Association Among Rheumatoid Arthritis, Oral Hygiene, and Periodontitis

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Background: A limited number of studies suggest a higher prevalence of periodontal disease among individuals with rheumatoid arthritis (RA); however, results have been inconsistent. Further, it is unclear to what extent poor oral hygiene among patients with RA may account for this association.

Methods: The association between RA and periodontitis was examined in 57 subjects with RA and 52 healthy controls, matched by age and gender. Oral examination included plaque index (PI), gingival index (GI), probing depth (PD), and clinical attachment loss (CAL). Potential risk factors for periodontal disease, such as smoking, education, alcohol consumption, and body mass index (BMI), as well as chronic diseases associated with RA and periodontal disease were assessed through questionnaires.

Results: In a stepwise logistic regression, including RA status, age, gender, education, smoking, alcohol consumption, and BMI, only RA status and age remained significant predictors of periodontal disease. Subjects with RA had a significant 8.05-fold increased odds (95% confidence interval: 2.93 to 22.09) of periodontitis compared to controls. The strength of the association was attenuated but remained statistically significant after further adjustment for PI, GI, or both. PI alone accounted for 12.4%, GI alone accounted for 11.1%, and PI and GI combined accounted for 13.4% of the association between RA and periodontitis.

Conclusions: Subjects with RA have significantly increased periodontal attachment loss compared to controls. Oral hygiene may only partially account for this association. *J Periodontol 2008;79:979-986.*

KEY WORDS

Oral hygiene; periodontal disease; rheumatoid arthritis; risk factors.

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Periodontitis is a common disease worldwide that has a primarily bacterial etiology and is characterized by a dysregulation of the host inflammatory response, eventually resulting in soft and hard tissue destruction.[1,2] The degree of inflammation varies among individuals with periodontal disease, independently of the degree of bacterial infection, suggesting that alteration of the immune function may substantially contribute to its extent. Factors such as smoking, education, and body mass index (BMI) are discussed as potential risk factors for periodontal disease.[3-5]

Rheumatoid arthritis (RA) is a systemic inflammatory disorder with a prevalence of 0.5% to 1.0% in Western populations; it affects women about three times more often than men.[6,7] In addition to alterations in systemic immune function, RA is characterized by the accumulation of proinflammatory cell infiltrates in the synovial membrane, which leads to synovitis, destruction of cartilage and bone tissue of the joints, and, ultimately, to physical impairment and disabilities.[7,8]

In addition, RA often affects the proximal interphalangeal and metacarpophalangeal joints,[7] which may lead to substantial manual disability. Oral hygiene may be impaired in these patients, making them susceptible to plaque accumulation and, consequently, inflammatory periodontal disease.

Only a limited number of studies[9-18] have examined the overall oral status and, in particular, the periodontal status,

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in subjects with RA. Although results have been inconsistent,[14,16,17] previous data suggested a higher prevalence of periodontal disease among individuals with RA, and a relative risk of 4.7 was suggested.[9-13,19]

The present study examined the prevalence of periodontal disease among subjects with RA and to what extent oral hygiene may account for this association.

MATERIALS AND METHODS

Study Population

A total of 109 subjects were included in the present study. Subjects with RA were recruited from individuals who attended the Department of Rheumatology and Clinical Immunology, Charité-Medical Faculty Berlin, for routine examination. RA was diagnosed according to the American Rheumatism Association (ARA).[20,21] According to the classification, subjects with RA have four of the following seven criteria: morning stiffness, arthritis of three or more joint areas, arthritis of hand joints, symmetric arthritis, rheumatoid nodules, serum rheumatoid factors, and radiographic changes. Between 2005 and 2006, 57 subjects with RA (eight males and 49 females; mean age: 52.1 ± 13.0 years; median disease duration: 10.5 years) provided informed consent and were enrolled into the study. Subjects with RA were taking disease-modifying antirheumatic drugs, non-steroidal anti-inflammatory drugs, corticosteroids, and/or tumor necrosis factor-alpha antagonists at the time of the investigation. A non-RA control group of 52 subjects (nine males and 43 females; mean age: 52.1 ± 13.7 years), frequency matched for age and gender, was also examined. Controls were recruited from patients attending the general dental office, Charité-Medical Faculty Berlin. Exclusion criteria for cases and controls were a history of periodontal therapy or the use of antibiotics during the last 3 months prior to examination, pregnancy, or lactation. The study protocol was approved by the ethics committee of the Charité-Medical Faculty Berlin. All study subjects provided a written informed consent.

Assessment of Clinical Rheumatologic Parameters

Disease activity in subjects with RA was assessed by the Disease Activity Score (DAS28).[22] This disease activity index ranges from 0 to 10 and includes a 28 tender-and-swollen joint count, the erythrocyte sedimentation rate (ESR, mm/hour[23]), and the patient's assessment of disease activity measured with a visual analog scale (100 mm). For example, DAS28 >5.1 meant that the subject had a high disease activity, whereas DAS28 <3.2 meant that disease activity was low. Also, the Health Assessment Questionnaire (HAQ)[24] score was recorded. In the present study, HAQ was based on self-reports and assessed the subject's disabilities (e.g., dressing, arising, eating, walking, hygiene, reach grip, and common activities). The disability index is scored from 0 to 3, ranging from no assistance needed (score 0) to the use of an aid or device (cane, walker, crutches, or wheelchair) as well as assistance from another person (score 3).

Laboratory Examinations

Blood plasma was collected and analyzed for C-reactive protein (CRP) by a highly sensitive, latex particle–enhanced immunoassay; ESR by the Westergren method;[23] and immunoglobulin (Ig)M and IgA rheumatoid factors and antibodies to cyclic citrullinated peptides by enzyme-linked immunosorbent assay at the Department of Rheumatology and Clinical Immunology, Charité-Medical Faculty Berlin.

Intraoral Examination

Oral examinations of the subjects with RA and of the control subjects were performed by the same examiner using a manual periodontal probe,¶ and the readings were recorded to the nearest 1 mm. All periodontal measurements were assessed at four sites of each tooth (mesio-buccal, disto-buccal, mesio-lingual, and disto-lingual). The plaque index (PI) was evaluated according to Silness and Löe,[25] and the gingival index (GI) was assessed according to Löe.[26] PI was calculated as the intraindividual mean from four sites per tooth: 0, absence of plaque on the tooth surface; 1, plaque disclosed after running a periodontal probe along the gingival margin; 2, visible plaque accumulation; and 3, abundance of plaque. GI was calculated as the intraindividual mean from four sites per tooth: 0, complete absence of visual signs of inflammation in the gingiva; 1, slight change in gingival color and texture; 2, visual inflammation: redness, edema, and glazing; bleeding tendency right after a periodontal probe ran along the gingival margin; and 3, overt inflammation: marked redness and edema, ulcerations, tendency to spontaneous bleeding. Probing depth (PD) was defined as the distance from the free gingival margin to the bottom of the sulcus or periodontal pocket. Gingival recession was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin. Clinical attachment loss (CAL) was defined as the distance from the CEJ to the bottom of the sulcus or periodontal pocket and was calculated as the sum of PD and gingival recession measurements. Periodontal disease was defined as a mean CAL >4.0, corresponding to the median level in our population. Bleeding on probing was dichotomously evaluated as bleeding present or absent 10 seconds following probing.[27] In addition, the Decayed, Missing Filled Teeth (DMFT) index, according to Klein,[28] and evaluation of the temporomandibular

¶ PCP 11, Hu-Friedy, Chicago, IL.

joint (pain, crepitation, limitation of movements)[29] were recorded.

Assessment of Risk Factors

To describe the present study population, we assessed sociodemographic characteristics, lifestyle factors, and medical history by a self-administered questionnaire. Potential risk factors for periodontitis, such as smoking, alcohol consumption, BMI, and education, were assessed.[3-5] Smoking status was classified as never-smoker, former smoker, or current smoker. Alcohol consumption was classified based on the reported drinking frequency as no to low (once a month or less), moderate (twice a month to several times a week), or heavy (daily or more frequent consumption). BMI was classified as <25, 25 to <30, or ≥30 kg/m^2. Educational attainment was classified as untrained, trained workers, or academic education.

In addition, the assessment of the medical history included diseases that are associated with RA, such as Sjögren's syndrome,[30] or with RA and periodontal disease, such as osteoporosis,[31] coronary heart disease,[32-35] and diseases that often co-occur as the metabolic syndrome, e.g., diabetes mellitus, hypertension, and dyslipidemia.[36,37]

Statistical Analyses

Frequency distributions, means, and standard deviations were determined to describe the data. Risk factors and dental variables were compared between subjects with RA and non-RA controls using the Student unpaired *t* test for continuous variables or the χ^2 test for categorical variables. The association of the individuals' characteristics with the risk for periodontitis was assessed using univariate logistic regression with periodontal disease defined as a mean CAL >4.0. Multivariable-adjusted unconditional logistic regression was used to examine the association of RA with the risk for periodontal disease. In a first step, we entered RA status according to ARA criteria, age, gender, educational attainment, smoking, alcohol consumption, and BMI into a stepwise logistic regression model and kept only those variables in the model that remained significant predictors of periodontal disease at the 5% level. In a second step, we forced PI, GI, or both into the final logistic regression model to estimate to what extent adjustment for these potentially intermediate variables attenuates the association between RA and periodontal disease. All *P* values presented are two-tailed, and *P* values <0.05 were considered statistically significant. Analyses were performed using a statistical program.#

RESULTS

Sociodemographic characteristics, lifestyle factors, and aspects of the medical history of subjects with RA and controls are shown in Table 1. A significantly higher prevalence of osteoporosis (36.8%) was found in subjects with RA compared to control subjects (1.9%; $P<0.005$).

Table 2 shows dental variables in patients with RA and controls. CAL and PD were significantly higher in subjects with RA compared to control subjects (4.37 ± 1.30 mm versus 3.40 ± 0.89 mm, $P<0.001$; and 3.71 ± 0.73 mm versus 3.16 ± 0.58 mm, $P<0.001$; respectively). Significant differences were also found in plaque accumulation, PI (0.71 ± 0.46 versus 0.44 ± 0.28; $P<0.001$) and inflammatory gingival status, GI (0.83 ± 0.48 versus 0.57 ± 0.39; $P<0.003$). Also, significantly ($P<0.001$) higher levels of bleeding on probing were found in subjects with RA.

In univariate analyses, we next examined the association of RA and individuals' characteristics with the odds of periodontitis (Table 3). Subjects with RA had a significant 5.7-fold increased odds of periodontal disease compared to healthy controls (95% confidence interval [CI]: 2.35 to 13.84). Potential risk factors for periodontal disease, such as higher age, low education, smoking, heavy alcohol consumption, and higher BMI, were related to a higher odds of periodontal disease, although only age was statistically significant at the 5% level.

Next, we used stepwise logistic regression to examine the association of RA with the odds of periodontal disease adjusted for potential confounders (Table 4). In addition to RA, we entered age, gender, education, smoking, alcohol consumption, and BMI into the logistic regression model. In this analysis, only age and RA status remained as significant predictors of periodontal disease. Thus, adjusted for age, subjects with RA had a 8.05-fold increased odds of periodontal disease (95% CI: 2.93 to 22.09) compared to controls (Table 4). Then, we examined to what extent PI and GI, as measures of oral hygiene, may account for the observed association between RA and periodontal disease by forcing these variables alone and in combination, in addition to age and RA, into the logistic regression model. PI and GI were markedly associated with CAL (Spearman correlation coefficient r = 0.59; $P<0.001$ and r = 0.70; $P<0.001$, respectively). The strength of the association of RA with periodontal disease was attenuated but remained statistically significant after further adjustment for PI, GI, or both. Thus, the odds ratio (OR) for the association of RA with periodontal disease decreased to 6.21 (95% CI: 2.04 to 18.91) when adjusted for PI, to 6.39 (95% CI: 1.93 to 21.23) when adjusted for GI, and to 6.09 (95% CI: 1.72 to 21.58) when adjusted for both. When comparing the regression coefficients (which are the natural log of the OR) from the logistic models, PI alone accounted for 12.4%, GI alone accounted for

SPSS 13.0, SPSS, Chicago, IL.

Table 1.

Characteristics of Individuals With RA and Controls

Characteristic	RA (N = 57)	Controls (N = 52)	*P* Value*
Female gender	49 (86.0)	43 (82.7)	0.64
Age (years; mean ± SD)	52.1 ± 13.0	52.1 ± 13.7	0.98
Educational attainment			0.10
Untrained	7 (13.0)	2 (3.8)	
Trained worker	33 (57.9)	26 (50.0)	
Academic	17 (31.5)	24 (46.2)	
Smoking			0.90
Never	23 (40.4)	31 (59.6)	
Former	22 (38.6)	11 (21.2)	
Current	12 (21.1)	10 (19.2)	
Alcohol consumption			0.13
No or low	32 (56.1)	23 (44.2)	
Moderate	17 (29.8)	25 (48.1)	
Heavy	8 (14.0)	4 (7.7)	
BMI (kg/m^2)			0.13
<25	36 (63.2)	23 (44.2)	
≥25 to <30	15 (26.3)	22 (42.3)	
≥30	6 (10.5)	7 (13.5)	
Hypertension	18 (31.6)	19 (36.5)	0.63
Diabetes mellitus	1 (1.8)	3 (5.8)	0.28
Dyslipidemia	10 (17.5)	11 (21.2)	0.63
Coronary heart disease	3 (5.3)	2 (3.8)	0.72
Osteoporosis	21 (36.8)	1 (1.9)	<0.005
Sjögren's syndrome	4 (7.0)	0	0.12

Data are expressed as number of individuals and percent (in parentheses) unless stated otherwise.
* Variables were compared between cases and controls using the χ^2 test for categorical variables and the Student unpaired *t* test for continuous variables.

Table 2.

Dental Variables in Individuals With RA and Controls

Characteristic	RA (N = 57)	Controls (N = 52)	*P* Value*
CAL (mm)			0.001
≤3.0	3 (5.3)	15 (28.8)	
>3.0 to ≤4.0	34 (59.6)	32 (61.5)	
>4.0 to ≤6.0	13 (22.8)	3 (5.8)	
>6.0	7 (12.3)	2 (3.8)	
Mean ± SD	4.37 ± 1.30	3.40 ± 0.89	<0.001
PD (mm)			0.002
≤3.0	8 (13.6)	18 (34.6)	
>3.0 to ≤4.0	31 (54.4)	31 (59.6)	
>4.0 to ≤5.0	15 (26.3)	3 (5.8)	
>5.0	3 (5.3)	0 (0.0)	
Mean ± SD	3.71 ± 0.73	3.16 ± 0.58	<0.001
Lost teeth (n)			0.875
None	11 (19.3)	13 (25.0)	
1 to 6	24 (42.1)	20 (38.5)	
7 to 12	9 (15.8)	9 (17.3)	
>12	13 (22.8)	10 (19.2)	
Mean ± SD	6.58 ± 6.94	6.50 ± 7.10	0.95
PI			0.004
≤0.3	7 (12.3)	19 (37.3)	
>0.3 to ≤0.6	22 (38.6)	21 (41.2)	
>0.6 to ≤0.9	13 (22.8)	7 (13.7)	
>0.9	15 (26.3)	4 (7.8)	
Mean± SD	0.71 ± 0.46	0.44 ± 0.28	<0.001
GI			0.003
≤0.4	9 (15.8)	22 (43.1)	
>0.4 to ≤0.8	20 (35.1)	19 (37.3)	
>0.8 to ≤1.2	18 (31.6)	5 (9.8)	
>1.2	10 (17.5)	5 (9.8)	
Mean ± SD	0.83 ± 0.48	0.57 ± 0.39	0.003
Bleeding on probing	0.60 ± 0.26	0.41 ± 0.28	<0.001
DMFT index	18.54 ± 6.58	18.12 ± 6.85	0.74
TMJ symptoms†	20 (35.1)	12 (23.1)	0.17

Values are numbers of individuals and percent (in parentheses) unless stated otherwise.
* Variables were compared between cases and controls using the χ^2 test for categorical variables and the Student unpaired *t* test for continuous variables.
† Temporomandibular joint symptoms (pain, crepitation, and limitation of movements).

11.1%, and PI and GI combined accounted for 13.4% of the association between RA and periodontal disease.

Table 5 shows the rheumatologic characteristics of subjects with RA with a mean CAL ≤4 or >4 mm. Disease duration, clinical rheumatologic data, laboratory measurements, and medication were not significantly different between subjects with RA with a mean CAL >4 mm and subjects with RA with a mean CAL ≤4 mm.

DISCUSSION

In the present study, RA was significantly associated with higher odds of periodontitis compared to control subjects. This association was independent of demographic and lifestyle characteristics, including age, gender, education, smoking status, alcohol consumption, and BMI. Impaired oral hygiene only partially accounted for this association.

The association between RA and periodontal disease has been examined in a few studies with inconsistent results. Although most earlier studies[14,16,17] did not find positive associations of RA with periodontal

Table 3.

Univariate Associations of RA and Individuals' Characteristics With Periodontitis

Characteristic	OR (95% CI) for Periodontitis
RA	
No	1 (reference)
Yes	5.70 (2.35 to 13.84)
Gender	
Male	1 (reference)
Female	0.45 (0.16 to 1.29)
Age (per 1 year)	1.07 (1.03 to 1.11)
Educational attainment	
Untrained	1 (reference)
Trained worker	0.51 (0.12 to 2.10)
Academic	0.33 (0.08 to 1.45)
Smoking	
Never	1 (reference)
Former	2.17 (0.87 to 5.37)
Current	2.17 (0.77 to 6.06)
Alcohol consumption	
No or low	1 (reference)
Moderate	0.35 (0.14 to 0.88)
Heavy	1.81 (0.51 to 6.41)
BMI (kg/m^2)	
<25	1 (reference)
≥25 to <30	1.55 (0.66 to 3.67)
≥30	2.66 (0.78 to 9.03)

Periodontitis was defined as a mean CAL >4.0 mm. The analysis includes 69 individuals without periodontitis and 40 individuals with periodontitis.

Table 4.

Multivariable Adjusted ORs and β-Coefficients for the Association of RA With Periodontitis With and Without Further Adjustment for PI and GI

	Adjustment for		OR for the Association of RA With Periodontitis (95% CI)	*P* Value	β (% change)*
	PI	GI			
Model 1†	No	No	8.05 (2.93 to 22.09)	<0.001	2.086
Model 2‡	Yes	No	6.21 (2.04 to 18.91)	0.001	1.827 (−12.4)
Model 3‡	No	Yes	6.39 (1.93 to 21.23)	0.002	1.855 (−11.1)
Model 4‡	Yes	Yes	6.09 (1.72 to 21.58)	0.005	1.806 (−13.4)

* The β-coefficient (also called the regression coefficient) is the natural log of the OR. The percent change in the regression coefficient in models 2 to 4 with adjustment for PI, GI, or both compared to the model without adjustment for PI or GI is shown in parentheses.
† Model 1 was obtained from stepwise logistic regression using RA status, age, gender, education, smoking, alcohol consumption, and BMI as independent variables and periodontitis as a dependent variable. Age and RA status remained as significant predictors in the final model.
‡ Models 2 through 4 include RA status and age, as well as PI, GI, or both as independent variables and periodontitis as a dependent variable.

disease, more recent evidence is accumulating that subjects with RA have higher odds of periodontal disease compared to non-diseased individuals,[9-13] which is in agreement with our results. However, most previous studies did not adjust for potential confounders.

In the present study, a significantly higher percentage of sites with a mean CAL >4 mm was observed in the RA group compared to the control group (35.1% versus 9.6%). Expressed as an estimate of the relative risk, in unadjusted analyses we found a significant OR of 5.70 for the association of RA with periodontal disease. This OR was substantially higher than the one we found for other potential predictors of periodontal disease, including smoking, heavy alcohol consumption, or obesity (BMI ≥30 kg/m^2). We used a mean CAL >4 mm to define periodontal disease, which corresponds to the median CAL in the present study. Nevertheless, results were similar when different cut-off points were used to define periodontal disease. We also found similar results when CAL was treated as a continuous variable using analysis of covariance (data not shown).

Furthermore, our analysis suggests that poor oral hygiene, as determined by higher PI and GI, accounts for 13.4% of the association between RA and periodontal attachment loss. These results indicate that oral hygiene may only partially account for this association and that other parameters may be among the mediators responsible for the increased prevalence of periodontal disease in individuals with RA. In recent years, evidence has accumulated that periodontal disease, similar to other inflammatory diseases, including RA, is not restricted to local tissue reaction but may have systemic impact.[3,32-35,38] Both diseases are characterized by increased secretion of proinflammatory mediators, which may explain why we found an association of RA with periodontal disease, even after adjustment for oral hygiene. Nevertheless, in the present study, adjustment for oral hygiene attenuated the relationship between RA and periodontal disease to some extent, which suggests that patients with RA may benefit from enforced individual and professional oral hygiene measures. Functional upper limb disabilities in patients with RA might contribute to poor manual dexterity with the toothbrush and a lower oral hygiene status.[39] Although some studies[9,13,17] found contrary results, other studies[11,12] are in accordance with our data, showing increased plaque accumulation in subjects with RA.

Table 5.

Rheumatologic Characteristics and Medication in Subjects With RA With Mean CAL ≤4 or >4 mm

Characteristic	CAL ≤4 mm	CAL >4 mm	*P* Value*
Disease duration (years)†	11 (4 to 19)	10 (5 to 13)	0.698
HAQ†	0.57 (0.13 to 1.25)	0.75 (0.38 to 1.00)	0.433
DAS28†	3.40 (2.50 to 5.10)	3.15 (2.70 to 4.70)	0.328
ESR† (mm/hour)	16 (10 to 30)	15 (8 to 28)	0.686
CRP† (mg/dl)	0.21 (0.09 to 0.64)	0.15 (0.10 to 0.34)	0.754
Rheumatoid factors† (IE)	70 (22 to 444)	104 (26 to 200)	0.561
Anti-CCP† (U/ml)	194.00 (33.20 to 978.00)	154.50 (15.00 to 639.50)	0.510
Corticosteroids (%)	65.4	67.7	0.851
TNF-α antagonists (%)	61.5	58.1	0.790
DMARD (%)	61.5	35.5	0.050
NSAID (%)	38.5	45.2	0.610

IE = international units; anti-CCP = anticyclic citrullinated peptides; TNF-α = tumor necrosis factor-alpha; DMARD = disease-modifying antirheumatic drugs; NSAID = non-steroidal anti-inflammatory drugs.
* *P* values are based on the Mann-Whitney *U* test for continuous data or the χ^2 test for frequency data.
† Data are medians (25th to 75th percentile).

The present study included subjects with prevalent RA with varying degrees of duration and severity of disease. However, the fact that, among subjects with RA, disease duration and clinical and laboratory parameters of disease severity were not significantly related to CAL (Table 5) indicates that the higher odds of periodontal disease among subjects with RA compared to controls may be independent of disease duration or severity. A recent study[13] found positive associations of CAL with laboratory parameters of RA when pooling subjects with aggressive periodontitis and non-diseased controls; however, that study did not provide information about these correlations among subjects with RA. Nevertheless, our data must be interpreted cautiously given that the subjects with RA were on drug treatment, which may mask any potential effect of disease severity on the odds of having periodontal disease. Drugs used to treat RA may affect the risk for periodontitis, which may confound the observed association between RA and periodontal disease. However, among subjects with RA, we observed no statistically significant association between drug use and CAL, arguing against such confounding.

Furthermore, osteoporosis could be another potential link, in addition to oral hygiene, between RA and periodontal disease. Medications taken by subjects with RA, such as glucocorticoids, are a known cause of osteoporosis, and decreased systemic bone mineral density due to osteoporosis might predispose to further bone loss in RA and periodontitis.[31,40] However, in the present study, adjustment for osteoporosis only moderately attenuated the association between RA and periodontal disease.

Our study has strengths and limitations. Among the strengths is the detailed assessment of the oral status that was performed by two independent dentists. Similarly, the subjects with RA were diagnosed by rheumatologists certified by the German Board of Physicians. Among the limitations of our study is the cross-sectional design, which complicates the drawing of causal inferences. Thus, based on our study design we cannot examine whether RA truly precedes the development of periodontal disease or vice versa. In addition, the controls included in our study were recruited from the general dental office and, therefore, may not have been representative of the general population. However, we expect that the relationship between RA and periodontal disease found in this study should be similar to men and women in general, although the strength of this association and the degree to which this association could be explained by oral hygiene may vary according to the oral hygiene of the underlying general population. The sample size of our study was relatively small, which may limit the precision of the risk estimates. Also, the sample size precluded us from adjusting for extensive sets of confounders. Therefore, we used stepwise logistic regression to select the most parsimonious model that best describes our data. However, the OR estimate for the association of RA with periodontal disease in the parsimonious model was similar to the estimate that we obtained when the non-significant covariates were forced into the regression model (data not shown). The limited sample size and the fact that controls were frequency matched to cases based on age and gender may have resulted in controls that were not representative of the general population. However, the primary intention of our study was not to estimate prevalences but to estimate the OR for the association of RA with periodontal disease independent of other predictors of periodontal disease that could potentially confound this association. Therefore, future studies with a larger sample size and a prospective design are warranted to

examine the association of RA with periodontal disease in more detail.

CONCLUSIONS

The present study suggests that patients with RA have an increased prevalence of periodontal attachment loss compared to non-diseased individuals. Further, oral hygiene may only partially account for this association, suggesting that other parameters may be among the potential mediators of this association. Nevertheless, our results indicate the need for a close collaboration among physicians, dentists, and dental hygienists when treating patients with RA.

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