by over four-fold. Furthermore, because the present study was cross-sectional in design, a definite cause-and-effect relationship could not be established for poor periodontal health and the occurrence of respiratory disease. Because the Scannapieco et al.[1] and Scannapieco and Ho[21] studies were cross-sectional in design, they could not demonstrate temporality. Temporality was demonstrated in the study of Hayes et al.[2] None of the studies demonstrated a dose effect (i.e., that the risk for respiratory disease increases with the increasing severity and duration of periodontitis). However, in light of the complexity and multifactorial nature of respiratory disease, a demonstration of a dose effect for the association between periodontitis and respiratory disease is unlikely. Hence, plaque accumulation or periodontal disease does not directly cause respiratory or other systemic diseases. They may, in fact, aggravate these systemic diseases in susceptible and high-risk individuals. The need for rigorous plaque control and treatment of oral infections, particularly in these risk groups, is highly justified.

CONCLUSIONS

The findings of the present analysis substantiates a potential association between periodontal disease and respiratory disease, indicating a positive correlation between poor periodontal health and the risk of developing respiratory disease. Dentists, especially periodontists, may be able to play a significant role in the prevention of respiratory disease by redoubling their efforts to prevent periodontitis and arresting the progression in patients already afflicted by periodontitis. Hence, in addition to an evaluation of the factors contributing to the associated risk, the patient should be educated regarding the risk, and when appropriate, suitable intervention strategies should be implemented. Relevant studies to be performed include prospective longitudinal studies comparing respiratory disease rates in institutionalized patients

with and without periodontal disease and interventional studies to assess the effect of periodontal therapy on the incidence of respiratory disease in these patients.

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