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Oral and Dental Health in Systemic Sclerosis: Is it a Window to Other Systemic Affections?

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ORIGINAL STUDY

Oral and dental health in systemic sclerosis: is it a window to other systemic affections?

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Abstract

Background: Oral and periodontal manifestations are common in systemic sclerosis (SSc), which may result in poor nutritional status and significant morbidity. A better understanding of these manifestations is crucial for better patient outcomes. We aimed to evaluate dental and oral manifestations in patients with SSc in comparison to healthy control and to study the relationship between these findings and SSc disease activity index, skin thickness, other systemic affection, as well as the oral health-related quality of life in these patients.

Patients and methods: Fifty patients with SSc were included in this case–control study (group A) and 50 healthy controls (group B). The assessment included; a modified Rodnan skin score (mRss), revised EUSTAR activity index measurements, and oral health impact profile 5 items (OHIP5). The dental and oral assessment included probing pocket depth (PPD), clinical attachment level (CAL), gingival recession height measurement (GH), gingival index (GI), mobility index, plaque index, the decayed, missing, and filled index (DMF index), bleeding on probing (BOP), dry mouth, and inter-incisal distance (ID) measurement.

Result: In group A, the dental and oral findings were statistically significantly higher compared to group B, except for the ID, which was reduced in group A. There was a negative correlation between ID and mRss and a positive correlation between CAL, BOP, and mRss. Dental parameters (PPD, CAL, GH, GI, plaque index, DMF, and BOP) correlated positively with the revised EUSTAR activity index, while ID correlated negatively. ID correlated negatively with gastrointestinal tract disorders and interstitial lung disease, while fibrosis of the buccal mucosa or the tongue, PPD, CAL, GH, GI, and DMF correlated positively. A positive correlation was found between OHIP5 and each of PPD, CAL, GH, GI, DMF, fibrosis of buccal mucosa or tongue, and negatively with ID.

Conclusion: The incidence of oral and periodontal manifestations was higher in patients with SSc than controls. An association was found between different oral and dental findings and disease activity index, skin thickness, and OHIP.

Keywords: Dental, Oral, Periodontitis, Systemic sclerosis

1. Introduction

Systemic sclerosis (SSc) is an autoimmune multisystemic rheumatic disease with an unclear etiology. It is thought to be caused by three major pathophysiological processes: immunological dysregulation, increased extracellular matrix deposition from fibroblast dysfunction and inflammation, and small vessels obstructive vasculopathy. One important finding of the condition is a thickening of the skin, which is caused by fibrosis, atrophy, and

changes in inflammation. Furthermore, internal organ fibrosis, particularly in the heart, kidneys, lungs, and gastrointestinal tract, promotes considerable morbidity and mortality [1].

Females are more likely than males to have SSc. The age range with the largest onset is 20–50 years. Its pathophysiology is thought to be caused by a variety of variables, including a genetic predisposition and environmental triggers that initiate the fibrotic and inflammatory cascades. The two primary types of SSc are diffuse cutaneous SSc and limited

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cutaneous type. Differentiation might be possible, depending on the antibodies involved, the internal organs impacted, and how the illness advances [2].

In patients with SSc, many oral and dental symptoms have been reported. Associated fibrosis and tongue stiffness, microstomia, and telangiectasia on the lips and hard palate mucosa are often reported in SSc conditions. Patients with distal esophageal dysmotility also suffer dysphagia and trouble speaking. Soft tissue involvement surrounding the temporomandibular joint (TMJ) is the cause of pseudoankylosis or reduced mandibular motion. Masticatory muscles can also be severely impaired by fibrosis. Consequently, at the masseter grasp position, the mandibular angle resorbs. Patients with SSc frequently experience hyposalivation and salivary gland fibrosis, which can lead to periodontitis, oral candidiasis, and a higher risk of dental caries [3].

The oral health-related quality of life (OHRQoL) of SSc patients is affected in areas such as eating, chewing, speaking, and the capacity to incise big solid foods by these common and unique oral and dental manifestations. This is particularly common in cases of xerostomia, which may result in affectionate feeding. According to Albilia et al. [4], microstomia also makes it challenging for patients to receive dental care from a dentist and to maintain regular oral hygiene.

Oral symptoms of SSc are rarely addressed in clinical practice and are sometimes obscured by other systemic complications. Moreover, management of them is not discussed in general therapy recommendations. For better patient outcomes, more research, as well as awareness of the different oral manifestations are essential [5].

This work aimed to evaluate oral and dental manifestations in patients with SSc in comparison to healthy control and to study the relationship between these findings and SSc disease activity index, skin thickness, and other systemic affection, as well as the OHRQoL in these patients.

2. Patients and methods

Fifty SSc patients were included in this case–control study (group A) based on their diagnosis as per the European League Against Rheumatism/American College of Rheumatology criteria [6]. Patients were chosen from the outpatient clinics of the Banha Teaching Hospital in Banha, Egypt's Departments of Rheumatology, Oral Medicine, and Periodontology. Fifty age-matched and sex-matched, apparently healthy persons (relatives of the patients with the same background) constitute

the control group (group B). Patients must be older than 18, have had an SSc diagnosis for at least a year, can tolerate dental and oral examination, and be willing and able to provide written informed permission to be included in this study. Patients with congenital oral anomalies, patients diagnosed with overlap syndrome, patients with pregnancy or nursing status, patients with diabetes, heart, liver, or renal illnesses, and heavy smokers were excluded from this study. Following a description of the study to each participant, they all signed an informed consent form. The Banha Teaching Hospital's Ethics Committee for Scientific Research approved this study.

2.1. Methods

The patients underwent the following evaluations:

- (1) A thorough history that included dental hygiene status and drug history.
- (2) A general and musculoskeletal examination, which included TMJ click or tenderness.
- (3) The modified Rodnan skin score (mRss) was used to assess skin thickness. On a scale of 0–3, with a maximum score of 51, the thickness of the skin was measured at 17 distinct locations [7].
- (4) The revised EUSTAR activity index was used to assess disease activity; according to Valentini et al. [8], a score of more than or equal to 2.5 denotes an active disease.
- (5) Based on the patient's examination and symptoms, systemic affection is assessed. Tests for systemic affection may include serum creatinine, bedside urine protein, thoracic high-resolution computed tomography, carbon monoxide diffusing capacity, ECG, and Doppler echocardiogram.
- (6) Oral health impact profile 5 items (OHIP5) assessed the OHRQoL. The questionnaire, comprising five questions corresponding to each of the four measurements, psychosocial impact, orofacial pain, orofacial appearance, and oral function, was given to patients in both the patient and control groups. According to Alhajj et al. [9], each question responded on a scale of 0 for never to 4 for very frequently.
- (7) Every participant underwent an oral and dental examination by an expert dentist blind to the patient's diagnoses. The following were the parameters of the examination.

2.1.1. Probing pocket depth

The probing pocket depth (PPD) was measured on the mesial, buccal, distal, and lingual surfaces of

each tooth in the arch, starting at the free gingival edge and moving toward the end of the pocket. Using a force of roughly 25 g, the standard William's graduated periodontal probe was used, with the long axis parallel to the tooth being examined [10].

2.1.2. Clinical attachment level

The attachment level was determined using the standard William's graduated periodontal probe, measuring from the cementoenamel junction to the sulcus base.

2.1.3. Gingival recession height measurement

A millimeter measurement of the gingival margin's distance from the cementoenamel junction was used to establish the following factors, which were then utilized to grade the gingival recession height (GH) using [11] categorization method:

- (1) Class I: the mucogingival junction (MGJ) is not reached by marginal tissue recession, and full root coverage is expected. In the interdental space, there is no loss of periodontal tissue, either bone or soft.
- (2) Class II: no periodontal loss in the interdental space, complete root coverage, and marginal tissue recession that generally reaches or exceeds the MGJ.
- (3) Class III: the MGJ is reached or exceeded by the marginal tissue recession. The inability to achieve 100% root coverage is hindered by the existence of bone or soft tissue loss in the interdental gap and by misaligned teeth.
- (4) Class IV: the marginal tissue recession reaches or exceeds the MGJ. Root coverage cannot be predicted due to severe tooth misalignment and/or bone or soft tissue loss in the interdental region.

2.1.4. Gingival index

The measure of gingival inflammation was evaluated using the Löe and Silness [12] gingival index (GI), which had a range of 0–3.

0: typical gingival.

1: minor edema, a tiny color shift, and minor irritation without bleeding on probing (BOP).

2: moderate inflammation manifests as glazing, edema, redness, and bleeding when prodded.

3: severe inflammation, noticeable edema, bleeding, and redness with a propensity for spontaneous hemorrhage.

2.1.5. Mobility index

Grade 0: no noticeable mobility.

Grade 1: notable mobility less than 1 mm in the buccolingual direction.

Grade 2: more than 1 mm but does not exceed 2 mm.

Grade 3: depressibility in the socket or more than 2 mm [11].

2.1.6. The plaque index [13]

0: there is no plaque.

1: a coating of plaque that adheres to the free gingival edge and surrounding tissue of the tooth. Plaque cannot be observed in situ until the disclosing solution has been applied or until the tooth surface has been probed with the probe.

2: there is a moderate soft deposits accumulation around the tooth and gingival edge that is seen with the unaided eye inside the gingival pocket.

3: a lot of soft matter is present in the gingival pocket, gingival edge, or teeth [13].

2.1.7. Dry mouth

Physical observation was used to evaluate cases of dry mouth [14].

2.1.8. The decayed, missing, and filled index

The decayed (D), missing (M), and filled (F) (DMF) refer to the total number of teeth or surfaces for an individual [15].

2.1.9. Bleeding on probing [16]

0: there is no bleeding.

1: there is only one obvious area of bleeding.

2: there are numerous small spots of blood or separate bleeding sites.

3: shortly after probing, blood filled the interdental triangle.

4: prolonged bleeding, blood begins to flow towards the margin of the gingiva.

2.1.10. Interincisal distance

To prevent bias caused by prolonged mouth stretching, the interincisal distance (ID) was measured at the start of the examination. The distance between the incisal margins of the upper central tooth and the lower central tooth is known as the ID measured after patients were instructed to open their mouths as wide as they could [17].

For statistical analysis, SPSS 24 (IBM, Armonk, New York, USA), was utilized. Quantitative data was expressed as mean \pm SD. The qualitative data were expressed using percentages and frequencies. An independent sample *t* test of significance was utilized to compare the two means. To assess the proportions between the two qualitative variables, a

significant χ^2 test was utilized. The Pearson's correlation coefficient test was used to correlate the data. It was determined that there is a statistically significant difference when the likelihood value is smaller than 0.05.

3. Result

In this study, two groups were included: 50 patients with SSc made up group A. There were four males and 46 females in this group, ages 45–56. Fifty individuals were included in group B, the control group, with ages ranging from 44 to 56 (47 female and three male). Ninety-two percent of the control group (group B) and 100% of our patients (group A) had poor oral hygiene. The two groups' ages, sexes, and dental hygiene status did not statistically differ, as indicated by [Table 1](#).

3.1. Disease characteristics of group A

Ninety-four of our patients had diffuse SSc, while 6% had limited type. The disease duration ranged between 5 and 11 years. The disease activity index measured by the revised EUSTAR index ranged from 7.75 to 10, and all our patients had an active disease. All our patients were receiving a combination of antirheumatic medications as shown in [Table 2](#). Other disease characteristics are also shown in [Table 2](#).

3.2. Oral and dental findings

[Table 3](#) shows the different oral and dental findings in the two groups. All oral and dental parameters were significantly higher in group A (patients) in comparison to group B (control), except for ID, which was lower in group A. The OIHP5 was statistically significantly higher in group A compared to group B ([Figs. 1–4](#)).

[Figures 1–4](#) show the dental and oral findings.

Table 1. Demographic data of the two groups.

| Demographics | Group A [n (%)] | Group B [n (%)] | t/ χ^2 | P |
|---------------------|------------------|-----------------|-------------|-------|
| Sex | | | | |
| Female | 41 (82) | 42 (84) | 0.070 | 0.791 |
| Male | 9 (18) | 8 (16) | | |
| Age (years) | | | | |
| Mean \pm SD | 52.07 \pm 3.86 | 50.9 \pm 3.48 | 1.230 | 0.224 |
| Range | 45–56 | 44–56 | | |
| Oral hygiene status | | | | |
| Poor | 50 (100) | 46 (92) | 0.002 | 0.967 |
| Good | 0 | 4 (8) | | |

P value less than 0.05 is statistically significant.

Table 2. Group A: disease characteristics.

| Disease characteristics | |
|--|-----------------------------------|
| Disease duration (years) (range, mean \pm SD) | 5–11 (7.92 \pm 2.23) |
| Systemic sclerosis form [n (%)] | Diffuse: 47 (94) Limited 3 (6) |
| Revised EUSTAR index (range, mean \pm SD) | 7.75–10 (9.5 \pm 0.92) |
| Active disease: value \geq 2.5 = active disease [n (%)] | 50 (100) |
| Modified Rodnan score (range, mean \pm SD) | 28–38 (34 \pm 2.26) |
| Musculoskeletal [n (%)] | 41 (82) |
| GIT [n (%)] | 34 (68) |
| Raynaud's [n (%)] | 40 (80) |
| Digital ulcers [n (%)] | 41 (82) |
| Renal [n (%)] | 12 (24) |
| Interstitial lung disease [n (%)] | 33 (66) |
| Pulmonary artery hypertension [n (%)] | 16 (32) |
| Cardiac [n (%)] | 12 (24) |
| Medication [n (%)] | |
| Methotrexate | 50 (100) |
| Cyclophosphamide | 46 (92) |
| Cyclosporine | 38 (76) |
| Mycophenolate mofetil | 50 (100) |

GIT, gastrointestinal tract.

The relationship between the different dental and oral findings and each of the disease characteristics and OHIP.

A correlation analysis was done to determine the association between each of the various oral and dental findings and each of the:

- (1) Skin thickness measure by mRss showed a negative correlation between ID and mRss, while clinical attachment level (CAL) and BOP correlated positively with mRss.
- (2) Disease activity index measured by revised EUSTAR activity index showed a positive correlation between PPD, CAL, GH, GI, plaque index (PI), DMF, and BOP and the disease activity index. While the ID correlated negatively with the disease activity index.
- (3) Systemic affection: the ID correlated negatively with mRss, gastrointestinal disorders, and interstitial lung disease. Fibrosis of the buccal mucosa or the tongue, PPD, CAL, GH, GI, and DMF correlated positively with gastrointestinal disorders and interstitial lung disease. No correlation was found with pulmonary artery hypertension, digital ulcers, renal affection, and Raynaud's disease.
- (4) OHRQoL measured by OHIP5: this showed a positive correlation between PPD, CAL, GH, GI, DMF, and fibrosis of buccal mucosa or tongue and OHIP5. There was a negative correlation between ID and OHIP5 ([Table 4](#)).

Table 3. Dental and oral findings.

| Variables | Group A | Group B | <i>t</i> test/ χ^2 | <i>P</i> |
|---|--------------------|------------------|-------------------------|----------|
| OHIP5 | 14–20 (17.3 ± 1.6) | 0–3 (0.8 ± 1.03) | 61.31 | 0.001 |
| ID | 36.23 ± 3.5 | 44.43 ± 5.5 | 8.894 | 0.001 |
| PPD (range, mean ± SD) | 5–7 (6.1 ± 0.84) | 1–5 (1.9 ± 1.16) | 16.074 | 0.001 |
| CAL (range, mean ± SD) | 5–12 (8.4 ± 2.44) | 0–3 (0.3 ± 0.92) | 17.001 | 0.001 |
| DMF (range, mean ± SD) | 5–12 (8.77 ± 2.21) | 0–7 (3.7 ± 1.76) | 9.819 | 0.001 |
| Mobility index [<i>n</i> (%)] | | | | |
| 0 | 8 (16) | 35 (70) | 43.846 | 0.001 |
| 1 | 0 | 15 (30) | | |
| 2 | 15 (30) | 0 | | |
| 3 | 27 (54) | 0 | | |
| Gingival index [<i>n</i> (%)] | | | | |
| 0 | 0 | 27 (54) | 60.001 | 0.001 |
| 1 | 0 | 23 (46) | | |
| 2 | 15 (30) | 0 | | |
| 3 | 35 (70) | 0 | | |
| Plaque index [<i>n</i> (%)] | | | | |
| 0 | 8 (16) | 22 (44) | 45.556 | 0.001 |
| 1 | 0 | 28 (56) | | |
| 2 | 10 (20) | 0 | | |
| 3 | 32 (64) | 0 | | |
| Gingival recession height [<i>n</i> (%)] | | | | |
| I | 0 | 45 (90) | 77.5002. | 0.001 |
| II | 12 (24) | 5 (10) | | |
| III | 9 (18) | 0 | | |
| IV | 29 (58) | 0 | | |
| Bleeding on bopping [<i>n</i> (%)] | | | | |
| 0 | 8 (16) | 25 (50) | 65.6742 | 0.001 |
| 1 | 0 | 25 (50) | | |
| 2 | 9 (18) | 0 | | |
| 3 | 33 (66) | 0 | | |
| 4 | 0 | 0 | | |
| Dry mouth [<i>n</i> (%)] | 37 (74) | 0 | 58.143 | 0.001 |
| TMJ [<i>n</i> (%)] | 33 (66) | 0 | 48.761 | 0.001 |
| Fibrosis of tongue or buccal mucosa [<i>n</i> (%)] | 26 (52) | 0 | 5.927 | 0.001 |
| Telangiectasia | 38 (76) | 0 | 7.829 | 0.001 |

CAL, clinical attachment level; DMF, decay missing filled index; ID, interincisal distance; OHIP5, oral health impact profile 5; PPD, probing pocket depth; TMJ, temporomandibular joint. *P* value less than 0.05 statistically significant.



Fig. 1. Periodontal pocket.



Fig. 2. Caries on tooth.



Fig. 3. Inflamed gingival, plaque, and recession.



Fig. 4. Restricted mouth opening.

4. Discussion

This study aimed to compare oral and dental findings between patients with SSc and healthy controls. Other risk factors for abnormalities in the oral cavity and teeth, such as smoking and systemic disorders, were not included in our population. No statistically significant difference was found in the oral hygiene level between the control and the patient groups.

Regarding the oral manifestations, all oral parameters were higher in patients with SSc (telangiectasia, dry mouth fibrosis, TMJ dysfunction, and dry mouth). However, our patients' ID was reduced compared to the control group.

Telangiectasia was our patient's most common oral finding, affecting 76% of them. This finding agrees with Leung et al. [18] study that found telangiectasia in 80% of the patients with SSc included in this study. Telangiectasia was also found in 84 and 81% of patients in the studies by Gomes da Silva et al. [1] and Chu et al. [19], respectively.

Sicca symptom is a prevalent complaint associated with SSc, although their frequency is unknown. Despite the fact that the study did not measure salivary flow rate, physical examination revealed that 74% of our patients had dry mouths. In contrast to Bajraktari et al. [20], who observed this oral manifestation in 32% of SSc patients, and Weisman and Calcaterra [21], who found Sicca syndrome features in only 12% of SSc patients, xerostomia was commonly reported in our study. Our finding

Table 4. Correlation study between oral and dental findings and disease characteristics and oral health impact profile.

| | PPD | CAL | GH | GI | MI | PI | DMF | BOP | Fibrosis | ID | Telangiectasia |
|----------------|-------|-------|-------|-------|--------|--------|-------|-------|----------|--------|----------------|
| DAI | | | | | | | | | | | |
| <i>r</i> | 0.559 | 0.506 | 0.606 | 0.454 | 0.232 | 0.393 | 0.411 | 0.393 | 0.241 | -0.519 | -0.126 |
| <i>P</i> | 0.000 | 0.000 | 0.000 | 0.001 | 0.104 | 0.005 | 0.003 | 0.005 | 0.091 | 0.000 | 0.383 |
| mRss | | | | | | | | | | | |
| <i>r</i> | 0.193 | 0.574 | 0.182 | 0.156 | 0.166 | 0.158 | 0.171 | 0.158 | 0.135 | -0.713 | 0.01 |
| <i>P</i> | 0.179 | 0.000 | 0.205 | 0.279 | 0.249 | 0.273 | 0.235 | .0000 | 0.349 | 0.000 | 0.945 |
| GIT | | | | | | | | | | | |
| <i>r</i> | 0.811 | 0.732 | 0.693 | 0.817 | 0.124 | 0.173 | 0.764 | 0.153 | 0.817 | -0.478 | -0.108 |
| <i>P</i> | 0.000 | 0.000 | 0.000 | 0.000 | 0.391 | 0.229 | 0.000 | 0.288 | 0.000 | 0.001 | 0.455 |
| ILD | | | | | | | | | | | |
| <i>r</i> | 0.590 | 0.732 | 0.901 | 0.816 | 0.187 | 0.253 | 0.764 | 0.173 | 0.808 | -0.466 | -0.207 |
| <i>P</i> | 0.000 | 0.000 | 0.000 | 0.000 | 0.193 | 0.076 | 0.000 | 0.229 | 0.000 | 0.001 | 0.149 |
| PHTN | | | | | | | | | | | |
| <i>r</i> | 0.137 | 0.202 | 0.147 | 0.208 | 0.124 | 0.226 | 0.240 | 0.227 | 0.07 | -0.120 | 0.01 |
| <i>P</i> | 0.342 | 0.159 | 0.308 | 0.147 | 0.390 | 0.114 | 0.093 | 0.112 | 0.629 | 0.406 | 0.945 |
| Renal | | | | | | | | | | | |
| <i>r</i> | 0.166 | 0.180 | 0.151 | 0.258 | 0.108 | 0.270 | 0.214 | 0.269 | 0.1 | -0.075 | -0.258 |
| <i>P</i> | 0.249 | 0.210 | 0.295 | 0.070 | 0.455 | 0.057 | 0.135 | 0.058 | 0.489 | 0.604 | 0.070 |
| Digital ulcers | | | | | | | | | | | |
| <i>r</i> | 0.093 | 0.215 | 0.234 | 0.258 | -0.104 | -0.067 | 0.107 | 0.067 | 0.258 | 0.075 | 0.261 |
| <i>P</i> | 0.520 | 0.133 | 0.101 | 0.070 | 0.472 | 0.643 | 0.459 | 0.641 | 0.070 | 0.604 | 0.067 |

(continued on next page)

Table 4. (continued)

| | PPD | CAL | GH | GI | MI | PI | DMF | BOP | Fibrosis | ID | Telangiectasia |
|-----------------|-------|-------|-------|-------|--------|--------|-------|--------|----------|--------|----------------|
| Raynaud's | | | | | | | | | | | |
| <i>r</i> | 0.273 | 0.214 | 0.238 | 0.258 | -0.102 | -0.067 | 0.107 | -0.067 | 0.258 | 0.075 | -0.258 |
| <i>P</i> | 0.055 | 0.135 | 0.096 | 0.070 | 0.480 | 0.643 | 0.457 | 0.643 | 0.0704 | 0.601 | 0.070 |
| Musculoskeletal | | | | | | | | | | | |
| <i>r</i> | 0.273 | 0.214 | 0.238 | 0.258 | 0.207 | 0.237 | 0.222 | 0.237 | 0.258 | 0.075 | -0.220 |
| <i>P</i> | 0.055 | 0.135 | 0.096 | 0.070 | 0.1475 | 0.097 | 0.121 | 0.097 | 0.417 | 0.070 | 0.124 |
| OHIP | | | | | | | | | | | |
| <i>r</i> | 0.709 | 0.508 | 0.611 | 0.628 | 0.276 | 0.267 | 0.509 | 0.256 | 0.711 | -0.659 | 0.054 |
| <i>P</i> | 0.000 | 0.000 | 0.000 | 0.001 | 0.052 | 0.060 | 0.000 | 0.072 | 0.000 | 0.000 | 0.708 |

BOP, bleeding on probing; CAL, clinical attachment level; DAI, disease activity index; DMF, decay missing filled index; GH, gingival recession height; GI, gingival index; GIT, gastrointestinal tract; ID, interincisal distance; ILD, interstitial lung disease; MI, mobility index; mRss, modified Rodnan skin score; OHIP, oral health impact profile 5; PHTN, pulmonary artery hypertension; PI, plaque index; PPD, probing pocket depth.

P value less than 0.05 statistically significant.

agrees with Wood and Lee [22] study, which revealed that 70% of their patients had xerostomia, and they connected this finding to a greater incidence of dental caries. In a study conducted by Avouac et al. [23], biopsy tissues from 78 patients with SSc who reported subjective xerostomia symptoms were analyzed; glandular fibrosis accounted for 58% of these cases and Sjögren's syndrome for 23% of the cases. These findings imply that the major etiology of Sicca syndrome in SSc patients is glandular fibrosis, not Sjögren's syndrome-associated lymphocytic sialadenitis.

Fifty-two percent of our patients had either tongue or buccal mucosa fibrosis, consistent with the 50% of patients who had fibrosis in the Jagadish et al. [24] study. The Crincoli et al. [25] study showed that only 26.3% of the patients had fibrosed tongues, which reflects the variability between the different studies.

There are differences in the reported frequencies of TMJ disorders. Crincoli et al. [25] discovered that 92.5% of their patients had temporomandibular problems, but Chu and colleagues found that only 7% of the included patients (42 individuals) had such abnormalities. Sixty-six percent of the participants in the current study had abnormalities with their TMJs. SSc differs from other rheumatological illnesses, such as psoriatic arthritis or rheumatoid arthritis, in that the pathophysiology of TMJ dysfunction is not related to joint inflammation. SSc is mostly caused by fibrosis of the skin, lips, and subcutaneous tissue in conjunction with a malfunction of the masticatory system; however, synovia and bone resorption may also have an impact on the TMJ in addition to atrophy and fibrosis [25].

One common finding in SSc cases is limited mouth opening, which may be related to the disease's characteristic skin thickening. Lip and cheek elasticity gradually diminishes, gradually reducing in the maximum mouth opening and lip perimeter

as these areas become harder. The ID was markedly reduced in our patients compared to controls, which agrees with previous studies by Marcucci and Abdala [26]; Baron et al. [27].

Regarding the dental manifestations, comparison of different studies is difficult due to the variation in the periodontal disease definition and the variation in the evaluation parameters. In our study, all the dental findings were significantly higher in our patients than in the control group. This result is consistent with one of the largest case-control studies [28], which comprised 231 controls and 163 SSc patients. In our study, patients with SSc had higher levels of periodontal disease as assessed by CAL, PPD, and DMF. It also concluded that SSc is a reliable indicator of tooth loss and dental attachment loss. Our results agree with Isola et al. [29], who included 55 controls and 54 cases. According to this study, CAL, PPD, PI, and BOP are all higher in SSc patients. Leung and colleagues case-control study had 36 patients who had higher BOP and PPD than the control group. In the Gomes da Silva et al. [1] study, patients with SSc had higher CAL and PAL, but the PPD, BOP, and gingival bleeding index were lower than those of the control group. The vasculopathy and enhanced collagen deposition associated with SSc explained the unique clinical profile of these patients. Another study by Chu et al. [19] revealed that while patients with SSc had higher rates of periodontal disease than the controls, no statistically significant difference was found between the two groups. All the participants in this study had calculus deposits none of them had healthy periodontium and most required advanced periodontal care as they had periodontal pockets.

Several factors can explain the prevalence of periodontal disease in patients with SSc. The restricted mouth opening is one of the factors contributing to the declining periodontal status in

patients with SSc. This restriction makes it challenging for patients to practice good oral hygiene daily and for dentists to perform dental exams and treatments. Furthermore, xerostomia always developed in SSc, which could be due to salivary gland fibrosis or secondary Sjögren's syndrome. Reduced salivary flow reduced the mouth cavity's capacity to self-clean and disrupted the buffer's equilibrium, creating an environment favorable for bacterial colonization and fuelling periodontal and dental pathologies [23]. Another concurrent symptom of SSc patients is esophageal reflux, which increases the acidity of the oral cavity and promotes the growth of periodontal pathogenic organisms [30]. It was found that SSc and periodontitis were similar. Studies show that these two chronic illnesses may share several pathomechanisms, including increased levels of circulating inflammatory markers like C-reactive protein, interleukin (IL)-6, 1, 17, and tumor necrosis factor- α [31]. In gingiva samples from SSc patients, there was increased gingival infiltration and microvessel density; however, there was a decrease in vascular endothelial growth factor expression, which resulted in vessel degeneration. Increased periodontal diseases may result from vascular changes that impede the angiogenic response to bacteria in periodontal tissues [32]. This has accelerated the breakdown of the alveolar bones and the periodontal ligaments. Furthermore, studies on bone metabolism, found that SSc patients had higher levels of soluble receptor activator for nuclear factor kappa beta ligand, which was connected to osteoporosis in SSc [33]. Similarly, in periodontitis, where the resorption of the periodontal bone and the loss of tooth result, receptor activator for nuclear factor kappa beta ligand was also an osteoclast-promoting mediator [34].

There are different measures of OHRQoL; in our study, we used the OHIP5, which is a short version of the original 49-item oral health profile. OHIP5 comprises five questions that correspond to the four measurements: psychosocial impact, oral function, orofacial appearance, and orofacial pain. Although it only contains 10% of the instrument's original components, it can still be able to extract nearly 90% of the data. This increases its acceptability and attractiveness as a tool for effective OHRQoL evaluation. Our patients showed a higher impact on OHRQoL in comparison to healthy controls. This finding agrees with a study by Baron et al. [28], which showed that patients with SSc had a greater impact on oral health quality of life measured by the OHIP's overall score and its included seven subscales. A similar finding was found in a study by Abdouh

et al. [35] that included 100 patients with SSc, where the OHRQoL was measured by OHIP14, the Mouth Handicap in Systemic Sclerosis, the Hospital Anxiety and Depression Scale, the general psychological health-related questionnaires (MDAS), and the Oral Impact on Daily Performance and concluded that OHRQoL and general quality of life are negatively impacted by SSc in affected individuals. A positive correlation was also found between OHIP5 and different dental and oral findings (fibrosis, PPD, CAL, GH, GI, and DMF), while it correlated negatively with ID. This data indicates that, even though they are not life-threatening, oral manifestations, and reduced OHRQoL are significant for SSc patients and require appropriate assessment and care.

Additionally, this study aimed to explore the relationship between dental and oral findings and each of the disease activity, the skin thickness, and other systemic affections. To the best of our knowledge, there are few studies that evaluated this relationship.

In the current study, there was a negative correlation between ID and skin thickness (mRss), which agrees the study by Türk et al. [36], and this finding was explained by fibrosis of the skin around the mouth, masticatory muscles, and buccal submucosa. Furthermore, dental parameters, including CAL and BOP correlated positively with mRss, which agrees with a study by Isola et al. [29] which found an association between mRss and PPD, CAL, and BOP. Restricted mouth opening in SSc is related to skin thickening and fibrosis in the perioral area (which is included in mRss), along with fibrosis of the masticatory muscles and buccal submucosa. Restricted mouth opening impairs proper oral hygiene and promotes periodontal diseases, which can explain this association.

The relationship between orodental findings and systemic affection was evaluated in this study. It showed a negative relation between the ID and interstitial lung disease and gastrointestinal disorders, while fibrosis of the buccal mucosa or the tongue and dental parameters (PPD, CAL, GH, GI, and is DMF) correlated positively with both interstitial lung disease and gastrointestinal disorders. A 2020 study by Türk and colleagues also found an association between interstitial lung disease and reduced mouth opening. This finding can be explained by fibrosis, one of the major pathologies in SSc that involves the skin, internal organs, and the submucosa. In 2015, a study by Baron and colleagues were conducted to evaluate the relationship between various disease characteristics in SSc and orofacial manifestations. The study revealed an association between GERD and tooth loss not tooth decay.

GERD can lead to tooth erosions, raise the acidity of the oral cavity, and increase the growth of harmful microorganisms that result in periodontal disease. Restricted mouth opening, collagen deposition, fibrosis, skin atrophy, and loss of mucosal elasticity all contribute to poor dental hygiene and periodontitis. Periodontal tissue deterioration may be caused by changes in the gingival tissues' microcirculation and vascular defects. Less periodontal capillaries and decreased vascular endothelium growth factor expression are found in the periodontal tissues of patients with SSc, which may restrict the angiogenic defensive response to oral bacteria.

To our knowledge, no previous study has been conducted to investigate the association between SSc disease activity and oral and dental findings. To evaluate disease activity, we employed the updated EUSTAR activity index. In this study, a positive correlation was found between disease activity and the following dental findings; PPD, CAL, GH, GI, PI, DMF, and BOP, while the disease activity correlated negatively with ID. The relationship this study found between several oral and dental factors and each of skin thickness and interstitial lung disease may explain this association as the activity index that was used in this study included mRss, skin changes within the previous month, and carbon monoxide diffusing capacity. To improve management and get a deeper understanding of the connection between periodontal disorders and SSc, more research on this relationship and the impact of periodontitis treatment on the disease's activity is necessary.

This study has some limitations. For example, it did not evaluate the salivary flow rate or the oral microbiota microbiologically. We also did not study the difference in oral and dental findings between different subtypes of SSc as the number of patients with limited SSc was small. Additionally, we did not investigate how various anti-rheumatic drugs affected dental and oral symptoms. Another limitation is that this study is only conducted at one center.

Although this study included a fairly large number of patients, given the rarity of the disease, we recommend larger, multicentre studies with further assessment of the relationship between the disease characteristics of SSc and different oral and dental manifestations. In addition, we recommend a regular dental examination and prompt treatment of any periodontal disorder in patients with SSc.

4.1. Conclusion

This study showed that patients with SSc have higher dental and oral manifestations and a greater

impact on OHRQoL than health control. Different orodental parameters were related to skin thickness, disease activity, and systemic affection.

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Conflict of interest

The authors declared no conflicts of interest concerning the authorship and/or publication of this article.

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