European Journal of Molecular & Clinical Medicine
ISSN 2515-8260 Volume 07, Issue 5, 2020

Systemic Lupus Erythematosis And Discoid Lupus Erythematosis- An Overview

Dr.L.Malathi, Dr S.Aafiya Reshma, Dr.N.Anitha, K.M.K.Masthan

Department of Oral and Maxillofacial pathology
Sree Balaji Dental College and Hospital
Bharath institute of Higher Education and Research

ABSTRACT:

Lupus is associated with multisystemic inflammation resulting from abnormal lupus erythematosus. Systemic Lupus erythematosus(SLE)is an autoimmune disease in immunlological function. The four main types of lupus are Systemic Lupus erythematosus(SLE), Discoid Lupus Erythematosus(DLE), drug induced lupus, neonatal and paediatric induced where the immune system attacks its own tissues, causing inflammation in widespread and tissue damage in the affected organs. It can affect the joints, skin, brain, oral cavity, lungs, kidneys, and blood vessels. Discoid lupus erythematosus (DLE) is a chronic, scarring, atrophy producing, photosensitive dermatosis. This article reviews about its pathophysiology, oral manifestations and its clinical management.

Keywords: *Systemic Lupus Erythematous, Discoid Lupus Erythematosus,autoimmune disease, oral lesions, management, immunosuppressives, corticosteroids*

INTRODUCTION:

Systemiclupus erythematosus(SLE) is an autoimmune disease characterized by autoantibodies, immune complex formation, and immune dysregulation resulting in damage to any organ, including the skin blood vessels, and CNS. Early diagnosis and careful treatment tailored to individual patient symptoms has improved the prognosis from what was once perceived as an often fatal disease.

PATHOPHYSIOLOGY:

The specific cause of SLE is not defined. Various research suggests factors contribute tlupus are genetics, hormones and the environment . It is a chronic disease affecting organ systems, as a consequence of formation and deposition of immune complexes and autoantibodies, which leads to organ damage. Hyperactive B cells, results in T-cell and antigen stimulation thus increasing the production of these antibodies against antigens which are exposed on the surface of apoptotic cells[1]. The antigens causing T-cells and B-cell stimulation leads to inappropriate disposal of apoptotic cells. Pieces of cellular material are found lying on the surface of dying cell during rocess of cellular death. The antigens which are identified in SLE patients are Nucleosomes and anionic phospholipids which have ability to trigger an immune response[1,2]. When a T-lymphocyte to an antigen presenting cell is introduced, SLE may develop. The release of cytokine, inflammation and B-cell stimulation are by the binding of T-cell receptor to the major histocompatibility complex(MHC) of the APC[1]. Production of immunoglobulin G (Ig G) autoantibodies and B-cell division stimulation causing tissues damage also occurs in SLE[1,3,4] Autoantibodies identified in SLE -the anti nuclear antibodies (ANA) target nuclear components of the cell and detection of ANA in SLE patients is essential for proper diagnosis[4]. The ANA's that has been tested extensively, with involvement confirmed in SLE, are the anti– double-stranded (ds) DNA antibodies[5]. ANA's also interact with single-stranded (ss) DNA as well as with RNA. Examples of ANAs are the anti-Ro and anti-La antibodies that, when detected during pregnancy, have been linked to fetal heart damage and the anti-Smith (Sm) antibodies, a marker of kidney disease[6,7]. The phospholipid moiety of the prothrombin activator complex and cardiolipin are targeted by the second group of autoantibodies and these antiphospholipid antibodies may lead to abnormal clotting as well as loss of pregnancy[8].

ORAL MANIFESTATIONS:

According to Andreasen, Oral mucous membrane involvement is slightly more frequent in SLE than DLE(20-50%). The involvement of oral mucosa may be either prior to or following or even in the absence of skin manifestations. Oral lesions of SLE are very similar to oral lesions of DLE expect in SLE, the severity of

i. Hyperemia, oedema and extension of the lesion
ii. Bleeding, petechiae, and superficial ulcerations surrounded by red halo as a result of localized telangiectasis

are noticed. Xerostomia and superimposed oral moniliasis are also reported. Painless shallow oral ulcers most often occurs on hard and soft palate. There is also a mild involvement of mucosal ulcers as symptom of this disease.Oral ulcers occur at beginning in 11% of patients, while at any time are present in 30% of patients.The lesions manifest as maculae (red patches) which will later change into irregular erosions and ulcers that often heal with scarring. Purpuric lesion such as ecchymoses and petechiae may occur[10,11]. According to Sugarman, variation of oral lesions exists and stimulate other diseases such as leukoplakia and lichen planus[9]. Lesions usually affects the palate, buccal mucosa and gingivae. Salivary gland involvement may occur leading to secondary Sjogren's syndrome, and severe xerostomia in 30% of SLE patients[12].

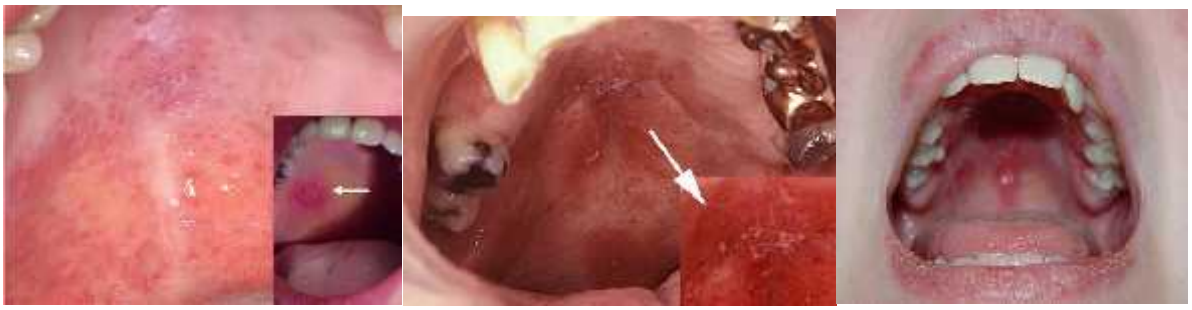

Fig 1 Systemic Lupus Erythematosus[10,11]

DIAGNOSIS: Oral or nasopharyngeal ulceration is recognized as a major diagnostic manifestation of SLE by the American Rheumatism Association Committee on Diagnosticand Therapeutic Criteria[12.]

Commonly used blood tests in the diagnosis of SLE are[9]:

1. Anti nuclear antibody test(ANA)
2. Anti- DNA antibody test
3. Anti-Sm antibody test
4. Serum(blood) complement test
5. Complement proteins C3 and C4 test

MANAGEMENT[13,14]:

General patient education on sun protection, proper diet and nutrition, exercise, smoking cessation, appropriate immunizations and management of comorbid conditions.

MANAGEMENT OF SLE USING MEDICATIONS:

Class of Drugs	Agents and dosages	Adverse effects
NSAIDs (including salicylates)	Various agents and dosages	Gastrointestinal irritation and bleeding, renal toxicity, hepatic toxicity, hypertension
Antimalarials	Hydroxychloroquine PO 200–400 mg daily	Macular damage, muscle weakness

Corticosteroids	Prednisone PO 0.5–2 mg/kg per day Methylprednisolone IV 500–1,000 mg daily for 3 to 6 days (acute flare)	Weight gain, hypertension, hyperglycemia, hyperlipidemia, osteoporosis, cataracts, edema, hypokalemia, muscle weakness, growth suppression, increased risk of infection, glaucoma
Immunosuppressants	Cyclophosphamide PO 1–3 mg/kg per day or 0.5–1 g/m2 IV monthly with or without a corticosteroid Azathioprine PO 1–3 mg/kg per day Mycophenolate PO 1–3 g daily	Myelosuppression, hepatotoxicity, renal dysfunction, infertility, increased risk of infection and cancer
Monoclonal antibodies	Belimumab IV 10 mg/kg (over a period of 1 hour), every 2 weeks for the first three doses, then every 4 weeks	Nausea, diarrhea, pyrexia, nasopharyngitis, insomnia, extremity pain, depression, migraine, gastroenteritis, infection(Eg;pneumonia, UTI).

TREATMENT:

Systemic Lupus Erythematosus is a lifelong illness and patients shoud be monitored indefinitely. It is a high risk disease with end organ damage possibilities. The damage can affect severely the function of orgnas and quality of life[9].

CONCLUSION:

With recent advances by understanding the pathogenesis and mechanism of SLE, the control of disease and management of comorbidities can be focused.

DISCOID LUPUS ERYTHEMATOSUS

INTRODUCTION:

Discoid lupus erythematosus(DLE) is a most common form of chronic cutaneous lupus erythematosus. It begin as a red purple macules,papules or small plaques and rapidly develop a hyperkeratotic surface. DLE may occur in patients with SLE and in some patients (>5%) DLE progresses to SLE. DLE patients rarely develop systemic disease and produce scarring and atrophy[9].

PATHOPHYSIOLOGY:

The pathophysiology of DLE may be suggested as a heat shock protein induced in the keratinocyte following UV light exposure or stress, and this protein may act as a target for T-cell mediated epidermal cell cytotoxicity[9]. The aetiology may be multifactorial which can be genetic and environmental factors. The interaction of multiple factors triggers an inflammatory cascade of cytokine, chemokine, inflammatory cell responses. TYK2,IRF5 and CTLA4 are the genes associated with SLE and also has an increased risk of DLE[15].

ORAL MANIFESTATIONS:

The oral mucous may be involved either prior or following the development of skin lesions or even in the absence of the skin manifestations. The oral lesions in the discoid form begin as erythematous area, more often depressed and slightly elevated typically with white spots without induration. Oral manifestation appears as superficial, painful ulceration with crusting or bleeding and the margins are not sharply demarcated showing the formation of narrow zone

of keratinization. Most common sites are buccal mucosa, tongue, palate, vermilion border of the lower lip. Atrophy of the papillae and severe fissuring is seen in tongue with erythematous, atrophic plaques surrounded by the keratotic border involving the entire lip.According to Andreasen, malignant transformation can occur with some frequency[9,16,17,18].

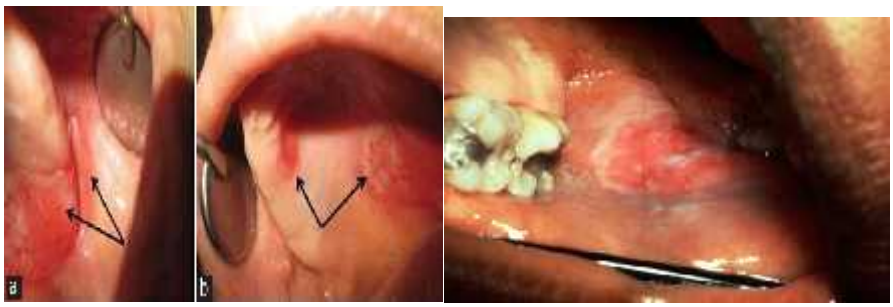

Fig 2 Discoid lupus erythematosus

TREATMENT AND MANAGEMENT:

Recent first line of treatment for DLE is photoprotection in conjugation with oral corticosteroids, topical calcineurin inhibitors and systemic antimalarial therapy. Currently, no medications have been approved specifically. The goals of management of DLEis to improve the patient's appearance, to control existing lesions and limit scarring, and to prevent the development of further lesions[9].

CONCLUSION:

The DLE and its associated lesions have impact on dental management and the oral lesions are difficult to resolve. With appropriate training and understanding the complex manifestation of DLE, the management can be provided.

References:

1. Rahman A, Isenberg, D. Systemic lupus erythematosus. N Engl J Med 2008;358:929–939.
2. Casciola-Rosen LA, Anhalt G, Rosen A. Autoantigens targeted in systemic lupus erythematosus are clustered in two populations of surface structures on apoptotic keratinocytes. J Exp Med 1994;179:1317–1330.
3. Ravirajan CT, Rahman MA, Papadaki L, et al. Genetic, structural and functional properties of an IgG DNA-binding monoclonal antibody from a lupus patient with nephritis. Eur J Immunol 1998;28:339–350. [Erratum, Eur J Immunol 1999;29:3052.]
4. Rahman A. Autoantibodies, lupus and the science of sabotage. Rheumatology (Oxford) 2004;43:1326–1336
5. Isenberg DA, Manson JJ, Ehrenstein MR, Rahman A. Fifty years of anti-dsDNA antibodies: Are we approaching journey's end? Rheumatology (Oxford) 2007;46:1052–1056.
6. Buyon JP, Clancy RM. Maternal autoantibodies and congenital heart block: Mediators, markers, and therapeutic approach. Semin Arthritis Rheum 2003;33:140–154.
7. McCarty GA, Harley JB, Reichlin M. A distinctive autoantibody profile in black female patients with lupus nephritis. Arthritis Rheum 1993;36:1560–1565.
8. Alarcon-Segovia D, Deleze M, Oria CV, et al. Antiphospholipid antibodies and the antiphospholipid syndrome in systemic lupus erythematosus: A prospective analysis of 500 consecutive patients. Medicine (Baltimore) 1989;68:353–365.
9. Rajendra R, Sivapathasundharam B (2012) Shafer's textbook of oral pathology. (7th edn), Elsevier, New Delhi, India.
10. Long, R.G. et al. (1998): Oral manifestations of systemic diseases, The Mount Sinai Journal of Medicine(N.Y.-USA) Vol. 65, No.5-6
11. Sultan, S.M. et al. (1999): A review of gastrointestinal manifestations of systemic lupus erythematosus, (Oxford Journal of Rheumatology, United Kingdom) Vol. 38, No.10
12. Long RG, Hlousek L, Doyle JL. Oral manifestations of systemic diseases. Mt Sinai J Med 1998;65:309-315.
13. American College of Rheumatology Ad Hoc Committee on Systemic Lupus Erythematosus Guidelines. Guidelines for referral and management of systemic lupus erythematosus in adults. Arthritis Rheum 1999;42:1785–1796. 82.

14. Bertsias GK, Ioannidis JPA, Arlinger M, et al. EULAR recommendations for the management of systemic lupus erythematosus. Report of a Task Force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics. Ann Rheum Dis 2008;67:195–205
15. Brianna McDaniel; Sukesh Sukumaran; Laura S. Tanner:Discoid Lupus Erythematosus
16. Archard HO, Roebuck NF, Stanley HR, Jr. Oral manifestations of chronic discoid lupus erythematosus: report of a case. Oral Surg, 16: 696, 1963.
17. Schiodt M, Andersen L, Shear M, Smith LJ. Leukoplakia-like lesions developing in patients with oral discoid lupus erythematosus. Acta OdontolScand, 39: 209, 1981.
18. Gallego H, Crutchfield CE 3rd, Lewis EJ, Gallego HJ. Report of an association between discoid lupus erythematosus and smoking. Cutis, 63(4): 231–34, Apr, 1999.