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# Increased Periodontal Attachment Loss in Patients With Systemic Sclerosis

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**Background:** Patients with inflammatory rheumatic diseases and periodontitis share common pathogenetic characteristics, such as proinflammatory traits causative for tissue degradation and loss of function. The aim of the present case control study is to investigate the association between systemic sclerosis (SSc) and periodontitis.

**Methods:** The association between SSc and periodontitis was examined in 58 SSc patients and 52 control patients, matched for age and sex. The periodontal examination included periodontal attachment loss (AL), probing depth, bleeding on probing, plaque index (PI), and gingival index (GI). Potential risk factors of periodontitis were assessed through patients' questionnaires.

**Results:** In unadjusted analyses, patients with SSc had a significant 0.61 mm higher AL (95% confidence interval [CI] 0.24 to 0.97; $P = 0.002$) when compared with controls. In a stepwise logistic regression, including SSc status, age, sex, education, smoking, alcohol consumption, and body mass index, only SSc status, age, and sex remained significantly associated with periodontitis. Adjusted for age and sex, patients with SSc had a 0.52 mm higher AL compared with controls (95% CI 0.16 to 0.88; $P = 0.005$). The strength of the association of SSc with AL remained statistically significant after additional adjustment for PI (0.44 mm; 95% CI 0.02 to 0.86; $P = 0.04$) or GI (0.61 mm; 95% CI 0.24 to 0.97; $P = 0.001$).

**Conclusions:** This study demonstrates higher AL in patients with SSc, which remained significant after adjustment. The study indicates a possible relationship between SSc and periodontitis. *J Periodontol 2016;87:763-771.*

**KEY WORDS**

**Fibrosis; infection; inflammation; oral hygiene; periodontitis; risk factors; scleroderma, systemic.**

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Periodontitis is a common inflammatory disease in humans, affecting 46% of adults in the United States, with 8.9% having severe periodontitis.[1] Periodontitis is induced by an anaerobic Gram-negative bacterial biofilm on the dental root surface and is characterized by dysregulation of the host immune response.[2] Immune cell activation by bacterial pathogens initiates the production of large amounts of inflammatory mediators, such as tumor necrosis factor α, which further stimulates the release of other mediators, including proteolytic enzymes and additional dysbiosis and inflammation, resulting in soft tissue and hard tissue periodontal breakdown and eventually in tooth mobility and loss.[3] The degree of inflammation varies among individuals with periodontitis independently of the degree of bacterial infection, suggesting that alterations of the immune function may contribute substantially to the extent of periodontal disease.[4]

In recent years, it has become apparent that patients with inflammatory rheumatic diseases and periodontitis share common pathogenetic characteristics, such as proinflammatory traits causative for tissue degradation and loss of function.[5-8] Evidence from epidemiologic studies suggests that periodontal disease is more common in patients with inflammatory rheumatic diseases, such as in patients with rheumatoid arthritis.[4,9-11] Intervention studies indicate a causal relationship by showing that

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periodontal therapy reduces rheumatic disease activity and severity.[9,12-14]

Systemic sclerosis (SSc) is a chronic systemic disease of unknown etiology, characterized by skin sclerosis, vasculopathy, and complications of inner organs, such as in the lung, heart, kidney, and gastrointestinal tract.[15,16] The prevalence varies from five to 30 individuals per 100,000, with a higher prevalence among females compared with males.[17,18] Only a limited number of studies have examined the overall oral status and in particular the periodontal status in patients with SSc.[19-23] Only two recent studies investigated the association between SSc and periodontal disease, indicating a positive correlation.[22,23] SSc has been reported to be associated with an increased risk of salivary hypofunction, dental caries, and abnormalities in periodontal microcirculation.[19-21,24] It is unknown whether a limitation attributable to SSc, such as progressive limited mouth opening, impedes individual and professional oral hygiene measures, making patients with SSc more susceptible to plaque accumulation and, consequently, to inflammatory periodontal disease and SSc.[23] However, it remains an open question whether major risk factors of periodontitis, such as poor oral hygiene, may account for the association. Therefore, the present case control study aims to investigate the association between SSc and periodontitis and whether potential risk factors of periodontal diseases (e.g., poor oral hygiene, smoking, low education, daily alcohol consumption, and high body mass index [BMI]) may account for this association.

## MATERIALS AND METHODS

### *Study Population*

From 2008 to 2011, a total of 110 patients (23 males and 87 females, aged 19 to 75 years; mean age: 53.7 years) were included and assessed in the present case control study: 58 patients with SSc and 52 control patients without SSc frequency matched for age and sex as a control group. Patients with SSc were recruited from individuals with prevalent SSc, who attended the Department of Rheumatology and Clinical Immunology, Charité–Universitätsmedizin Berlin, Berlin, Germany. Control patients were recruited from patients attending the general dental outpatient office. Patients with SSc fulfilled classifications according to the recently revised European League Against Rheumatism/American College of Rheumatology criteria.[25] Based on the current recommendations of the German network for SSc, four types of the disease have been distinguished with regards to clinical outcomes: 1) limited SSc; 2) diffuse SSc; 3) overlap syndrome; and 4) sclerosis sine scleroderma.[26] Exclusion criteria for cases and controls included the following: 1) <12 teeth; 2) a history of periodontal therapy or use of antibiotics during the past 6 months before the examination; and 3) pregnancy or lactation. The study protocol was developed in full accordance to the declaration of Helsinki and was approved by the ethics committee of the Charité–Universitätsmedizin Berlin (EA 1/244/08). All study patients provided written informed consent.

### *Assessment of Clinical Rheumatologic Parameters*

Disease activity in patients with SSc was investigated by the activity index according to European Scleroderma Study Group and Research.[27] The involvement of the lung was assessed by measuring the diffusing capacity per single breath and the forced expiratory vital capacity of the lung. Thickness of the skin was assessed by the modified Rodnan skin score.[28]

### *Laboratory Examinations*

Blood plasma was collected from patients with SSc and analyzed for antinuclear antibodies and anti-RNA polymerase (SCL70) antibody by routine laboratory testing.#

### *Intraoral Examination*

Oral examinations of all study participants were performed by SK, who was trained by NP, a German Academy of Periodontology specialist for periodontology. An intraexaminer calibration was not performed. A manual periodontal probe** was used, 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), as described previously.[4,9-11] Plaque accumulation as a measurement of oral hygiene status was recorded with a plaque index (PI) according to Silness and Löe.[29] Gingival index (GI) to measure the extent of inflammatory gingivitis was evaluated according to Löe.[30] PI and GI were calculated as intra-individual means from four sites per tooth of degrees 0 to 3. Probing depth (PD) was defined as the distance from the free gingival margin to the bottom of the sulcus or periodontal pocket. The mean of all sites per individual was calculated for additional analysis. Gingival recession was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin. Periodontal attachment loss (AL) 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. Bleeding on probing (BOP) was evaluated dichotomously as bleeding present or absent 10 seconds after probing at all sites >3 mm.[31] An index was calculated between 0 (no site with BOP) and 1 (all sites with BOP). Severity categories of periodontal disease (no, mild, moderate, or severe periodontitis) were assigned

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according to Eke et al.[32] In addition, the decayed, missing, filled teeth index (DMF-T) according to Klein and evaluation of the temporomandibular joint (TMJ) (pain, crepitation, and limitation of movements) was recorded.[33,34] Because of the observational design of the study, oral radiographs were not routinely performed.

### *Assessment of Risk Factors*

Sociodemographic characteristics, lifestyle factors, and medical history were assessed by a self-administered questionnaire. Anthropometric data (height and weight) were measured.

Smoking status was classified as never smoker, former smoker, or current smoker. Alcohol consumption was classified based on the reported drinking frequency as seldom (less than or equal to once a month), moderately often (twice a month or several times a week), or daily (daily or even more frequently). BMI (calculated from the body weight in kilograms divided by the square of height in meters) was classified as normal weight (<25 kg/m$^2$), overweight (25 to <30 kg/m$^2$), or obese (≥30 kg/m$^2$). Educational attainment was classified as untrained (none), trained workers, or academic education. In addition, the assessment of the medical history included diseases that are associated with SSc.

### *Statistical Analyses*

Power calculation was performed by expecting a difference in mean AL of 0.1 mm with a standard deviation of 1.2 mm between the SSc group and the control group. Forty-eight individuals per group were examined and given a power of 80% and a level of significance of $\alpha = 0.05$. This was rounded up to 50 persons per group.

Frequency distributions, means, and standard deviations, as well as medians and interquartile ranges (IQRs), were determined to describe the data. Data distribution was checked graphically. Risk factors and dental variables were compared between patients with SSc and controls using Student unpaired *t* test for continuous normally distributed variables, Wilcoxon test for continuous variables without normal distribution, or the $\chi^2$ test for categorical variables. The association between SSc and periodontal status was assessed by linear regression procedures, taking the mean AL as continuous dependent variable and different independent variables. In a first step, the association of individuals' characteristics with the mean AL was assessed using univariate linear regression. In addition, the association among different SSc types (limited SSc, diffuse SSc) with AL was analyzed separately. In a second step, a stepwise multivariable-adjusted logistic regression was used with SSc as the primary independent variable and periodontitis as the dependent variable, with additional inclusion of age, sex, educational attainment, smoking status, alcohol consumption, and BMI into the model. In a third step, the final multivariable regression model was additionally adjusted for PI, GI, or both to estimate to what extent adjustment for these potentially intermediate variables influence the association between SSc and periodontitis. Regression analyses were performed by complete case analysis.

All *P* values presented are two tailed, and *P* values <0.05 were considered statistically significant. Analyses were performed using statistical software.††

## RESULTS

### *Sociodemographic Characteristics, Lifestyle Factors, and Aspects of Medical History of Individuals With SSc and Controls*

Sociodemographic characteristics, lifestyle factors, and aspects of medical history of patients with SSc and controls are shown in Table 1. BMI was significantly ($P = 0.03$) higher in control patients (25.3 ± 4.4 kg/m$^2$) compared with patients with SSc (23.5 ± 3.7 kg/m$^2$). Significant differences among patients with SSc and controls were also observed in educational attainment ($P = 0.02$), as well as alcohol consumption ($P = 0.02$).

### *Rheumatologic Parameters*

Clinical parameters and laboratory examinations of patients with SSc are shown in Table 2. Six patients suffered from Hashimoto thyroiditis, three from Graves disease, one from Waldenström macroglobulinemia, one from multiple myeloma, and one from sarcoidosis. Patients with SSc were under medication with low-dose glucocorticoids (62%), cyclophosphamide (31%), iloprost (28%), bosentan (19%), and methotrexate (7%) at the time of the investigation.

### *Intraoral Examination*

Table 3 shows dental variables in patients with SSc and controls. The mean AL was significantly higher in patients with SSc compared with control patients (4.01 ± 1.04 versus 3.40 ± 0.89 mm; $P = 0.002$). However, the mean PD was not statistically different between patients with SSc compared with the control group (2.99 ± 0.59 versus 3.16 ± 0.58 ($P = 0.11$). Severity of periodontitis was significantly different between the two groups ($P < 0.001$). Significantly higher plaque accumulation was found in patients with SSc (median [IQR], 0.88 [0.46 to 1.73] versus 0.35 [0.20 to 0.58]; $P < 0.001$). However, a lower GI was measured in patients with SSc (median [IQR], 0.16 [0.07 to 0.40]) compared with control patients (0.49 [0.26 to 0.73]; $P < 0.001$). Also, significantly more numbers of patients with SSc suffered from TMJ symptoms compared with control patients (55 versus 12; $P < 0.001$).

†† SAS v.9.3, SAS Institute, Cary, NC.

**Table 1.**

## Sociodemographic Characteristics, Lifestyle Factors, and Medical History of Individuals With SSc and Controls

| Characteristics | SSc (n = 58)* | Controls (n = 52)* | P† |
|---|---|---|---|
| Males | 14 (24.1) | 9 (17.3) | 0.38 |
| Age (years), mean ± SD | 55.1 ± 12.9 | 52.1 ± 13.7 | 0.23 |
| Educational attainment | | | 0.02 |
| None | 9 (15.5) | 2 (3.9) | |
| Trained worker | 33 (56.9) | 26 (50.0) | |
| Academic | 14 (24.1) | 24 (46.2) | |
| Missing value | 2 (3.4) | 0 (0) | |
| Smoking status | | | 0.06 |
| Never smoker | 26 (44.8) | 31 (59.6) | |
| Former smoker | 24 (41.4) | 11 (21.2) | |
| Current smoker | 7 (12.1) | 10 (19.2) | |
| Missing value | 1 (1.7) | 0 (0.0) | |
| Alcohol consumption frequency | | | 0.02 |
| No or low | 37 (63.8) | 23 (44.2) | |
| Moderately often | 12 (20.7) | 25 (48.1) | |
| Daily | 6 (10.3) | 4 (7.7) | |
| Missing value | 3 (5.1) | 0 (0.0) | |
| BMI (kg/m$^2$) | | | 0.02 |
| <25 (normal) | 40 (69.0) | 23 (44.2) | |
| ≥25 to <30 (overweight) | 14 (24.1) | 22 (42.3) | |
| ≥30 (obese) | 3 (5.2) | 7 (13.5) | |
| Missing value | 1 (1.7) | 0 (0) | |
| BMI (kg/m$^2$), mean ± SD | 23.5 ± 3.7 | 25.3 ± 4.4 | 0.03 |
| Hypertension | 28 (49.12) | 19 (36.5) | 0.19 |
| Diabetes mellitus | 0 (0.0) | 3 (5.8) | 0.11 |
| Dyslipidemia | 13 (23.2) | 11 (21.2) | 0.80 |
| Coronary heart disease | 5 (9.6) | 2 (3.9) | 0.44 |
| Osteoporosis | 3 (5.1) | 1 (1.9) | 0.62 |

* Data are numbers of individuals (percentages) unless stated otherwise.
† Variables were compared between cases and controls using the $\chi^2$ test or Fisher exact test (two-tailed) for categorical variables and using Student unpaired *t* test for continuous variables.

### *Association of SSc and the Individuals' Characteristics With Periodontitis*

The univariate association of SSc and individuals' characteristics with the mean AL is shown in Table 4. In univariate logistic regression analysis, patients with SSc had a 0.61-mm higher AL (95% confidence interval [CI] = 0.24 to 0.97; $P = 0.002$) when compared with control patients. According to the SSc types, patients with diffuse SSc had a 0.81-mm (95% CI = 0.39 to 1.22; $P < 0.001$) higher AL and patients with limited SSc had a 0.29-mm (95% CI = –0.29 to 0.87; $P = 0.32$) higher AL compared with control patients.

Potential risk factors of periodontitis, such as higher age, low education, smoking, daily alcohol consumption, and increased BMI, were related to AL, but only age and education were statistically significant at the 5% level.

Next, a stepwise multivariate logistic regression with SSc as the primary independent variable and AL as the dependent variable, including age, sex, education, smoking, alcohol consumption, and BMI into the model, was applied to examine the association of SSc with the odds ratio (OR) of periodontitis adjusted for potential confounders.

**Table 2.**

## Rheumatologic Characteristics and Laboratory Examinations of Patients With SSc

| Disease Characteristics of Individuals With SSc (n = 58) | n (%)* |
|---|---|
| Type of SSc | |
| Diffuse cutaneous SSc | 36 (62.1) |
| Limited cutaneous SSc | 14 (24.1) |
| Overlap syndrome | 5 (8.6) |
| Sclerosis sine scleroderma | 1 (1.7) |
| Undifferentiated SSc | 2 (3.5) |
| Disease duration (5-year categories) | |
| ≤5 | 23 (39.7) |
| >5 to 10 | 18 (31.0) |
| >10 | 9 (15.5) |
| Disease duration (years), mean ± SD | 6.2 ± 5.1 |

* Data are numbers of individuals (percentages) unless stated otherwise. Disease duration data was derived from 50 of 58 patients.

Only SSc status remained statistically significant at the 5% level associated with AL. To examine which variables were the next in a multivariable model, $\alpha$ was stepwise increased by 1%. The next variables entering into the model after adjusting $\alpha$ to 8% were age and sex. Thus, adjusted for age and sex, patients with SSc had a 0.52-mm higher AL (95% CI = 0.16 to 0.88; $P = 0.005$) compared with controls (Table 5).

To examine whether oral hygiene as a potentially intermediate factor may account for the observed association between SSc and periodontitis, the final multivariable regression model was additionally adjusted for PI, GI, or both (Table 5). The strength of the association of SSc with AL remained statistically significant after additional adjustment for plaque accumulation (0.44 mm; 95% CI = 0.02 to 0.86; $P = 0.04$), gingival inflammation (0.61 mm; 95% CI = 0.24 to 0.97; $P = 0.001$), or both plaque accumulation and gingival inflammation (0.64; 95% CI = 0.20 to 1.08; $P = 0.005$).

## DISCUSSION

In the present case control study, a significantly higher mean AL was observed in the SSc group compared with the control group (4.01 ± 1.04 versus 3.40 ± 0.89; $P = 0.002$). This association remained significant independent of demographic and lifestyle characteristics, including age, sex, education, smoking status, and alcohol consumption. When adjustments for age and sex were made, the mean AL did not change substantially (0.52 mm; 95% CI = 0.16 to 0.88 mm; $P = 0.005$).

Also, AL is considered to be a measure of the elapsed periodontal disease process rather than parameters such as PD.[35,36] Interestingly, despite the finding of higher AL in patients with SSc, clinical parameters, including increased PD, BOP, and gingival inflammation, were not significantly increased in patients with SSc. Because SSc is often associated with impaired mouth opening, personal oral hygiene may be limited, as shown in the present study by increased plaque accumulation in patients with SSc. Recently, it was shown that SSc oral hygiene measures are beneficial for patients with SSc.[37] However, in the present study, the association between SSc and AL remained statistically significant after additional adjustment for plaque accumulation, suggesting that poor oral hygiene may only partially account for the higher prevalence of periodontitis among patients with SSc and that other parameters among the mediators may be responsible for the increased prevalence of periodontitis in individuals with SSc. It remains to be analyzed whether periodontal therapy affects SSc disease activity.

In SSc, despite increased plaque accumulation, chronic fibrosis and alterations of the gingival microvasculature of the periodontal tissues may be responsible for greater periodontal breakdown, despite less acute periodontal inflammatory signs. A reduced number of periodontal capillary loops and reduced levels of vascular endothelial growth factor expression have been found in SSc.[21,38] Vascular alterations may limit the angiogenic responsiveness to plaque bacteria in periodontal tissues. In the present study, a higher number of patients with SSc with higher AL (mean AL >4 mm) were under iloprost medication compared with patients with SSc with lower AL (mean AL <4 mm) (data not shown). Administration of iloprost indicates severe vasculopathy, which could also affect periodontitis outcome. In addition, tissue fibrosis, characterized by excessive deposition of the extracellular matrix, especially fibrous collagen by pathologic fibroblasts, is the main characteristic of SSc.[39] Affecting the skin and many internal organs, fibrosis represents a major cause for the high morbidity and mortality in SSc.[15,16] Inflammation seems to play a major role in the pathologic process leading to fibrosis.[40] Fibrous alterations of the vasculature and surrounding connective tissues may lead to chronic ischemia and impairment of immune cell activation and diapedesis.[40] Increased collagen accumulation and collagen cross-linkage in SSc impairs overall tissue remodeling, specifically in response to bacterial challenge. Interestingly, similar to the present findings in patients with SSc, smokers tend to have reduced clinical signs of periodontal inflammation regardless of the levels of plaque accumulation.[41,42] Tissue fibrosis and gingival vasculopathy is responsible for the suppression of inflammatory clinical signs.[41,43] Masked clinical signs, as seen in patients with SSc, may complicate the usual approach to the periodontitis

**Table 3.**

## Dental Characteristics in Patients With SSc and Controls*

| Dental Characteristics | SSc (n = 58)* | Controls (n = 52)* | P† |
|---|---|---|---|
| AL (mm), mean ± SD | 4.01 ± 1.04 | 3.40 ± 0.89 | 0.002 |
| PD (mm), mean ± SD | 2.99 ± 0.59 | 3.16 ± 0.58 | 0.11 |
| Severity categories of periodontitis | | | <0.001 |
| No periodontitis | 0 (0) | 13 (25.0) | |
| Mild periodontitis | 7 (12.1) | 11 (21.2) | |
| Moderate periodontitis | 22 (37.9) | 22 (42.3) | |
| Severe periodontitis | 29 (50.0) | 6 (11.5) | |
| PI, median (IQR) | 0.88 (0.46 to 1.73) | 0.35 (0.20 to 0.58) | <0.001 |
| GI, median (IQR) | 0.16 (0.07 to 0.40) | 0.49 (0.26 to 0.73) | <0.001 |
| BOP, median (IQR) | 0.29 (0.2 to 0.45) | 0.41 (0.23 to 0.58) | 0.05 |
| Number of lost teeth, median (IQR) | 5.50 (2.0 to 10.0) | 5.0 (0.5 to 8.5) | 0.32 |
| Number of lost teeth categories | | | 0.20 |
| None | 6 (10.3) | 13 (25.0) | |
| 1 to 6 | 28 (48.3) | 20 (38.5) | |
| 7 to 12 | 14 (24.1) | 9 (17.3) | |
| >12 | 10 (17.2) | 10 (19.2) | |
| DMF-T index, mean ± SD | 17.66 ± 6.00 | 18.12 ± 6.85 | 0.71 |
| TMJ symptoms, n (%) mean ± SD | 55 (94.8) | 12 (23.1) | <0.001 |

* Data are numbers of individuals (percentages) unless stated otherwise.
† Variables were compared between cases and controls using the $\chi^2$ test for categorical variables, Student unpaired *t* test for continuous normally distributed variables, and Wilcoxon test for continuous variables without normal distribution.

diagnosis, which must be acknowledged by periodontists and dental hygienists.

Leung et al.[22] found increased mean PD in patients with SSc, but they did not adjust for potential risk factors. A recent study by Baron et al.[23] showed an adjusted (age, sex, ethnicity, education, and smoking) OR of 1.84 (95% CI = 1.39 to 2.43) for number of teeth with periodontitis, defined as PD >3 mm or AL ≥5.5 mm. In the present study, patients with SSc had a significantly lower BMI than control patients. Abnormal BMI predisposes to inflammatory systemic and oral diseases, such as periodontitis.[44] Low BMI reflects catabolic processes, observed in a variety of chronic diseases. Cachexia as a result of chronically inflammatory processes has been described as an important underlying mechanism in patients with SSc.[45] Patients with SSc have specific changes in their body composition, and they show signs of malnutrition and reduced energy intake, which reflects disease activity and severity.[46-48]

In recent years, evidence has been accumulated that periodontitis, similar to other inflammatory diseases including SSc, is not restricted to local tissue reaction but may have a systemic effect.[49,50] Both diseases are characterized by increased secretion of proinflammatory mediators, which in part may also explain the association. Similar to the present findings and expressed as the OR instead of AL, the OR of periodontitis was increased in patients with other inflammatory rheumatic diseases. In previous studies, OR of periodontitis was 6.81 (95% CI = 1.96 to 23.67) in spondyloarthritis ankylosans and 8.05 (95% CI = 2.93 to 22.09) in rheumatoid arthritis compared with control patients.[5,11]

The present study has strengths and limitations. Among the strengths is that the patients with SSc were assessed by experienced SSc specialists. Among the limitations is the cross-sectional design, which complicates the drawing of causal inferences. Thus, based on the study design, whether SSc truly precedes the development of periodontitis or vice versa cannot be examined. To the best of the authors' knowledge, to date, there exist no reports on the prevalence of SSc in patients with periodontitis compared with periodontal healthy individuals. In addition, a high number of the diffuse type of SSc is enrolled in the present study, which may differ from the overall distribution of SSc types. Also, controls included in the present study are recruited from the general dental outpatient office and, although matched for age and sex, may therefore not

**Table 4.**

## Linear Associations of SSc and Individuals' Characteristics With Mean AL

| Characteristics | AL (mm) (95% CI) | P |
|---|---|---|
| SSc | | |
| No | (reference) | |
| Yes | 0.61 (0.24 to 0.97) | 0.002 |
| Type of SSc | | |
| No | (reference) | |
| Diffuse SSc | 0.81 (0.39 to 1.22) | <0.001 |
| Limited SSc | 0.29 (–0.29 to 0.87) | 0.32 |
| Sex | | |
| Females | (reference) | |
| Males | 0.43 (–0.04 to 0.89) | 0.07 |
| Age (per 1 year) | 0.015 (0.00 to 0.29) | 0.04 |
| Educational attainment | | |
| None | (reference) | |
| Trained worker | –0.71 (–1.32 to –0.11) | 0.02 |
| Academic | –0.63 (–1.27 to 0.003) | 0.05 |
| Smoking status | | |
| Never smoker | (reference) | |
| Former smoker | 0.06 (–0.37 to 0.49) | 0.77 |
| Current smoker | 0.08 (–0.48 to 0.64) | 0.78 |
| Alcohol consumption frequency | | |
| Seldom | (reference) | |
| Moderately often | –0.37 (–0.78 to 0.04) | 0.08 |
| Daily | 0.43 (–0.24 to 1.01) | 0.21 |
| BMI ($kg/m^2$) | | |
| <25 | (reference) | |
| 25 to 30 | 0.20 (–0.22 to 0.62) | 0.36 |
| >30 | –0.00 (–0.69 to 0.68) | 0.99 |

CI = confidence interval.

**Table 5.**

## Adjusted AL (95% CI) of Patients With SSc Compared With Control Individuals With and Without Adjustment to Oral Hygiene Indices (PI and GI)

| | Adjustment for | | | |
|---|---|---|---|---|
| Model | PI | GI | AL (mm) for the Association of SSc With Periodontitis (95% CI) | P |
| 1* | No | No | 0.52 (0.16 to 0.88) | 0.005 |
| 2† | Yes | No | 0.44 (0.24 to 0.86) | 0.04 |
| 3† | No | Yes | 0.61 (0.24 to 0.97) | 0.001 |
| 4† | Yes | Yes | 0.64 (0.21 to 1.08) | 0.004 |

* Model 1 included SSc status, age, and sex as independent variables and mean AL as the dependent variable.
† Models 2 to 4 include SSc status, age, and sex, as well as PI, GI, or both as independent variables and periodontitis as a dependent variable.

be representative for the general population. However, it is expected that the relationship between SSc and periodontitis found in this study should be similar to males and females in general, although the strength of this association and the extent to what degree this association could be explained by oral hygiene may vary according to the extent of oral hygiene, dental problems, and the underlying general population. The sample size of this study is relatively small, and confidence intervals are relatively large, which limits the precision of the estimates. Thus, although they are statistically significant, the point estimates found in this study have to be interpreted cautiously. Therefore, future studies with larger numbers of individuals and prospective design are warranted to examine the association of SSc with periodontitis in more detail.

## CONCLUSION

The present study shows that patients with SSc, although showing fewer signs of periodontal BOP and gingival inflammation, have an increased AL compared with healthy individuals, which stayed significant after adjustment for known risk factors of periodontitis.

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