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Navigating Oral Health in Autoimmunity: A Case of Systemic Lupus Erythematosus with Oral Ulcerations

[1]Dr. Nabanita Chatterjee, MDS PGT, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata

[2]Dr Swagata Gayen, Associate Professor, Guru Nanak Institute of Dental Sciences and Research Kolkata; Ph. D Scholar, Department of Oral and Dental Sciences, JIS University, Kolkata

[3]Prof (Dr) Rudra Prasad Chatterjee, Professor, Guru Nanak Institute of Dental Sciences and Research Kolkata; Ph. D Scholar, Department of Oral and Dental Sciences, JIS University, Kolkata

[4]Prof (Dr) Sk. Abdul Mahmud, Professor, Guru Nanak Institute of Dental Sciences and Research, Kolkata

[5]Dr. Arundhati Bhattacharyya, MDS PGT, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata

Corresponding Author: Dr. Nabanita Chatterjee, MDS PGT, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata

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Abstract

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease which can affect multiple organs and tissues throughout the body. SLE generally has an involvement of oral cavity and quality of life is impacted due to the disease process. A 50-year-old female presented with a complaint of painful oral lesions. Clinical examination revealed multiple ulcerative lesions on the buccal mucosa and the hard palate. The patient also reported a recent flare-up of rashes on sun exposed area as well as joint pain, fever and fatigue. Incisional biopsy was done. Histopathological analysis confirmed the presence of characteristic features of auto-immune disorder, including degeneration of the basement membrane and lymphocytic infiltration. A diagnosis of SLE was made. The oral lesions were conservatively managed which led to significant improvement. The case highlights the importance of recognizing oral manifestations of SLE for timely and effective management.

Keywords: SLE, Oral Lesions in SLE, EULAR/ACR

Introduction

The Latin word "lupus," which means "wolf," was first used in 1817. Prior to the middle of the 19th century, all illnesses of different origins that were marked by ulceration or necrosis were referred to as lupus since the name conjured up the idea of "tearing apart" or "pulling or stripping off." Biett and Kaposi initially presented it in 1828 and 1872,[1] respectively. Discoid Lupus Erythematosus (DLE) and Systemic Lupus Erythematosus (SLE) are the two types of LE that can affect people of any age, with the mean being between 21

and 50. A chronic, multisystem inflammatory disease with an autoimmune etiology, systemic lupus erythematosus primarily affects young women.[2]

Raynaud's syndrome, arthralgias and arthritis, pericarditis or pleuritis, involvement of the central nervous system or kidneys, autoimmune cytopenia, and malar and other rashes are common symptoms. Clinical and serologic criteria are necessary for diagnosis. Corticosteroids and immunosuppressants are necessary for the treatment of severe, persistent, aggressive illnesses.

Because of the vast range of appearances of oral mucosal lesions in LE patients, clinicians may find it difficult to distinguish this lesion from other oral disorders including Erythema Multiforme (EM) and Oral Lichen Planus (OLP), which result in delay in diagnosis and treatment. LE patients may have higher survival chances if they receive therapy early.

Here, we describe a case involving early SLE detection using clinical. hematological, histological, and immunological findings.

Case Report

A 50-year-old woman arrived in our OPD with the complain of psoriasiform lesions and facial edema that had been present for the last 3 to 4 weeks, along with ulcerations in her mouth with fever.

Gross clinical examination revealed superficial ulcers on the skin of the back, wrists, and feet. A history of photosensitivity was also provided by the patient. and rashes on sun-exposed areas involving the whole face, particularly in the malar region and the bridge of the nose giving the specific butterfly rash appearance was noted. She had also multiple large, irregular, dome-shaped swellings on her left leg that reached her hips.

Multiple erythematous erosive ulcerated lesions on the soft palate, retromolar region, and bilateral buccal mucosa were found during the intraoral examination. These lesions were painful in character.

The patient was given a provisional diagnosis of lupus erythematosus with a differential diagnosis of erosive lichen planus based on the clinical manifestations along with negative Nikolsky's sign to exclude another vulnerable vesicullo-bullous lesion i.e., pemphigus vulgaris.

The patient was asked to do routine hemogram along with ANA profile and the results are given in Table 1. An incisional biopsy was performed from a representative site on the right buccal mucosa on the retro-molar region under local anesthesia and two bits of tissue were taken. One bit was submitted for routine histopathological examinations in buffered formalin. The other bit was stored in Michel's solution for immunofluorescence studies.

H and E-stained sections reveal the presence of parakeratotic stratified squamous epithelium having flattened and irregular rete ridges. Epithelium revealed basal cell degeneration at places (FIG 2A, 2B). Stroma is characterized by perivascular intense chronic inflammatory cell infiltrate (FIG 2C). No cellular atypia was noted. The overall histopathological features were suggestive of "Auto-Immune Disease Favoring Lupus Erythromatosus".

According to EULAR/ACR classification criteria for systemic lupus erythematosus in children and adults by clinical rheumatology (2019) [3] diagnosis of "Systemic Lupus Erythromatosus" was given. (Table 2)

The patient was further referred to the School of Medical Science Sagar Dutta for consultation with the dermatology and rheumatology departments. Following a color doppler test, the rheumatology department

diagnosed the patient with extensive varicosity over the left leg.

Medical treatments comprised immunosuppressive treatment, antimalarial as immunosuppressive drug, corticosteroid, and sunscreen with the main ingredient of zinc oxide application twice during the day. Using antibacterial gels and multivitamin therapy, dental treatment was intended to alleviate the symptoms of cheilitis and palatal and buccal ulcers. Benzydamine Hydrochloride (0.15% w/v) mouthwash and 0.1% triamcinolone acetonide buccal paste were used to treat the clinical signs of oral ulcers, which caused disappearance of the oral ulcers by the fourth week.

ANA – 1.4 AU/ml (POSITIVE) (TABLE 1)

Additive Criteria Points Were Calculated: 17 (>10) (SLE) (as shown in TABLE 3)

The values listed in TABLE 1 points us to the direction that the patient is suffering from an auto- immune disease and keeping TABLE 3 in mind along with histopathological, direct immunofluorescence test it was established that the case is of Systemic Lupus Erythromatosus.

Discussion

Lupus erythematosus (LE) is a relatively uncommon disease that can affect almost any part of the body, at any time. Traditionally, LE has been subdivided into systemic and localized types, the latter being confined to the skin and/or mucous membrane, whereas the former is a syndrome characterized by a widespread involvement of various organ systems.[4] In the present case the patient was presented with cutaneous, musculoskeletal, oral lesions indicating multi-organ involvement.

This disease has its peak age of onset at about 30 years in females but about 40 years in males. The disease may occur in childhood as reported by Jacobs. Prevalence rates are higher in females than in males. A female-to-male ratio of approximately 2:1 occurs before puberty, and a ratio of 4:1 occurs after puberty.[5] In our case the patient was a 50-year-old female.

The course of Lupus disease is one of exacerbation and relative quiescence. The skin is affected in about three fourths of patients, in the form of butterfly rash, photosensitivity rash, mucous membrane lesions, and alopecia. SLE produces oral lesions in approximately 20% of cases. Symptomatic patients may present early to dentists, oral surgeons or physicians, whereas asymptomatic patients may not present until much later when they develop cutaneous or systemic manifestations. The oral lesions are caused by vasculitis and appear as frank ulceration or mucosal inflammation. The lip lesions often have a central atrophic and occasionally ulcerated area with small white dots, surrounded by a keratinized border composed of small radiating white striae. The intra oral mucosal lesions of LE most frequently affect the buccal mucosa or the palate They are composed of a central depressed red atrophic area surrounded by a 2 to 4 mm elevated keratotic zone that dissolves into small white lines in the buccal or labial mucosa.[6] In our case too the patient was presented to our department because of painful oral ulcerations. On examination extraoral malar rash was appreciable, and intraorally multiple erythematous erosive ulcerated lesions on the soft palate, retromolar region and bilateral buccal mucosa which were tender in nature was noted.

The diagnosis of DLE is usually made clinically. For confirmation of diagnosis, serological and histopathological analysis with immunofluorescence is done. Histologically SLE is characterized by hyperkeratosis with keratotic plugging, atrophy of the rete pegs, liquefaction, degenerative features of the basal cell layer and are usually very prominent in the epithelium. In connective tissue stroma diffused

lymphocytic infiltration in superficial and deep part, perivascular infiltration of lymphocytes as well as degeneration of collagen and elastic fibers, with hyalinization, edema and fibrinoid change is noted.

Direct immunofluorescent testing is often used to confirm a suspected diagnosis of lupus erythematosus. It is basically a test used to detect the presence of immunoglobulins at the epidermal-dermal junction or basement membrane zone of skin or oral mucosa of patients with the disease by incubating a biopsy specimen with a fluorescein-conjugated antiglobulin. These immunoglobulins were present at this specific histologic location in oral lesions in all patients with the systemic form. In patients with SLE, DIF of oral lesions typically reveals:

1. Linear Band: Deposition of immunoglobulins (predominantly IgG) and complement components along the basement membrane zone.
2. Lupus Band Test: This characteristic finding, known as the "lupus band test," is indicative of SLE and helps differentiate it from other conditions with similar clinical presentation.[7]

In the present case the DIF was positive with granular deposition of IgG, IgM and IgA at the epidermal-dermal junction.

The antinuclear antibodies (ANA) test is the serological hallmark of SLE. Up to 98% of patients with SLE will have a positive ANA, making it highly sensitive and useful as a screening test. A negative ANA makes SLE very unlikely and other diagnoses should be sought to explain symptoms. Antibodies to double-stranded DNA (dsDNA) are specific for SLE. In some patients, an increase in anti-dsDNA titer may signify onset of disease flare. Other autoantibodies, available on the extractable nuclear antigen-testing panel, can also be associated with SLE or other connective tissue diseases. Antibodies to Sm (anti-Smith), for example, are highly specific autoantibodies in SLE. Tissue deposition of immune complexes can fix complement in the classical pathway and therefore results in a reduction of serum complement levels. C3 and C4 can be measured readily.[8] In our case the ANA was positive and the C4 was low.

Systemic lupus erythematosus is a chronic inflammatory condition driven by a dysfunctional immune system. Sometimes patients can report known triggers, such as environmental factors like sunlight, drugs or hormonal exposure. Avoidance of these triggers would be sensible in preventing flares. The overall aim of therapy is to control disease activity. Mild activity can be managed with non-steroidal anti-inflammatory drugs (NSAIDs) or low-dose steroids, but more severe manifestations require prompt treatment with moderate-to-high doses of steroids to minimize organ damage. Steroid-sparing immunosuppressive medications should be considered early to prevent steroid-related morbidities. Hydroxychloroquine is an effective treatment in SLE, especially for arthritis and rash. Furthermore, it has a protective effect in reducing damage accrual in the long term and confers a survival benefit in SLE patients.[10] Hydroxychloroquine is well tolerated and, when dosed appropriately, ocular toxicity is very rare. A range of immunosuppressive medications has been used as a steroid-sparing agent in SLE, such as cyclophosphamide and mycophenolate for lupus nephritis, although azathioprine and methotrexate are used commonly. Belimumab, which is a human monoclonal antibody that inhibits the activation of B-cells by interfering with a protein necessary for B-cell activity, are also being used nowadays. Other general measures that should be considered in SLE patients include cardiovascular risk reduction and optimization of bone protection. Patients with SLE are at significantly increased risk of premature

atherosclerosis, so smoking cessation and control of hypertension, dyslipidemia, obesity and hyperglycemia are strongly recommended. Strategies to prevent osteoporosis should be considered in most patients because many are likely to require long-term glucocorticoid therapies.[9] In our case the patient was treated by corticosteroid buccal paste.

Specialist referrals to a rheumatologist are important to establish the diagnosis, to gauge disease activity and severity, and to guide disease management. By better understanding the disease process, its oral manifestations and management, general dentists can help patients comprehend the complexity of disease pathogenesis and priorities in treatment. Dental practitioners working with treating specialists play a key part in the monitoring and management of the disease and associated co-morbidities. Furthermore, offering the patients ongoing support and counselling also plays a key role in management.

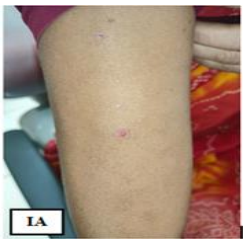

Figure IA: Multiple ulcerations on the forearm,

Figure IB: Ulcerations on the collar region

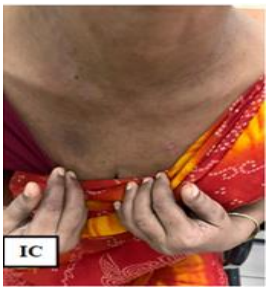

Figure IC: Gross varicosity on the left leg

Figure IIA: Extra oral picture showing prominent rash extending from the left malar region to the bridge of the nose upto the right malar region,

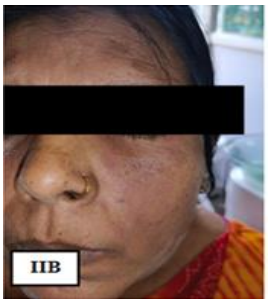

Fig IIB: Photograph of the left side showing the malar rash

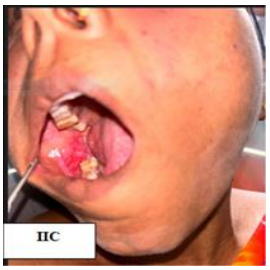

Figure IIC: Intra oral picture showing erythematous ulceration on the left buccal mucosa

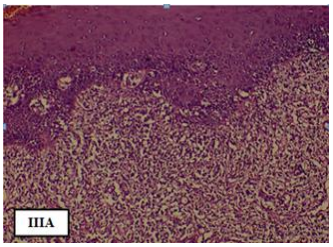

Figure IIIA: H&E-stained section in low Power Magnification showing basal cell degeneration,

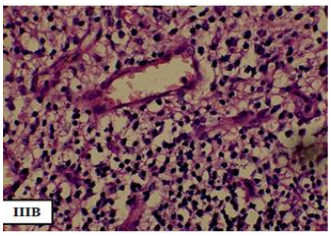

Figure III B: H&E-stained section high power magnification showing perivascular infiltration.

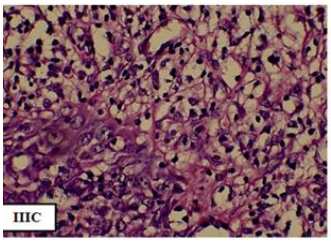

Figure III C: H&E-stained section in high power magnification showing presence of chronic non-specific

Table 1: Hemogram Report

Blood Test	Value	Normal Range
C4	8.79 mg/dl	10-40 mg/dl
ANA	1.4 AU/ml (POSITIVE)	<0.9 - Undetected 0.9-1.1 - Borderline >1.1- Detected
ESR	33 mm/hr	0-15 mm/hr
C3	99.4 (NORMAL)	90-180mg/dl

Table 2: EULAR/ACR classification criteria for systemic lupus erythematosus in children and adults by clinical rheumatology (2019) [3]

Entry criterion
Antinuclear antibodies (ANA) at a titer of ≥1:80 on HEp-2 cells or an equivalent positive test (ever)

↓

If absent, do not classify as SLE
If present, apply additive criteria

↓

Additive criteria
Do not count a criterion if there is a more likely explanation than SLE.
Occurrence of a criterion on at least one occasion is sufficient.
SLE classification requires at least one clinical criterion and ≥10 points.
Criteria need not occur simultaneously.
Within each domain, only the highest weighted criterion is counted toward the total score§.

Clinical domains and criteria	Weight	Immunology domains and criteria	Weight
Constitutional		***Antiphospholipid antibodies***	
Fever	2	Anti-cardiolipin antibodies OR	
Hematologic		Anti-β2GP1 antibodies OR	
Leukopenia	3	Lupus anticoagulant	2
Thrombocytopenia	4	***Complement proteins***	
Autoimmune hemolysis	4	Low C3 OR low C4	3
Neuropsychiatric		Low C3 AND low C4	4
Delirium	2	***SLE-specific antibodies***	
Psychosis	3	Anti-dsDNA antibody* OR	
Seizure	5	Anti-Smith antibody	6
Mucocutaneous			
Non-scarring alopecia	2		
Oral ulcers	2		
Subacute cutaneous OR discoid lupus	4		
Acute cutaneous lupus	6		
Serosal			
Pleural or pericardial effusion	5		
Acute pericarditis	6		
Musculoskeletal			
Joint involvement	6		
Renal			
Proteinuria >0.5g/24h	4		
Renal biopsy Class II or V lupus nephritis	8		
Renal biopsy Class III or IV lupus nephritis	10		

Total score:

↓

Classify as Systemic Lupus Erythematosus with a score of 10 or more if entry criterion fulfilled.

Table 3: Addition of the Critical Criteria's for SLE

Clinical Criteria	Points
Fever	2
Oral Ulcers	2
Subacute/Cutaneous Le	4
Joint Involvement	6
Low C3 Below	3
Total	17 (>10)

Conclusions

Systemic lupus erythematosus (SLE) is a complex autoimmune disorder with multisystem involvement, often presenting with both cutaneous and oral manifestations. Early recognition of oral lesions, such as erythematous erosive ulcerations, can aid in prompt diagnosis and intervention. Histopathological and immunofluorescence testing, along with serological markers like ANA and anti-dsDNA, play a crucial role in confirming the diagnosis. Management focuses on controlling disease activity, with corticosteroids, immunosuppressants, and hydroxychloroquine being key treatment options. Dental practitioners play a vital role in identifying oral manifestations, coordinating with specialists, and providing supportive care. A multidisciplinary approach is essential to optimize patient outcomes and improve quality of life.

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References

1. Smith CD, Cyr M. The history of lupus erythematosus: from Hippocrates to Osler. Rheumatic Disease Clinics of North America. 1988 Apr 1;14(1):1-4.
2. Rai H, Dayakar A, Supriya H, Sojan E. Discoid lupus erythematosus with epithelial dysplasia: a case report. International Journal of Research in Medical Sciences. 2017 Jun;5(6):2833
3. Silver R, Craigo S, Porter F, Osmundson SS, Kuller JA, Norton ME, Society for Maternal-Fetal Medicine (SMFM. Society for Maternal-Fetal Medicine Consult Series# 64: systemic lupus erythematosus in pregnancy. American Journal of Obstetrics and Gynecology. 2023 Mar 1;228(3):B41-60.
4. Angel A. Justiz Vaillant. (n.d.). Systemic Lupus Erythematosus - StatPearls - NCBI Bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK535405/
5. Shafer's Textbook of Oral Pathology, 9th Edition. (n.d.). https://dental-books.com/shafers-textbook-of-oral-pathology-9th-edition/
6. Oral Manifestations of Systemic Lupus Erythematosus: A Systematic ... (2022). https:// pmc. ncbi.nlm.nih.gov/articles/PMC9565705/
7. Discoid Lupus Erythematosus: A Cross-Sectional Study From the ... (2020). https:// pmc. ncbi. nlm. nih.gov/articles/PMC7704007/
8. Caroline H. Siegel, MD, MS. (2022). Lupus Blood Test Results: What to Know | HSS Rheumatology. https://www.hss.edu/conditions_understanding-

laboratory-tests-and-results-for-systemic-lupus-erythematosus.asp

9. Hahn BH. Systemic lupus erythematosus and accelerated atherosclerosis. The New England journal of medicine. 2003 Dec 1;349(25):2379-80
10. EULAR recommendations for the management of systemic lupus ... (2024). https://pubmed.ncbi. nlm. nih.gov/37827694/