Review Article

Oral manifestation and management of systemic lupus erythematosus - A short review

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ABSTRACT

Systemic lupus erythematosus (SLE) is a disease of unknown origin that can affect organs and cause severe damage. Lupus is diagnosed through biopsies and laboratory examinations; however, certain clinical characteristics and the presence of lesions can help with early diagnosis and improve the disease prognosis. SLE patients generally receive immune suppressants that may cause systemic implications - such as suture dehiscence, increased risk of infection, and delayedhealing - that deserve specific attention during dental treatment. This review presents studies done on SLE patient with oral manifestations: Ulcerative lesions in the mouth and brief about the treatment modality.

KEY WORDS: Management, Oral diseases, SLE

INTRODUCTION

Lupus erythematosus (LE) is an autoimmune disorder, in which the body's own immune system attacks its own tissues, especially components of the cellular nuclei. There are two main forms of lupus, discoid LE (DLE), affecting skin and mucous membranes, and systemic LE (SLE), which may also affect joints, visceral organs, and other tissues. The classification may now also include a bullous form, a neonatal form, an acute cutaneous form, a subacute cutaneous form, and a chronic cutaneous form, also very early onset and childhood onset SLE. In addition, there is a drug-induced form of SLE. SLE affects women 9–10 times as frequently as men and is mostly prevalent in ages from 18 to 65 years with a peak between 25 and 45 years. Studies show that SLE has high prevalence among Asian population. LE occurs as DLE and SLE.[1] Oral lesions occur in 25B50% of patients with DLE versus 7B26% of patients with SLE.[2,3] In DLE, these lesions usually begin as an irregular whitish area that extends peripherally as they extend, the central area may become red and ulcerated while the border remains elevated and hyperkeratotic.

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Oral lesions of lichen planus (LP) are similar to those of DLE both clinically and histologically. Strict histologic criteria must be applied to distinguish one from the other.[4] Oral or nasopharyngeal ulceration is recognized as a major diagnostic manifestation of SLE by the American Rheumatism Association Committee on Diagnostic and Therapeutic Criteria.[5] These ulcerations are generally painless and often involve the palate.[2] Purpuric lesions such as ecchymoses and petechiae may also occur. In up to 30% of patients with SLE, salivary gland involvement may occur concomitantly, leading to secondary Sjogren's syndrome and severe xerostomia.[6]

ORAL MANIFESTATION OF SLE

Oral ulceration is one of the revised diagnostic criteria proposed by the American College of Rheumatology for the classification of SLE.[7] The prevalence of oral lesions is reported to be 7–52% of patients with SLE.[8-11] Lesions due to the underlying disease process have been broadly classified into erythematosus, discoid, and ulcerative type.[12] However, other factors such as associated Sjögren's syndrome and mucosal alterations occurring as a result of treatment or intraoral infections need to be considered. One study specified an association of oral

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ulceration with clinical systemic activity according to defined guidelines on the basis of history and physical findings, although this did not correlate with significant changes in titers of serum complement (C3) or anti-DNA antibodies.[12] It has even been suggested that patients with oral ulcers have a higher mortality than those without oral ulcers,[9] although this has not been confirmed by further studies. The most recent study addressing this issue, by Jonsson *et al*.,[11] showed that the overall prevalence of oral lesions was not related to disease activity as defined by an arbitrary scale from clinical and laboratory data; however, discoid lesions and ulceration were only seen in patients with active disease. The buccal mucosa, hard palate, and vermilion border are the sites most frequently involved.[12] Discoid lesions appear as central areas of erythema with white spots surrounded by radiating white striae and telangiectasia at the periphery.[12,13] Erythematous lesions are often accompanied by edema and petechial reddening on the hard palate, although they are usually found incidentally as flat macular areas with poorly defined borders.[12,14] Ulcers tend to occur in crops and are shallow. They are usually 1–2 cm in diameter and in one-third of patients may extend into the pharynx.[13,14] All three lesions may coexist or merge into one another, leading to edema and petechiae.[12,14]

SLE and Sjogren Clinical Manifestation

Morgan[15] first described Sjogren's syndrome associated with SLE of 266 patients with SLE under long-term follow-up in our own unit, 13% have been diagnosed as having Sjögren's syndrome.[16] The prevalence of secondary Sjogren's syndrome occurs in SLE by some studies due to the lack of specific diagnostic criteria for classifying patients. For example, in a prospective study of manifestations of Sjogren's syndrome in 50 patients with SLE, all were questioned for sicca symptoms, and tests performed included Schirmer's and Rose Bengal tests, parotid sialography, salivary scintiscans, and isotopic excretion in saliva and lip biopsies.[17,18] Studies that have used well-defined histological and objective clinical criteria have shown that Sjogren's syndrome occurs in 20% of patients with SLE.[19,20] Sjogren's syndrome may precede SLE by many years, although it most frequently appears late in the course of the disease and in elderly patients with SLE.[21,22] Patients with SLE and secondary Sjogren's syndrome also tend to exhibit fewer systemic manifestations, particularly renal involvement, compared to those with SLE alone.[23,24]

PATHOLOGY OF SLE

The histology and immunopathology of oral lesion that are tend to be similar to changes seen in the skin are the presence of interface mucositis, hyperkeratosis, keratotic plugging, liquefactive degeneration, and inflammatory perivascular infiltrate, intraepithelial microabscess, spongiosis, civatte bodies, and deposition of immunoglobulin M (IgM), IgG, complement, and fibrogen along with dermal-epidermal junction.[2] Oral lesions in SLE are often difficult to distinguish from lesions of LP and other causes of leukoplakia. Karjalainen and Tomich[25] have revised Schiodt's original five features that occur in oral lupus and help to distinguish them from LP on light microscopy, and they are vacuolization of keratinocytes, patchy periodic acid-Schiff-positive deposits subepithelial, edema in upper lamina propria, and severe deep/perivascular infiltrate.

MANAGEMENT

Dental Consideration

Drugs used to control disease activity can cause significant intraoral pathology. Corticosteroids taken for many years can lead to root canal calcification.[26] Steroids have also been shown to be associated with acute necrotizing gingivitis in a few SLE patients.[27] Drugs like nonsteroidal anti-inflammatory drugs (NSAID's) have improved periodontal health in patients with SLE[28] possibly because NSAIDs inhibit alveolar bone resorption.[29] Drug-induced lupus secondary to hydralazine can cause orogenital ulceration,[30] and when a patient develops erosive or keratotic lesions on the buccal mucosa, the possibility of a lichenoid drug reaction secondary to hydroxychloroquine should be considered. Cyclosporin A, which may be of the use for some patients with active lupus, and the antihypertensive nifedipine are common causes of gingival hyperplasia. Although SLE itself has major adverse effects on normal immune functioning,[31] its immunosuppressive treatment undoubtedly facilitates intraoral infections, particularly *Candida* and herpes simplex virus. Preventative dental care is important. Patients have a tendency to consume a diet that promotes dental decay due to impaired taste. The use of chlorhexidine mouthwashes will help to contain periodontal disease and infection.[32] Symptomatic local treatment of mucous membrane ulcers with hydrogen peroxide gargle, buttermilk gargle, or steroid-impregnated gel may be of benefit. Intralesional injection of corticosteroids may be required.[33] Suspected or proven infections should be treated with antiviral, antifungal, or antibacterial agents after a swab has been taken for culture and sensitivities. Evidence suggests that dentists should probably not undertake dental work during a lupus flare and should treat lupus patients with prophylactic antibiotics before dental procedures due to the high incidence of bacterial endocarditis.[34]

Management of Sjogren's Syndrome

The treatment of secondary Sjogren's syndrome includes sugar-free gum, artificial saliva, and systemic therapy with pilocarpine hydrochloride which may increase salivary secretion.[35] Patients should receive oral hygiene instructions, prevention plans designed for people with oral dryness, and avoid antihistamines, tricyclic antidepressants, and decongestants when possible.

CONCLUSION

The purpose of this article was to describe the prevailing oral symptoms and signs of a population of patients with SLE. The results indicated certain prevalent objective manifestations: Xerostomia; angular cheilitis; chapped lips; mucositis; glossitis; caries; and periodontal disease; difficulties in chewing, swallowing, and eating; dysgeusia; and glossodynia. The high prevalence of oral problems indicates the necessity for thorough oral examinations as well as recognition and preventive management of these prevalent oral manifestations in dental patients with SLE.

REFERENCES

1. Hymes SR, Jordon RE, Arnett FC. Lupus erythematosus. Dermatol Clin 1986;4:267-76.
2. Urman JD, Lowenstein MB, Abeles M, Weinstein A. Oral mucosal ulceration in systemic lupus erythematosus. Arthritis Rheum 1978;21:58-61.
3. Rose LF, Kaye D. Internal Medicine for Dentistry. 3rd ed. St. Louis, MO: Mosby Yearbook; 1990. p. 800-1.
4. Rose LF, Kaye D. Internal Medicine for Dentistry. 3rd ed. St. Louis, MO: Mosby Yearbook; 1990. p. 93-4.
5. Cohen AS, Canoso JJ. Criteria for the classification of systemic lupus erythematosus. Arthritis Rheum 1972;15:540-3.
6. Grennan DM, Ferguson M, Williamson J, Mavrikakis M, Dick WC, Buchanan WW, *et al*. Sjogren's syndrome in SLE. N Z Med J 1977;86:374-9.
7. Tan E, Cohen A, Fries J, Masi A, McShane D, Rothfield N, *et al*. Special article: The 1982 revised criteria for the classification of systemic lupus erythematosis. E-Arthritis Rheum 1982;25:1271-7.
8. Dubois E, Tuanelli D. Clinical manifestations of systemic lupus erythematosus. Computer analysis of 520 cases. J Am Med Assoc 1964;190:104-11.
9. Fries J, Holman HR. Systemic Lupus Erythematosus: A Clinical Analysis. Philadelphia, PA: WB Saunders; 1975.
10. Harvey AM, Shulman LE, Tumulty PA, Conley CL, Schoenrich EH. Systemic lupus erythematosus: Review of the literature and clinical analysis of 138 cases. Medicine (Baltimore) 1954;33:291-437.
11. Estes D, Christian CL. The natural history of systemic lupus erythematosus by prospective analysis. Medicine (Baltimore) 1971;50:85-95.
12. Jonsson R, Heyden G, Westberg NG, Nyberg G. Oral mucosal lesions in systemic lupus erythematosus-a clinical, histopathological and immune-pathological study. J Rheumatol 1984;11:38-42.
13. Schiødt M. Oral discoid lupus erythematosus. III. A histopathologic study of sixty-six patients. Oral Surg Oral Med Oral Pathol 1984;57:281-93.
14. Sultan SM, Ioannou Y, Isenberg DA. A review of gastrointestinal manifestations of systemic lupus erythematosus. Rheumatology (Oxford) 1999;38:917-32.
15. Morgan WS. The probable systemic nature of Mikulicz's disease and its relation to Sjögren's syndrome. N Engl J Med 1954;251:5-10.
16. Vitali C, Bombardieri S, Moutsopoulos HM, Balestrieri G, Bencivelli W, Goo RM, *et al*. Preliminary criteria for the classification of Sjögren's syndrome. Results of a prospective concerted action supported by the European community. Sch Arthritis Rheum 1993;36:340-7.
17. Ramos-Niembro F, Alarcon-Segovia D. Development of sicca symptoms in systemic lupus erythematosus patients with existing subclinical abnormalities of lacrimal and/or salivary glands. Arthritis Rheum 1979;22:935-6.
18. Alarcio-Segova D, Ibanez G, Velazquez-Forero F, Hernandez-Ortiz J, Gonzalez-Jimerez Y. Sjögren's syndrome in systemic lupus erythematosus: Clinical and suclinical manifestations. Ann Intern Med 1974;81:557-83.
19. Andonopoulos AP, Skopouli FN, Dimou GS, Drosos AA, Moutsopoulos HM. Sjögren's syndrome in systemic lupus erythematosus. J Rheumatol 1990;17:201-4.
20. Moutsopoulos H, Webber B, Vlagopoulos T, Chused T, Decker J. Differences in the clinical manifestations of sicca syndrome in the presence and absence of rheumatoid arthritis. J Rheumatol 1979;22:1152-8.
21. Romero RW, Nesbitt LT Jr, Ichinose H. Mikulicz disease and subsequent lupus erythematosus development. JAMA 1977;237:2507-10.
22. Zuerey P, Meyer O, Bourgeois P, Vayssairat M, Kahn M. Primary systemic Sjögren's syndrome (SS) preceding systemic lupus erythematosus: A retrospective study of 4 cases in a cohort of 55 SS patients. Lupus 1995;4:23-7.
23. Grennan DM, Ferguson M, Ghobarey AE, Williamson J, Dick WC, Buchanan WW. Sjögren's syndrome in SLE Part 2. An examination of the clinical significance of Sjögren's overlap syndrome by comparison of its frequency in typical and atypicale forms of SLE, overlap syndromes and scleroderma. N Z Med J 1977;86:376-9.
24. Grennan DM, Ferguson M, Williamson J, Mavrikakis M, Dick WC, Buchanan WW. Sjögren's syndrome in SLE: Part 1. The frequency of the clinical and subclinical features of Sjögren's syndrome in patients with SLE. N Z Med J 1977;86:374-6.
25. Karjalainen TK, Tomich CE. A histopathologic study of oral mucosal lupus erythematosus. Oral Surg Oral Med Oral Pathol 1989;67:547-54.
26. Gold SI. Root canal calcification associated with prednisone therapy: A case report. J Am Dent Assoc 1989;119:523-5.
27. Jaworski CP, Koudelka BM, Roth NA, Marshall KJ. Acute necrotizing ulcerative gingivitis in a case of systemic lupus erythematosus. J Oral Maxillofac Surg 1985;43:43-6.
28. Feldman R, Szet B, Ghauncey H, Goldhober P. Non-steroid anti-inflammatory drugs in the reduction of human alveolar bone loss. J Clin Periodontol 1983;10:131-6.
29. Neville E, Graham PY, Brewis RA. Orogenital ulcers, SLE and hydralazine. Postgrad Med J 1981;57:378-9.
30. Callen JP. Oral manifestations of collagen vascular disease. Semin Cutan Med Surg 1997;16:323-7.
31. Iliopoulos AG, Tsokos GC. Immunopathogenesis and spectrum of infections in systemic lupus erythematosus. Semin Arthritis Rheum 1996;25:318-36.
32. Scully C, Laskaris G. Mucocutaneous disorders. Periodontol 2000 1998;18:81-94.
33. Soto-Rojas A, Villa A, Sifuentes-Osornio J, Alarcon-Segovia D, Kraus A. Oral manifestations in patients with Sjögren's syndrome. J Rheumatol 1998;25:906-10.
34. Luce EB, Presti CF, Montemayor I, Crawford MH. Detecting cardiac valvular pathology in patients with systemic lupus erythematosus. Spec Care Dentist 1992;12:193-7.
35. Rhodus NL, Schuh MJ. Effects of pilocarpine on salivary flow in patients with Sjögren's syndrome. Oral Surg Oral Med Oral Pathol 1991;72:545-9.