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Oral Lupus Erythematosus Masquerading As Lichen Planus – A Case Report

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Abstract:

Lupus erythematosus (LE) is an immunologically mediated condition that can present as chronic discoid type or chronic cutaneous lupus erythematosus with lesions localized to skin or mucosa and usually with no systemic signs or symptoms. Oral lesions may be present in LE and it can be confused with lichen planus based on its clinical presentation. But LE has distinct histopathology which will help to confirm the diagnosis. In this case report, we record a case that mimicked lichen planus in its clinical presentation but was confirmed as LE by laboratory investigations and histopathological correlation.

Keywords:	Lupus erythematosus, Lichen planus, PAS positivity, ANA positivity
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1. Introduction

Lupus erythematosus was first described by Biett in 1828. It is considered to be an immune-mediated collagen vascular disease or a connective tissue disease. It can present either as a systemic form with involvement of any body organs or as a chronic discoid lesion localized to skin or mucosa. [1] Lupus erythematosus is an auto-immune disease in which auto-antibodies are directed against nuclear components. [2]

The first indication of systemic disease could be the occurrence of oral lesions. [2] Oral lesions have been reported in various dermatological, rheumatological and dental literature with a frequency varying from 3% to 45%. [3]

In this case report, we report an oral presentation of Lupus erythematosus that was clinically mimicking lichen planus but histological and laboratory investigations confirmed the diagnosis.

2. Case Report

A 72-year old male reported to the Department of Oral and Maxillofacial surgery, Surendera Dental College and Research Institute, Sriganganagar, Rajasthan, India. His chief complaint was burning sensation on the inner aspect of his cheeks after chewing food since three to four months.

History revealed that the lesion started as a small ulcer in the inner aspect of right and left cheeks but he did not receive any treatment and the ulcer healed

spontaneously. The lesion recurred 5 days back and was not relieved by self-medication. He also had a habit of tobacco chewing for 20 years and had quit the habit before 12 months. His past medical and surgical history was non-contributory.

Extra-oral examination did not reveal any abnormality. Intra-oral examination revealed a bluish-white erythematous lesion with variable brownish pigmentation. The lesion extended from the left labial mucosa to the buccal mucosa till the molar region. It had also involved the alveolar mucosa in the premolar-molar region. The periphery of the lesion showed multiple, linear striae. (Figure 1) The lesion was tender on palpation and firm in consistency. The condition was provisionally diagnosed as Lichen planus or Lichenoid reaction on the left buccal mucosa. An incisional biopsy was performed and sent to Department of Oral Pathology for further analysis.

Gross findings of the biopsy sample revealed a single soft tissue specimen which was brownish-white in color and an irregular surface. It measured 8mm x 4mm x 3mm in length, width and thickness. It was firm in consistence and the entire specimen was kept for tissue processing.

H & E stained tissue section revealed parakeratinized stratified squamous epithelium of variable thickness predominantly showing atrophy in many areas. There was evidence of basal cell degeneration, interface mucositis and sub-epithelial edema. The underlying fibrous connective tissue showed diffuse infiltrate comprising of lymphocytes. (Figure 2). There was presence of degeneration in few areas. There was evidence of mucous acini, adipose tissue, muscle tissue and areas of hemorrhage.

PAS staining was performed and it revealed deposits of PAS-positive material at the epithelial-connective tissue interface (Figure 3 & 4). Hence, the patient was advised for ANA test and it turned to be positive.

Hence, correlating the clinical features, histopathology, PAS positivity and ANA values, the lesion was diagnosed as Lupus erythematosus.

3. Discussion

Although LE is an autoimmune disease, it is caused by an interplay between various genetic and environmental factors. [3] LE is characterized by the reduction in T-cell control over the auto-reactive B-cells leading to production of numerous non-organ specific autoantibodies. [4] The pathogenesis may be attributed to post-zygotic mutation in the keratinocytes leading to expression of neoantigens that can elicit local immune response. [5] The disease can be triggered by trauma, ultraviolet light, drugs, heavy metals or other elements. The apoptosis of keratinocytes or expression of anomalous MHC or release of abnormal cytokines could be the possible mechanisms. [6]

DLE is frequently seen in older patients with a female predilection twice more often than men. [2,3] We present a case of LE involving a 72-year-old male patient.

Burge et al have reported that only 9% of LE cases had involved the buccal mucosa and palate. [7] When it involves oral mucosa, it can have an identical appearance to oral lichen planus. It can present as a relatively non-descript ulcer with an irregular outline, surrounded by erythematous mucosa perhaps bordered by radiating white striae or white papules. [2] Our patient presented an extensive lesion involving the buccal mucosa, alveolar mucosa and labial mucosa bordered by radiating striae that clinically looked like Lichen planus or Lichenoid reaction.

Histological assessment is a vital tool for confirming the diagnosis of LE. Thickening of basement membrane best demonstrated by periodic acid – Schiff staining along with vacuolar degeneration of basal cells with peri-vascular inflammatory cell infiltrate is a common feature. Hyperkeratosis and follicular plugging can be seen in mature lesions. [8] Karjalainen et al reported that the presence of pronounced edema, PAS positive thickening of blood vessel walls, deeper perivascular infiltrates and presence of mucin in lamina propria will help in diagnosis of LE. [9] Our case also presented with basal cell degeneration, pronounced edema and PAS positive deposits at the epithelial-connective tissue interface.

Histopathological distinction between acute, subacute and chronic cutaneous LE is based on the intensity of epithelial affection, severity of follicular damage, nature and level of the inflammatory infiltrate in the dermis. Oral lesions frequently show loss of epithelium and high frequency of ulcerated lesions due to marked basal cell liquefaction. [10] Our patient also presented with overlying epithelium of variable thickness and evident basal cell degeneration.

Serological tests for autoantibodies is essential for diagnosis and treatment of patients with collagen vascular diseases. The auto-antibody production is time-dependent with anti-nucleosome antibodies preceding the anti-DNA and anti-histone antibodies. [4] Our patient also showed serum positivity with ANA that helped us in the final diagnosis.

Indirect immunofluorescence with anti-nuclear antibodies can be performed as a first-level test in such patients. [4] Direct immunofluorescence is useful in differential diagnosis between LE and other related diseases. IgG, IgA, IgM and complement components can be seen in a linear or granular pattern in LE. It can be observed on up to 29% of oral lesions. [10]

4. Conclusion

Lupus erythematosus can present in diverse forms with variable histopathology. Strict histological assessment along with laboratory investigations is essential to differentiate it from similar conditions like lichen planus or lichenoid reactions.

References

1. Ranginwala AM, Chalishazar MM, Panja P, Buddhdev KP, Kale HM. Oral discoid lupus erythematosus: a study of twenty-one cases. J Oral Maxillofac Pathol 2012 Sep-Dec; 16 (3): 368-373.
2. Talacko AA, Gordon AK, Aldred MJ. The patient with recurrent oral ulceration. Australian Dental Journal 2010; 55: (1 suppl): 14-22.
3. Sarkar A, Ramalingam K, Verma P, Muddaiah S. Lupus erythematosus localized to oral cavity – a case report. BFUDJ 2011; 2: 2: 1-3.
4. Parmar S, Mayavanshi G, Sutaria A, Shah B. A study of Anti-Nuclear Antibody (ANA) in collagen vascular diseases. IAIM, 2016; 3(9): 34-39.
5. Seitz CS, Brocker EB, Trautmann A. Linear variant of chronic cutaneous lupus erythematosus: a clue for the pathogenesis of chronic cutaneous lupus erythematosus? Lupus 2008; 17:1136
6. Julià M, Mascaró JM Jr, Guilabert A, Navarra E, Ferrando J, Herrero C. Scleroermiform linear lupus erythematosus: a distinct entity or coexistence of two autoimmune diseases? J Am Acad Dermatol. 2008; 58:665-7.
7. Burge SM, Frith PA, Juniper RP, Wojnarowska F. Mucosal involvement in systemic and chronic cutaneous lupus

erythematosus. Br J Dermatol. 1989 Dec;121(6):727-41.
8. Panjwani S. Early diagnosis and treatment of discoid lupus erythematosus. J Am Board Fam Med. 2009 Mar-Apr;22(2):206-13.
9. Karjalainen TK, Tomich CE. A histopathologic study of oral mucosal lupus erythematosus. Oral Surg Oral Med Oral Pathol 1989; 67 (5): 547-554.
10. Nico MMS, Apparecida M, Vilela C, Rivitti EA, Lourenco SV. Oral lesions in lupus erythematosus: correlation with cutaneous lesions. Eur J Dermatol 2008; 18 (4): 376-81.

Figures

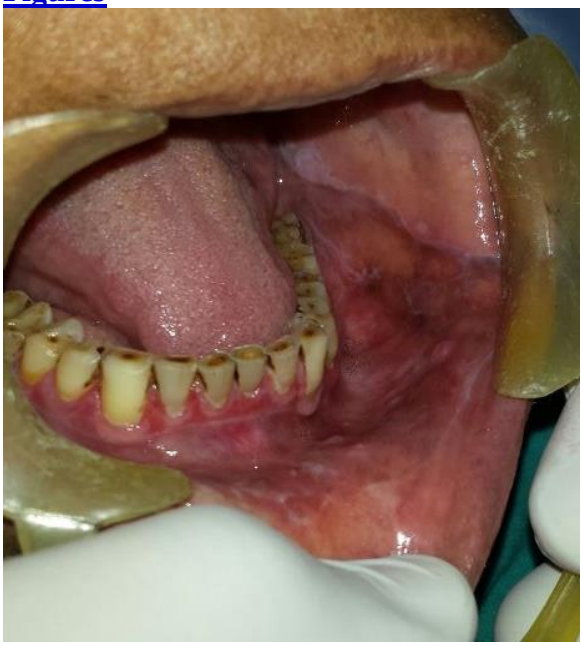

Figure 1: Clinical picture showing the erythematous patch with variable pigmentation and radiating white striae on the left buccal mucosa, labial mucosa and alveolar mucosa.

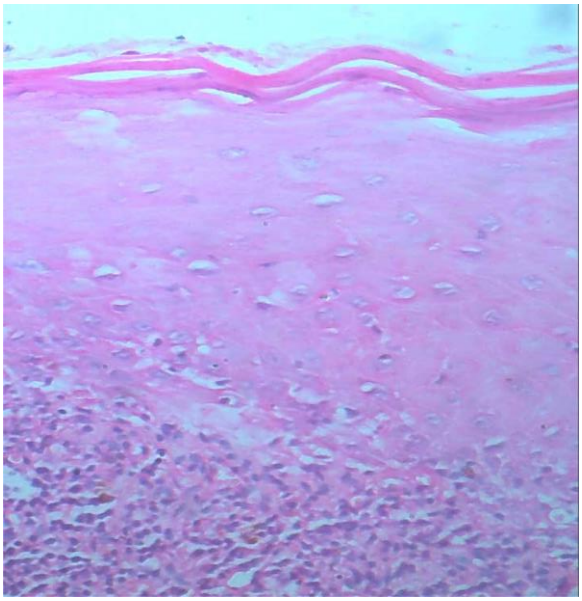

Figure 2: Photomicrograph showing stratified squamous epithelium of variable thickness with basal cell degeneration and intense inflammation in the connective tissue (H & E, 10x).

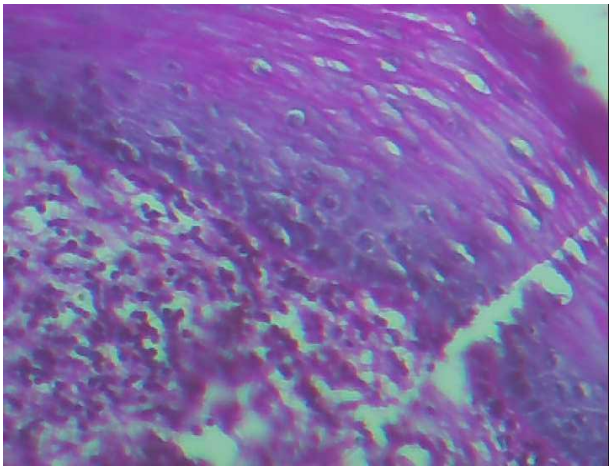

Figure 3: Photomicrograph showing highly vascular connective tissue stroma with diffuse inflammation (H & E, 10x).

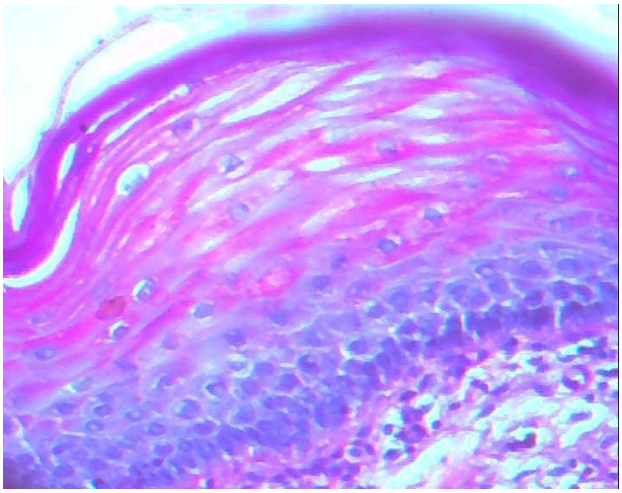

Figure 4: Photomicrograph showing hyperkeratosis and basal cell degeneration along with PAS positive material at the epithelium-connective tissue interface. (PAS, 10x).