Quality Resource Guide

Dental Management of the Patient with Systemic Sclerosis

Author Acknowledgements

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Educational Objectives

Following this unit of instruction, the practitioner should be able to:

1. Understand basic physiologic elements of autoimmunity.
2. Recognize the importance of the medical history and clinical exam in assessing the patient with systemic sclerosis.
3. Recognize potential oral findings associated with systemic sclerosis.
4. Recognize the implications of systemic sclerosis on dental treatment planning and prognosis.

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Autoimmunity Overview

The term autoimmunity literally means "immunity against self" and approximately 10% of the population has an autoimmune disorder.[1] Up to a third of these patients experience polyautoimmunity (concurrent presence of two or more autoimmune diseases).[2] Overall, women are four times more likely to be afflicted with an autoimmune disease than men.[3] Autoimmunity occurs as consequence of a lack of adequate elimination of autoreactive T cells during immunological maturity and/or an alteration of the normal homeostatic balance between autoreactive T cells and T regulatory cells.[4,5] Most autoimmune diseases are highly complex disorders thought to arise from a combination of patient genetic predisposition and influence of environmental factors.

The classic manifestation of an autoimmune disease is inflammation and disease specific clinical manifestations that are dictated by implicated autoreactive T cells.[6] Patients suffering an autoimmune disease often experience some degree of waxing and waning throughout the course of their clinical disease, a likely reflection of the underlying struggle between autoreactive T cells and protective T regulatory cells, both promoted by the disease. A listing of the more common autoimmune diseases is summarized in **Table 1**.[6] Some autoimmune disorders such as vitiligo and alopecia cause largely cosmetic issues, and others such as lupus and systemic sclerosis may prove fatal.

Systemic Sclerosis

Systemic sclerosis (SSc), or scleroderma, is an uncommon autoimmune disease characterized by vascular abnormalities and progressive fibrosis of the skin and internal organs.[7] The estimated prevalence of SSc is 242 cases per million adults with an incidence of 20 new cases per million adults annually.[8] The disease predominately affects women with an average age of onset of 45 years and is associated with significant morbidity and mortality. The most readily observable vascular dysfunction of SSc is Raynaud's phenomenon (RP). RP is a frequently painful condition of arterial constriction in the fingers, feet, nose, or earlobes typically precipitated by stress or cold temperatures.[9] The clinical pattern of RP is characterized by sequential triphasic color changes associated with initial constriction ("white"), followed by acrocyanosis ("blue"), and eventual reperfusion hyperemia ("red")."[8] Over time, RP may result in painful digital ischemia and ulceration. While RP may present as an isolated condition, >95% of patients with SSc will manifest RP.

Table 1 - Some Common Autoimmune Diseases[6]

Alopecia areata
Autoimmune hemolytic anemia
Autoimmune hepatitis
Dermatomyositis
Diabetes type 1
Glomerulonephritis
Grave's disease
Guillian-Barré syndrome
Idiopathic thrombocytopenia purpura
Myasthenia gravis
Multiple sclerosis
Pemphigus
Pemphigoid
Pernicious anemia
Polyarteritis nodosa
Polymyositis
Primary biliary cirrhosis
Psoriasis
Rheumatoid arthritis
Systemic Sclerosis
Sjögren's syndrome
Systemic lupus erythematosus
Some forms of thyroiditis
Some forms of uveitis
Vitiligo
Wegener's granulomatosis

Clinical Manifestations

The clinical presentation of SSc is highly variable, but two predominant subsets are recognized: limited cutaneous SSc (lcSSc) and diffuse cutaneous (dcSSc).[7] The patient with lcSSc demonstrates skin involvement distal to the elbows and knees, while the patient with dcSSc demonstrates more extensive skin involvement that typically includes the proximal limbs and/or trunk. The patient with dcSSc is more likely to manifest more frequent and often earlier internal organ involvement, while the classical features of CREST (**C**alcinosis cutis, **R**aynaud phenomena, **E**sophageal dysmotility, **S**celerodactyly, and **T**elagiectasia) syndrome (**Table 2**)[10] are more commonly observed in a patient with lcSSC.[1]

Virtually every patient with SSc will manifest extracutaneous manifestations of the disease, with frequent involvement of the musculoskeletal, pulmonary, renal, cardiac, and gastrointestinal (to include the oral cavity) systems.[8-13]

Musculoskeletal: Frequently (>50%) observed early signs and symptoms of SSc include hand swelling, arthralgia, myalgia and fatigue.[11] Disease-induced fibrosis around the tendons and periarticular structures results in contractures, typically affecting the fingers, but also potentially involving the wrists, elbows, knees and ankles. The presence of inflammatory arthritis and tendon involvement is more commonly associated with dcSSc.[8]

Pulmonary: Up to 70% of patients with SSc will develop interstitial lung disease (pulmonary fibrosis) and/or pulmonary arterial hypertension (PAH).[11] The most common symptoms of interstitial lung disease are dyspnea and a nonproductive cough. PAH usually occurs in lcSSc and is typically a late complication. It may progress to right side ventricular enlargement (cor pulmonale) and eventually right-sided heart failure. Patients with SSc also have five-times greater risk of developing lung cancer than controls.

Renal: Sixty percent to 80% of patients with dcSSc will manifest some degree of kidney damage. Progression to kidney failure is uncommon,[11] however, a severe form of renal involvement called "scleroderma renal crisis" may occur in up to 15% of patients. It usually occurs in patients with early stage dcSSc and is characterized by acute oliguric renal failure, mild proteinuria, moderate to malignant hypertension, and thrombocytopenia.

Cardiac: Most cases of cardiac complications associated with SSc develop as a secondary consequence of PAH. Men suffer more severe involvement than women.[11] Examples of primary SSc cardiac disease include myocardial fibrosis, myocarditis, myocardial infarction, heart failure and conduction abnormalities. Myocardial fibrosis is patchy, and the pathogenesis appears to follow a pattern similar to that of RP described above.

Gastrointestinal: Approximately 90% of all patients with SSc have some degree of gastrointestinal involvement, although many patients exhibit no symptoms.[11] If symptoms are present, they commonly include dysphagia, hoarseness, heartburn, cough after swallowing, bloating, alternating constipation/diarrhea, and pseudo-obstruction. Gastroesophageal reflux is a common initial finding in SSc and likely results from underlying esophageal dysmotility.[8] Further complications include Barrett's esophagus and pulmonary microaspiration which may contribute to or aggravate interstitial lung disease.

Orofacial: Orofacial involvement is observed in about 80% SSc cases.[9] Commonly observed orofacial findings reflect both direct and indirect outcomes of the underlying vascular dysfunction and fibrosis associated with SSc. Features may include:[10,12-17]

1. "mask-like" or "mouse" facies;
2. widened palpebral fissures;
3. microstomia;
4. decreased interincisal distance;
5. oral telangiectasias;
6. thickened periodontal ligaments, and;
7. resorption of the mandibular angle;
8. oropharyngeal carcinoma.

Microstomia is defined as an interlabial distance of <45 mm or an interincisal distance of <40 mm.[15] The radiographic presence of periodontal ligament thickening is evident in up to two-thirds of patients with SSc.[17] Xerophthalmia and xerostomia are frequently noted in SSc; however, some believe these findings more correctly represent the concurrent presence of secondary Sjögren's syndrome and not direct manifestations of SSc per se.[12] The risk of the SSc patient developing oropharyngeal carcinoma is nine times greater than that of controls.[14]

Diagnosis

SSc should be suspected in any patient who presents with skin thickening, hand stiffness, puffy or swollen fingers and painful fingertip ulcers.[11] The most current American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) Classification Criteria for Systemic Sclerosis (SSc) is summarized in **Table 3**.[18] A total score of nine is necessary to establish the diagnosis.[18]

Table 2 - CREST Syndrome[9]

Calcinosis cutis	Calcific deposits; usually within the dermis in the extremities and bony prominences, but also in deeper periarticular tissues around or within the joints.
Raynaud phenomenon	Triphasic color changes of the extremities due to stress or cold temperature.
Esophageal dysmotility	Uncoordinated disorganized and ineffectual pattern of contractions resulting in low amplitude or no peristalsis.
Sclerodactyly	Fibrosis of the skin of the fingers or toes; often with subsequent atrophy and ulceration.
Telangiectasia	Widened small blood vessels of the skin.

Table 3 - ACR/EULAR Diagnostic Criteria for SSc[17]

Finding	Weight/Score
Skin thickening of the fingers of both hands extending proximal to the metacarpophalangeal joints	9
Skin thickening of the fingers	2
Fingertip lesions (with pitting)	2 (3)
Telangiectasia	2
Abnormal nailfold capillaries	2
Pulmonary arterial hypertension and/or interstitial lung disease	2
Raynaud's phenomenon	3
SSc-related autoantibodies (anti-centromere, anti-topoisomerase I [anti-Scl-70], anti-RNA polymerase III)	3

Autoantibodies specifically associated with SSc are anticentromere antibody (ACA), antitopoisomerase I (anti-Scl-70) antibody, and/or anti-RNA polymerase III antibody. These autoantibodies are almost always mutually exclusive. ACA is usually associated with lcSSc. Scl-70 antibodies are typically associated with dcSSc and a higher risk of severe interstitial lung disease. Antibodies to RNA polymerase III usually occur in patients with dcSSc, rapidly progressive disease, increased cancer risk, and an increased risk for scleroderma renal crisis.[11]

Medical Management and Prognosis

Due to the heterogeneous nature of SSc, medical therapy is tailored to the individual patient, considering the disease subset and extent of internal organ involvement.[19] Therapy is predominately pharmacologic and targeted to reduce tissue ischemia, inhibit the auto-inflammatory process, and inhibit excess collagen formation (**Table 4**).[9] It is initiated as early as possible to treat active phase disease in an attempt to reduce irreversible fibrotic outcomes. Therapies, such as non-steroidal anti-inflammatory drugs (NSAIDs) or acetaminophen (when NSAIDs are contraindicated) to manage arthralgia and a proton-pump inhibitor to manage reflux, may be prescribed. Patients with dcSSc and/or severe inflammatory organ involvement are typically managed with systemic immunosuppressive therapy.

Unfortunately, SSc remains a serious, potentially deadly, disease for which a cure remains elusive. A recent meta-analysis revealed a cumulative survival rate of 62.5% at 10 years after diagnosis and a standard mortality ratio of 2.72 times that of controls.[20] For patients with SSc, most deaths are attributed to pulmonary fibrosis, pulmonary arterial hypertension, or cardiac causes.[19] Other significant causes of death include renal disease, malignancy, gastrointestinal, and infectious causes.

Table 4 - Some Pharmacologic Agents Prescribed for SSc[19]

Indications	Agent	Possible Oral Complications
Arthralgia/arthritis	NSAIDs Acetaminophen Methotrexate Hydroxychloroquine	Erythema multiforme (EM) Oral ulcers, glossitis, EM EM, angioedema, mucosal discoloration
Calcinosis cutis	Minocycline Methotrexate Infliximab Rituximab	Glossitis, candidiasis, xerostomia Oral ulcers, glossitis, EM Candidiasis EM, angioedema
GER	Proton-pump inhibitors	EM, angioedema
Pruritus	Antihistamines	Xerostomia
Raynaud phenomena	Ca^{++} channel blockers	Gingival enlargement
Renal disease	ACE inhibitors	Angioedema, EM
Skin manifestations	Methotrexate Mycophenolate mofetil Cyclophosphamide	Oral ulcers, glossitis, EM Oral ulcers, candidiasis, xerostomia Stomatitis, mucositis
Severe disease	Abatacept Rituximab Tocilizumab	EM, angioedema EM, angioedema Oral ulcers, stomatitis

Dental Management Considerations for the Patient with SSc

The patient with SSc may present for dental care to address both routine and SSc-related concerns. As with all patients, the clinician's first goal is to establish good rapport and attain a thorough medical history. Information concerning the presence of comorbid illnesses or conditions, current medical therapies, and disease status must be determined to generate an individually tailored therapeutic treatment plan. Ultimately, the patient with SSc must understand that the oral and physical manifestations of their disease may profoundly compromise not only the therapeutic options available for dental care but also the outcomes of care.

Oral disease risk for patients with SSc is increased over that of healthy individuals for a variety reasons:

1. The common presence of hyposalivation with SSc increases the risk of developing oral infections (caries, oral candidiasis, and periodontitis).
2. Microstomia and or sclerodactyly, frequently observed in SSc, may compromise both the patient's ability to accomplish effective oral hygiene and the clinician's ability to adequately access the oral cavity and/or render care.
3. Medications prescribed to manage the myriad of SSc disease manifestations may adversely affect the oral cavity (oral dryness, mucositis, immunosuppression, gingival enlargement, etc.), further increasing the risk of oral disease.

The cumulative effect of these considerations present numerous potential challenges for the dental team when managing the patient with SSc. Universal preventive and maintenance measures that should be recommended as necessary for all patients with SSc are summarized in **Tables 5 and 6**. Some authorities recommend against the use of local anesthetics with a vasoconstrictor, on the premise that it may aggravate underlying vascular dysfunction.[9,21] Patients with early onset SSc and minimal oral involvement should be able to tolerate the delivery of routine dental care (simple restorations, conservative endodontics, prophylaxis). Patients with progressing or advanced oral involvement of SSc may present unique challenges for the dental practitioner.

Prospective treatment plans must account for not only the practitioner's ability to deliver the intended care, but also the progressive nature of the disease and the patient's ability to successfully accomplish oral hygiene.[12,13,16,21,22]

Summary

Systemic sclerosis is an uncommon and potentially fatal autoimmune disease. The astute oral healthcare professional may be the first to identify the observable characteristic findings of skin thickening, hand stiffness, puffy or swollen fingers and painful fingertip ulcers and initiate referral. The oral and systemic manifestations of the disease present unique challenges to the dental professional and patient both in terms of therapeutic options and outcomes.

Table 5 - Measures to Manage Hyposalivation and Xerostomia[21]

Measure	Examples	Comments
Topical stimulation of salivation	• Sugar-free gum • Sugar-free mints	• Xylitol containing products should be used
Moisturizers	• Sip water • Water mist • Saliva substitutes	• Formulations vary • Liberal use • Variable patient acceptance • Avoid products with pH < 6
Sialagogues	• Pilocarpine (Salagen®, generic)* (10 mg tid, titrated as needed) • Cevimeline (Evoxac™, generic)* (30 mg tid, titrated as needed)	• Common side effects include sweating, headache, nausea, gastrointestinal upset, urinary frequency, rhinitis, flushing • Allow 7 days between dosing changes to determine overall effect and tolerance. Allow up to 8 weeks to establish effect.

* Off-label indication

Table 6 - Patient Directions to Reduce Oral Infections

- Perform daily mouth opening exercises to improve / maintain oral opening range.[13,23-25]
- Perform thorough oral hygiene measures (brushing with a fluoridated toothpaste and flossing) after meals and at bedtime. If dexterity or oral access issues are present, use powered toothbrush and adaptive flosser.[16,21,23]
- Avoid drinking cariogenic liquids, eating sugar-containing mints or gums, and frequent between meal snacks that contain large amounts of sugar.
- Use a prescription topical fluoride gel daily as instructed by your dentist.
- Commit to regular and periodic follow-up dental examinations and cleanings on a schedule determined by your clinician, typically every three months.
- Report any difficulties in oral hygiene measures or mucosal irritations/sores to your provider promptly.

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(1.0 CE Credit Contact Hour) Please circle the correct answer. 70% equals passing grade.

1. **Approximately _____ of the general population experiences polyautoimmunity.**
   a. 3%
   b. 8%
   c. 10%
   d. 20%

2. **Autoantibodies specific for SSc include all of the following except one. Which is the exception?**
   a. Anticentromere antibody (ACA)
   b. Anti-RNA polymerase III antibody
   c. Rheumatoid factor antibody (RF)
   d. Antitopoisomerase I (anti-Scl-70)

3. **Women are _____times more likely to be afflicted with an autoimmune disease than men**
   a. 2
   b. 4
   c. 6
   d. 8

4. **RP is a painful condition that affects ___ of patients with SSc.**
   a. <50%
   b. 70%
   c. 90%
   d. >95%

5. **The lcSSc subset of SSc is characterized as:**
   a. having cutaneous involvement distal to the elbows.
   b. having cutaneous involvement affecting the trunk.
   c. being indicative of aggressive disease progression.
   d. a & c
   e. b & c
   .

6. **About 50% of patients with SSc manifest gastrointestinal involvement, but many patients do not exhibit symptoms.**
   a. The first part of the statement is true, but the second part of the statement is false.
   b. The first part of the statement is false, but the second part of the statement is true.
   c. Both parts of the statement are true.
   d. Both parts of the statement are false.

7. **Potential oral complications associated with SSc or its management include:**
   a. Stomatitis
   b. Dry mouth (xerostomia)
   c. Candidiasis
   d. Inadequate oral hygiene
   e. a, b, c
   f. a, b, c, d

8. **Microstomia is defined as an interincisal distance of less than _____.**
   a. 40mm
   b. 45mm
   c. 50mm

9. **The patient with lcSSc demonstrates skin involvement distal to the elbows and knees. Typical serology for lcSSc is positive for anti-RNA polymerase III antibodies.**
   a. The first statement is true, but the second statement is false.
   b. The first statement is false, but the second statement is true.
   c. Both statements are true.
   d. Both statements are false.

10. **The commonly observed dry mouth in the patient with SSc increases the risk of all of the following except**
    a. Caries
    b. Periodontal disease
    c. Herpes labialis
    d. Candidiasis

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