

Systemic lupus erythematosus with overlapping symptoms of Sjögren's syndrome and dermatomyositis

Natalia P. Bielecka-Kowalska¹, Szymon Roszczyk², Dominika Kostrzewa², Sebastian Kłosek^{1,3}

¹Department of Oral and Periodontal Diseases, Medical University of Lodz, Poland

²Student Scientific Circle at the Department of Oral Mucosal and Periodontal Diseases, Medical University of Lodz, Poland

³Department of Oral Pathology, Medical University of Lodz, Poland

Medical Studies

DOI: <https://doi.org/10.5114/ms/213571>

Key words: dermatomyositis, systemic lupus erythematosus, Sjögren's syndrome.

Abstract

Dermatomyositis is a rare autoimmune idiopathic inflammatory condition characterised by proximal muscle weakness and distinctive cutaneous manifestations such as heliotrope rash and Gottron's papules, often associated with systemic involvement and an increased risk of malignancy. Sjögren's syndrome is caused by accumulation of lymphocytes in the exocrine glands. The most common clinical manifestations include xerostomia and xerophthalmia, which occur as a result of malfunctioning salivary and lacrimal glands. Systemic lupus erythematosus is an autoimmune disease in which the body's immune system mistakenly attacks healthy tissues in many parts of the body. Typical symptoms include painful and swollen joints, fever, chest pain, hair loss, mouth ulcers, swollen lymph nodes, fatigue, and a red rash that most often appears on the face. The intensity of these symptoms may be different in individual patients. Periods of exacerbation and remission of the disease are common.

Introduction

Dermatomyositis (DM) is a rare autoimmune idiopathic inflammatory condition characterised by proximal muscle weakness and distinctive cutaneous manifestations such as heliotrope rash and Gottron's papules, often associated with systemic involvement and an increased risk of malignancy [1]. Sjögren's syndrome (SS) is caused by accumulation of lymphocytes in the exocrine glands. The most common clinical manifestations include xerostomia and xerophthalmia, which occur as a result of malfunctioning salivary and lacrimal glands [2]. Systemic lupus erythematosus (SLE) is an autoimmune disease in which the body's immune system mistakenly attacks healthy tissues in many parts of the body. Typical symptoms include painful and swollen joints, fever, chest pain, hair loss, mouth ulcers, swollen lymph nodes, fatigue, and a red rash that most often appears on the face [3–5]. The intensity of these symptoms may be different in individual patients. Periods of exacerbation and remission of the disease are common.

Case report

A 66-year-old woman presented to the Department of Periodontology, Medical University of Lodz, in May 2024 on account of aggravating oral mucosal lesions manifested by dry mouth, numerous small aphthae, reddened, dry soft palate with papillary hyperplasia, and swollen gingiva (Figures 1, 2). According to the patient's history, the first symptoms

occurred in 1998 in the form of erythematous and oedematous skin lesions on the cheeks, eyelids, and the arms. The lesions were poikilodermic in nature. There are no data on the treatment applied at that time. Over the years, other symptoms developed, and several attempts were made to establish a diagnosis. Finally, in 2005, the patient was diagnosed with DM without muscle involvement (Tables 1, 2) [5–8]. There are no data on the therapy applied at that time in the patient's medical history. In 2007, in differential diagnosis, DM was confirmed, and discoid lupus erythematosus (DLE) was excluded. The woman was hospitalised in the Dermatology Department. She was prescribed prednisolone at a dose of 40 mg/day. In 2008 only persistent skin lesions and low laboratory tests (absence of ANA antibodies, borderline dsDNA levels, creatine kinase test [CPK], and transaminase within normal limits, no transaminase within normal limits, no inflammatory lesions on skin biopsy) that do not allow the diagnosis of a specific disease entity. The diagnosis was confirmed, and chloroquine phosphate was added to the treatment regimen. Due to lack of response, the medication was discontinued.

Due to a suspicion of SS, fine-needle aspiration biopsy (FNAB) was performed in 2014. The microscopic image indicated chronic inflammation of the salivary glands. After a few months, the skin lesions aggravated. The patient also developed dysphagia, dry mouth and eyes, and joint pain. Tests showed no Ro or LA

Medical Studies



Figure 1. Changes in the tongue



Figure 2. Changes in the mucosa of the hard and soft palate

Table 1. Diagnosis of the diseases discussed [3, 4, 6, 7]

Disease	Dermatomyositis	SLE	Sjögren's syndrome
Diagnosis	• Muscle involvement, mainly of the shoulder and pelvic girdles (difficulty lifting arms and doing squats) • Erythematous-oedematous skin lesions, mainly on the face and the extremities • Gottron's sign • Histological examination of muscles (inflammatory and degenerative changes) • Electromyographic examination (primary muscle lesions) • Muscle enzyme testing (increased creatine kinase and aldolase)	The patient reports one of the following symptoms: • Regular eye dryness of at least 3-month duration • Recurrent sensation of the presence of sand or gravel in the eyes • Use of artificial tears at least three times a day • Regular feeling of dry mouth of at least 3-month duration • Consider the need to have a drink while eating dry food. • Salivary gland ultrasound nodular honeycomb-like structure • Biopsy of small salivary glands on the lips for histopathological evaluation • H&E staining, evaluation of the glands for the number of inflammatory foci containing a minimum of 50 lymphocytes per 4 mm² biopsy	• Lichen-like lesions with perivascular inflammation-characteristic but not pathognomonic • Presence of antibodies to native DNA or antibodies to Sm confirmed by serological tests • Meeting at least four American Rheumatism Association (ARA) criteria for initial diagnosis

antibodies. Methotrexate at a dose of 20 mg/week was discontinued after two weeks because of fungal lesions in the gastrointestinal tract.

In 2017, laboratory tests revealed low inflammatory parameters, slightly reduced C3 complement component, antinuclear antibodies present in a titre of 1 : 320 with homogeneous spot luminescence type, borderline anti-dsDNA antibodies in typing, which was not confirmed by quantitative testing. Additionally, laboratory tests showed the following: borderline immune complexes; absence of antibodies characteristic of polymyositis p and c; absence of ANCA (anti-neutrophil cytoplasmic antibodies), anticardiolipin,

lupus anticoagulant, against β_2 -microglobulin Ro and La; absence of rheumatoid factor and anti-cyclic citrullinated peptide (anti-CCP) antibodies;

cryoglobulin negative – no antibodies specific for *Borrelia* antigens. Phosphocreatine kinase was within normal limits at that time, indicating absence of muscle tissue destruction. X-ray of the hands revealed degenerative changes in some of the interphalangeal joints. Single geodes were identified in the stylo-mastoid processes of the elbows. Mild degenerative changes were also seen on knee X-ray.

During the same period, irregular exophytic lesions were observed intraorally on the upper pharyngeal

Table 2. EULAR/ACR criteria for the classification of systemic lupus erythematosus [5, 8]

Domainb	Weightc
Clinical domains	
Constitutional:	2
Fever (> 38°C)	
Hematologic:	
Leukopenia (< 4000/ μ l)	3
Thrombocytopenia (platelet count < 100,000/ μ l)	4
Autoimmune haemolysisd	4
Neuropsychiatric:	
Delirium	2
Psychosis	3
Seizure (generalized or partial/focal)	5
Mucocutaneous:	
Nonscarring alopeciae	2
Oral ulcerse	2
Subacute cutaneous lupus erythematosus or discoid lupus erythematosus	4
Acute cutaneous lupus	6
Serosal:	
Pleural effusion or pericardial effusion	5
Acute pericarditis	6
Musculoskeletal:	
Joint involvementf	6
Renal:	
Proteinuria (> 0.5 g/day)	4
Renal biopsy class II or V lupus nephritis	8
Renal biopsy class III or IV lupus nephritis	10
Immunologic domains	
Antiphospholipid antibodies:	
Anticardiolipin antibodies or Anti- β_2 glycoprotein 1 antibodies or Lupus anticoagulant	2
Complement proteins:	
Low C3 or low C4	3
Low C3 and low C4	4
SLE-specific antibodies:	
Anti-dsDNA antibody or Anti-Smith antibody	6

mucosa, from the base of the tongue to the supraglottic region. The patient was prescribed general and topical antifungals and intranasal steroids (nystatin for oral mucosa brushing, fluconazole 1 $\times$ 100 mg/14 days). Due to the patient's reported headaches and mild muscle weakness in the upper and lower limbs, a head MRI was performed. It showed hyperintense lesions in the white matter of both hemispheres on T2-TIRM images and T2-weighted iso-/hypointense lesions on T1-weighted images, the largest one in the parietal lobe on the left side, which may correspond to non-specific vascular foci, i.e. lesions occurring after a vascular episode. The muscle fatigue test was negative. The patient was consulted by a neurologist. The examination revealed a discrete myopathic syndrome manifested by quadriceps weakness with questionable myasthenic symptoms. Acetylsalicylic acid, 1 $\times$ 75 mg daily and further neurological diagnosis were recommended.

During the same hospitalisation in 2017, due to the significant feeling of dryness in the oral cavity and the previously identified chronic inflammation of the salivary glands, Schirmer's test and lissamine green staining were performed (RE 15 mm, LE 12 mm, RE 3 pts, LE 3 pts, respectively, with a normal value defined as > 10 mm of filter paper wetting within 5 minutes). Salivary gland ultrasound could suggest chronic inflammation. Differential diagnosis was required to exclude or confirm SS. A salivary gland biopsy could not be performed at that time due to increased fungal lesions in the oral cavity. In the histopathologist's opinion, previous samples collected for endoscopic ultrasound-guided fine-needle biopsy (EUS-FNB) did not allow for reliable diagnosis.

The diagnosis of undifferentiated systemic connective tissue disease was made, which required further histopathological evaluation for SS (Tables 1, 3). Treatment with hydroxychloroquine (200 mg/day) was initiated. During this period, the patient received the following pharmacotherapy: hydroxychloroquine 200 mg/day and levothyroxine sodium 1 $\times$ 75 μ g in the morning, sertraline 100 mg 1 $\times$ 1, mianserin hydrochloride 10 mg 1 $\times$ 1 in the evening, vitamin D₃ (1000 IU 2 $\times$ 1), calcium carbonate 1 $\times$ 1000 mg, nystatin for oral mucosa brushing, fluconazole 1 $\times$ 100 mg for 14 days, acetylsalicylic acid 1 $\times$ 75 mg.

The patient had a follow-up visit after 5 months. Significant improvement in the oral cavity was observed. However, the examination also showed eye reddening and increased lacrimation (conjunctivitis was suspected). Laboratory tests showed no blood count abnormalities, and inflammatory markers and vitamin D₃ levels were normal.

Follow-up examinations during this period revealed reduced C3 complement component (61.9 mg/dl), antinuclear antibodies present in a clinically insignificant titre of 1 : 160, and borderline immune complexes. The results of biopsy of the minor salivary glands, together with the characteristic histopatho-

Table 3. Classification criteria for primary Sjögren's syndrome (ACR/EULAR, 2016) [12]

Criterion	Scoring
Inflammatory foci with lymphocytic infiltration in the labial salivary gland and the number of foci ≥ 1 per 4 mm ² assessment according to Daniels et al. ^a	3
Presence of anti-SSA/Ro antibodies	3
Degree of conjunctival and corneal staining according to the method by Whitcher <i>et al.</i> ($\geq 5^b$ in at least one eye) or by van Bijsterveld ($\geq 4^{c,d,e}$ in at least one eye)	1
Schirmer test ≤ 5 mm after five minutes in at least one eye ^d	1
Unstimulated salivary flow assessed by the Navazesh and Kumar method ≤ 0.1 ml/min ^e	1
Exclusion conditions: prior head and neck irradiation, active HCV infection (confirmed by polymerase chain reaction), AIDS, sarcoidosis, amyloidosis, graft-versus-host disease, IgG4-related disease.	
Interpretation: A total score of ≥ 4 supports the diagnosis of primary Sjögren's syndrome.	
Criteria sensitivity 96%, specificity 95%.	

^aArthritis Rheum. 2011; 63: 2021-2030, ^bAm J Ophthalmol. 2010; 149: 405-415, ^cArch Ophthalmol. 1969; 82: 10-14, ^dIn patients taking anticholinergic drugs, the test may be performed after a break from the treatment. ^eJ Am Dent Assoc. 2008; 139 Suppl.: 35S-40S. Based on: Arthritis Rheum. doi: 10.1002/art.39859.

logical changes and a positive Schirmer test, fulfilled the diagnostic criteria for SS. The salivary glands and the surrounding lymph nodes were re-evaluated by ultrasound. The salivary glands were heterogeneous with increased perfusion, and large multiple lymph nodes with cortical hyperplasia were present. Based on the overall clinical picture and the results of the additional investigations, primary SS was diagnosed. Treatment with hydroxychloroquine, 200 mg/day, was continued. The pharmacological treatment prescribed on the previous visit was maintained, with the exception of antifungals and addition of ofloxacin 4×/day for 5 days, sodium hyaluronate, sodium chloride, actinoin, trometamol, hydrochloric acid 1.5 mg/ml 6×/day continuously, dexpanthenol, and polyacrylic acid 3×/day for 7 days.

Due to a period of symptom remission, the patient had not been assessed at a specialist clinic for seven years. However, a sudden exacerbation of oral lesions prompted the patient to visit the Department of Periodontology in May 2024.

At the time of the examination, multiple, diffuse small aphthous, exophytic, and scar-like lesions were present on the mucosa of the soft palate, descending to the posterior pharyngeal wall.

At the first visit, the sharp edges of the teeth were softened. The following recommendations were given: a sodium lauryl sulphate-free toothpaste, compound preparation (ingredients: aqua, glycerine, *Linum usitatissimum* seed extract, *Salvia officinalis* extract, *Chamomilla recutita* flower extract, *Arnica montana* flower extract, *Rosmarinus officinalis* leaf extract, panthenol, PEG-40 hydrogenated castor oil, aroma, xylitol, allantoin, chlorhexidine digluconate, sodium saccharin), moisturising spray (oleum hippophaes, oleum calendulae, *Oleum Bergamii* S. 5×/day, prescription ointment with nystatin, neomycin, anaesthesin, and hydrocortisone,

glycerine S.) 4×/day. A fungal culture test was also ordered. At the follow-up visit after 1 week, the aphthous-like lesions were found to have healed, and the patient's condition improved significantly. Another follow-up visit in 2 months was recommended.

The test results showed no deficiency of ferritin, folic acid, vitamin B or D₃, elevated levels of ESR (30 mm/1 h) and CRP (13.4 mg/l). Further diagnosis by primary care physician (PCP) was ordered, showing nystatin-sensitive *Cryptococcus* in culture (the patient applied nystatin brushing as recommended by PCP). Sample collection was deferred, and the patient was prescribed moisturising and soothing mouth rinse (colostrum, PVP-VA, ozonated oil, tea tree oil, and stevia) 3×/day and nystatin topically 4×/day.

The next diagnostic step was histopathological examination. A biopsy taken from the soft palate revealed the following: "A fragment of mucosa covered by squamous epithelium with features of parakeratosis, without signs of dysplasia. Features of basal layer damage. Intraepithelial inflammatory infiltrates of neutrophils visible. An intense chronic inflammatory infiltrate present in the subepithelial area, partially damaging the lower epithelial layers and concentrating around blood vessels. Features of fibrosis. The microscopic image may correspond to lupus erythematosus lesions". The patient also reported remission of symptoms during this period. Continuation of the previously prescribed pharmacotherapy and dermatological consultation were recommended.

Discussion

DM is a variant of polymyositis. The condition is characterised by lesions that particularly affect the muscles of the shoulder and pelvic girdles, as well as erythematous and oedematous skin lesions located mainly on the face and the extremities. Other signs

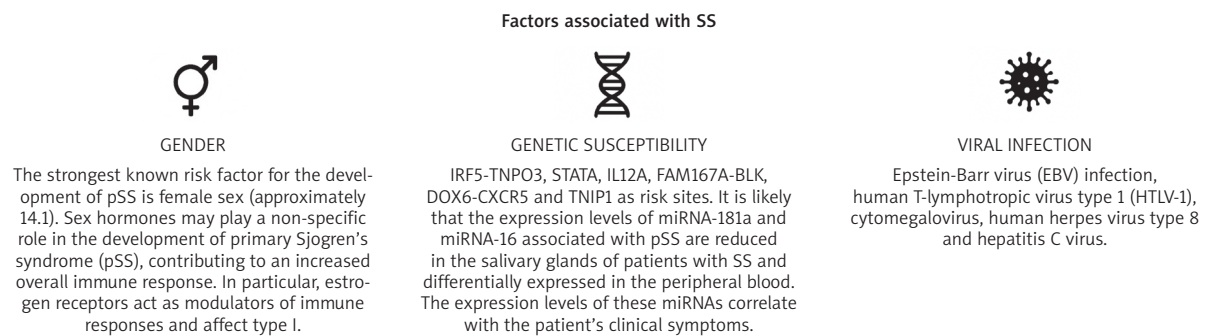


Figure 3. Factors associated with SS [13–15]

include erythema and telangiectasias on the back of the hands and uneven thickening of the skin, the so-called Gottron's papules (Gottron's sign), over small joints. In the chronic variety, scleroderma-like or scabby skin lesions are present. Also, calcium deposits in subcutaneous and muscular tissue sometimes occur, and organ changes mainly involve the smooth muscles of the gastrointestinal tract, respiratory tract, and the myocardium [1, 9].

Pharmacological strategies in DM differ depending on the severity of symptoms. Systemic corticosteroids are considered the gold standard in the treatment of myopathy associated with DM. Therefore, oral immunosuppressants or biologic therapy are commonly prescribed in the early stages of the disease, especially in patients with myopathy or other systemic symptoms. The recommended pharmacotherapy is prednisone 60–100 mg in the initial period and subsequently in maintenance doses [10]. The regimen often includes immunosuppressive drugs, e.g. cyclophosphamide 100–150 mg daily, or azathioprine 100–200 mg daily [7]. Antimalarial drugs are also used in the treatment of cutaneous forms of DM, with hydroxychloroquine considered as the first-line therapy and the most frequently prescribed medication. However, recent reports suggest that the use of hydroxychloroquine may be associated with an increased risk of exacerbation of skin lesions in some patients [10, 11]. Rituximab and bisphosphonates have been increasingly applied in the treatment of DM, but there is still a lack of clinical trials to clearly assess their efficacy. Some clinicians also use other medications, such as mycophenolate mofetil (MMF), methotrexate (MTX), or intravenous immunoglobulin (IVIG), but calcineurin inhibitors (usually cyclosporine and less frequently tacrolimus) are more common, despite a higher risk of serious side effects [10, 11].

SS is a chronic autoimmune connective tissue disease affecting the exocrine glands. In recent years, it has been increasingly suggested that the aetiology of this condition is multifactorial, with factors such as endocrine disruption, genetics, or infection playing a potential role (Table 3, Figure 3) [12–15].

In most cases, primary SS manifests as xerophthalmia, which is characterised by dryness of the cornea and conjunctiva, a gritty sensation in the eyes, and sensitivity to light or cigarette smoke. Other symptoms include dry mouth, a burning sensation in the mucous membranes, difficulty eating and swallowing when wearing dentures, and taste disorders or even ageusia. In the advanced stage, the oral mucosa becomes dry and reddened. It is covered with plaque consisting of food debris, exfoliated epithelial cells, and bacteria. Dry and cracked lips may be accompanied by angular cheilitis. The tongue is often smooth and coated, and some patients experience enlargement of the parotid and submandibular salivary glands. Systemic symptoms include a subfebrile state, muscle and joint pain, fatigue, and the Raynaud phenomenon, i.e. paroxysmal blanching of distal parts of the extremities (and sometimes the nose and ears) [13, 14].

There is no effective general treatment available. Dentists should focus preventive measures on limiting the development of caries, preventing periodontitis, ringworm, and damage to the oral mucosa. Saliva and tear substitutes are recommended in the treatment of xerostomia and xerophthalmia, while sugarless sweets, chewing gum, and saliva stimulants (such as pilocarpine and cevimeline) may help stimulate saliva secretion. The study shows that the two drugs are equally effective; however, pilocarpine causes more reported side effects [15–17].

SLE is a complex autoimmune disease characterised by dysregulation of T and B lymphocytes. This leads to changes in various organs, such as the skin, kidneys, heart, blood vessels, and joints, as well as the nervous system. In the early stages, symptoms are non-specific, including fever, general malaise, joint pain, weight loss, and fatigue. Characteristic skin lesions appear as erythematous foci arranged in a butterfly pattern across the zygomatic region and the nasal dorsum (that does not cross the nasolabial folds). Renal lesions can lead to organ failure. Cardiac complications are also common. In the oral cavity, the most common manifestations are hyperkeratosis, discoloration, and ulceration around the hard and

soft palates. Xerostomia and secondary SS are extremely significant symptoms. Less common abnormalities are glossodynia, a fissured tongue, cheilitis, and ulcers on the lips and cheeks [4, 5, 18].

Glucocorticosteroids, immunosuppressants, and cytotoxic drugs are used in the treatment of severe SLE. The choice of drug depends on the clinical phenotype and disease activity, with specific indications including lupus nephritis, neuropsychiatric manifestations, and systemic vasculitis. Due to their potent immunomodulatory effect, glucocorticosteroids remain the mainstay of SLE therapy. They are the traditional treatment option ensuring rapid action and quick control of the disease activity. However, a prolonged therapy with high doses is associated with a higher risk of complications; therefore, the effective dose should be minimised. The dosage is adjusted based on severity of symptoms. In mild cases, doses of around 5–10 mg of prednisone per day are usually effective. More severe situations may require higher doses of 0.5–1 mg/kg body weight (converted to prednisone), with the option of an intravenous bolus of methylprednisolone, particularly in cases of lupus nephritis, severe haematological disorders, or involvement of the central nervous system. The findings of observational and clinical studies have demonstrated that low-dose methylprednisolone pulses (125–500 mg daily) constitute an efficacious treatment for severe acute exacerbations of lupus, with a paucity of adverse effects [5, 18–21]. Antimalarial drugs, primarily hydroxychloroquine, are recommended for all SLE patients due to their immunomodulatory effects and beneficial impact on disease progression. They also reduce the risk of thrombotic complications, improve metabolic parameters, and may be applied safely even in pregnancy. Non-steroidal anti-inflammatory drugs (NSAIDs) can be used in the treatment of mild forms of the disease, particularly joint pain and serous inflammation. Selective COX-2 inhibitors are preferred due to their better safety profile. Methotrexate mainly acts by increasing levels of the anti-inflammatory adenosine and is used in the treatment of joint and skin lesions. Azathioprine, a purine analogue, inhibits nucleic acid synthesis and has been shown to be effective in treating joint, skin, and serous membrane lesions. Cyclophosphamide, an alkylating agent, is used in the treatment of severe and life-threatening forms of SLE, particularly lupus nephritis (LN) and neuropsychiatric complications. Mycophenolate mofetil prevents rejection of transplanted organs by inhibition of inosine monophosphate dehydrogenase. It is used in the treatment of lupus nephritis and other symptoms of lupus not associated with the kidneys. It is more effective in preventing relapses than azathioprine. Sirolimus, an inhibitor of the mTOR pathway with a favourable safety profile, shows therapeutic potential by increasing the number of regulatory T cells

(Tregs) and reducing the production of pro-inflammatory cytokines. Recently, there have been studies conducted on special medicines, such as monoclonal antibodies and inhibitors of signalling pathways (e.g. belimumab and anifrolumab). They may be the future of SLE treatment because they can be adjusted to individual needs, thus reducing the side effects. The patients commonly have vitamin D deficiencies, which correlate with higher disease activity, more severe fatigue, and increased risk of thrombosis. Because vitamin D supplementation is well-tolerated, the recommended target level for 25(OH) vitamin D is 40 ng/ml – higher concentrations have not been shown to provide any additional therapeutic benefits. Dysbiosis of the intestine microbiome probably plays a role in the onset and activity of the disease; however, conclusive studies are still lacking [5, 19–21].

SS is a chronic autoimmune disease that can coexist with other connective tissue disorders, including SLE.

Such co-occurrence is not uncommon, with estimates suggesting that it may affect up to several percent of patients with SLE. Some experts argue that these patients represent a distinct subgroup characterised by specific clinical and serological features. Increasingly, it is being emphasised that the coexistence of multiple autoimmune disorders poses a growing clinical challenge, which may have significant implications for future diagnostics and therapeutic strategies [22].

Summary

SLE and primary Sjögren's syndrome (pSS) are often comorbidities. In their course, oral symptoms such as xerostomia, salivary gland inflammation, ulceration, and mucosal lesions are often observed. Xerostomia results from infiltration of the salivary glands by lymphocytes and their dysfunction. As a result of reduced saliva production, patients are at greater risk of developing dental caries, periodontal disease, and fungal infections.

Distinguishing SLE from pSS can be difficult due to overlapping clinical and serological signs. Laboratory tests, including antinuclear antibodies (ANA), anti-dsDNA, and anti-SSA/SSB, are crucial. The presence of anti-dsDNA antibodies is more specific for SLE, while anti-SSA/SSB are characteristic of pSS. In addition, salivary gland biopsy can confirm the diagnosis of pSS.

Treatment of patients with SLE and pSS requires multidisciplinary teams of rheumatologists, dermatologists, and dentists. Immunosuppressive treatment, including the use of hydroxychloroquine, is the mainstay of SLE treatment and may also be beneficial in pSS.

Identifying those with overlapping syndromes is important because such patients may require different monitoring and treatment regimens.

The overlap of further autoimmune diseases, the symptoms of which may affect the same areas, may pose further clinical challenges.

Further research is needed to explore optimal therapeutic strategies and emphasise the importance of an interdisciplinary approach in the care of these patients.

In the analysed case, the collaboration of specialists in periodontology, rheumatology, and dermatology, as well as comprehensive treatment, led to a significant improvement in the patient's quality of life. Optimal patient care requires close, long-term monitoring of the patient's health, with particular attention to the risk of fungal infections. The incidence of SSA and SSB antibodies is similar in patients with primary SS and in patients with SS coexisting with SLE. In patients with SLE alone, however, the incidence is much lower.

In autoimmune diseases such as SLE, SS, and DM, oral manifestations represent a frequent and clinically significant component of the disease course. They most commonly include xerostomia, ulcerations, aphthous lesions, and recurrent fungal infections, all of which substantially impair patients' quality of life. Early recognition and an interdisciplinary therapeutic approach are crucial to limiting disease progression and improving patient well-being.

Funding

No external funding.

Ethical approval

Not applicable.

Conflict of interest

The authors declare no conflict of interest.

References

- Didona D, Solimani F, Caposiena Caro RD, Sequeira Santos AM, Hinterseher J, Kussini J, Cunha T, Hertl M, Dido-na B. Dermatomyositis: a comprehensive review of clinical manifestations, serological features, and therapeutic approaches. *Ital J Dermatol Venereol*. 2023; 158(2): 84-98.
- Negrini S, Emmi G, Greco M, Borro M, Sardanelli F, Mura-daca G, Indiveri F, Puppo F. Sjögren's syndrome: a system-ic autoimmune disease. *Clin Exp Med*. 2022; 22(1): 9-25.
- Fortuna G, Brennan MT. Systemic lupus erythematosus: epidemiology, pathophysiology, manifestations, and man-agement. *Dent Clin North Am*. 2013; 57(4): 631-655.
- Siegel CH, Sammaritano LR. Systemic lupus erythemato-sus: a review. *JAMA*. 2024; 331(17): 1480-1491.
- García-Ríos P, Pecci-Lloret MP, Oñate-Sánchez RE. Oral manifestations of systemic lupus erythematosus: a sys-tematic review. *Int J Environ Res Public Health*. 2022; 19(19): 11910.
- Górska R. Choroby błony śluzowej jamy ustnej. Podręcz-nik dla studentów i do LDEK. Edra Urban & Partner, War-szawa 2023.
- Jabłońska S, Chorzelski T. Choroby skóry dla studentów medycyny i lekarzy. Wydawnictwo Lekarskie PZWL, Warszawa 1994.
- Aringer M, Costenbader K, Daikh D, Brinks R, Mosca M, Ramsey-Goldman R, Smolen JS, Wofsy D, Boumpas DT, Kamen DL, Jayne D, Cervera R, Costedoat-Chalumeau N, Diamond B, Gladman DD, Hahn B, Hiepe F, Jacobsen S, Khanna D, Lerström K, Massarotti E, McCune J, Ruiz-Irasto-rza G, Sanchez-Guerrero J, Schneider M, Urowitz M, Bert-sias G, Hoyer BF, Leuchten N, Tani C, Tedeschi SK, Tou-ma Z, Schmajuk G, Anic B, Assan F, Chan TM, Clarke AE, Crow MK, Czirják L, Doria A, Graninger W, Halda-Kiss B, Hasni S, Izmirly PM, Jung M, Kumánovics G, Mariette X, Padjen I, Pego-Reigosa JM, Romero-Diaz J, Rúa-Figueroa Fernández I, Seror R, Stummvoll GH, Tanaka Y, Tektoni-dou MG, Vasconcelos C, Vital EM, Wallace DJ, Yavuz S, Meroni PL, Fritzler MJ, Naden R, Dörner T, Johnson SR. 2019 European League Against Rheumatism/American College of Rheumatology Classification Criteria for Sys-temic Lupus Erythematosus. *Arthritis Rheumatol*. 2019; 71(9): 1400-1412.
- Yu K, Wang T, An D, Li X, Tang Z. Investigating the di-sparities among drug categories in drug-induced derma-tomyositis: a systematic review. *Semin Arthritis Rheum*. 2024; 67: 152478.
- Waldman R, DeWane ME, Lu J. Dermatomyositis: dia-gnosis and treatment. *J Am Acad Dermatol*. 2020; 82(2): 283-296.
- Traineau H, Aggarwal R, Monfort JB, Senet P, Oddis CV, Chizzolini C, Barbaud A, Francès C, Arnaud L, Chasset F. Treatment of calcinosis cutis in systemic sclerosis and dermatomyositis: a review of the literature. *J Am Acad Dermatol*. 2020; 82(2): 317-325.
- Shiboski CH, Shiboski SC, Seror R, Criswell LA, Labeto-ulle M, Lietman TM, Rasmussen A, Scofield H, Vitali C, Bowman SJ, Mariette X; International Sjögren's Syndrome Criteria Working Group. 2016 ACR-EULAR classification criteria for primary Sjögren's syndrome: a consensus and data-driven methodology involving three international patient cohorts. *Arthritis Rheumatol*. 2016; 69(1): 35-45.
- André F, Böckle BC. Sjögren's syndrome. *J Dtsch Derma-tol Ges*. 2022; 20(7): 980-1002.
- Tian Y, Yang H, Liu N, Li Y, Chen J. Advances in pathogenesis of Sjögren's syndrome. *J Immunol Res*. 2021; 2021: 5928232.
- Thorlacius GE, Björk A, Wahren-Herlenius M. Genetics and epigenetics of primary Sjögren syndrome: impli-cations for future therapies. *Nat Rev Rheumatol*. 2023; 19(5): 288-306.
- Noaiseh G, Baker JF, Vivino FB. Comparison of the di-scontinuation rates and side-effect profiles of pilocarpine and cevimeline for xerostomia in primary Sjögren's syn-drome. *Clin Exp Rheumatol*. 2014; 32(4): 575-577.
- Farag AM, Holliday C, Cimmino J, Roomian T, Papas A. Comparing the effectiveness and adverse effects of pilo-carpine and cevimeline in patients with hyposalivation. *Oral Dis*. 2019; 25(8): 1937-1944.
- Accapezzato D, Caccavale R, Paroli MP, Gioia C, Nguyen BL, Spadea L, Paroli M. Advances in the pathogenesis and treatment of systemic lupus erythematosus. *Int J Mol Sci*. 2023; 24(7): 6578.
- Su X, Yu H, Lei Q, Chen X, Tong Y, Zhang Z, Yang W, Guo Y, Lin L. Systemic lupus erythematosus: pathogene-sis and targeted therapy. *Mol Biomed*. 2024; 5(1): 54.

20. Lazar S, Kahlenberg JM. Systemic lupus erythematosus: new diagnostic and therapeutic approaches. *Annu Rev Med.* 2023; 74: 339-352.
21. Pawlak-Buś K, Schmidt W, Dudziec E, Leszczyński P. Current treatment of systemic lupus erythematosus: a clinician's perspective. *Rheumatol Int.* 2023; 43(8): 1395-1407.
22. Santos CS, Morales CM, Álvarez Castro C, Alvarez ED. Polyautoimmunity in systemic lupus erythematosus: secondary Sjogren syndrome. *Z Rheumatol.* 2023; 82 (Suppl 1): 68-73.

Address for correspondence

Natalia P. Bielecka-Kowalska
Medical University of
Lodz, Poland
E-mail: natalia.bielecka-kowalska@umed.lodz.pl

Received: 15.07.2025

Accepted: 24.10.2025

Online publication: 23.10.2026