Systemic Lupus Erythematosis And Discoid Lupus Erythematosis- An Overview

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ABSTRACT:

Lupus is associated with multisystemic inflammation resulting from abnormal lupus erythematosus. Systemic Lupus erythematosus(SLE)is an autoimmune disease in immunological 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:

Systemic lupus 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 lupus 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. 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. 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. Production of immunoglobulin G (Ig G) autoantibodies and B-cell division stimulation causing tissues damage also occurs in SLE. 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. The ANA’s that has been tested extensively, with involvement confirmed in SLE, are the anti– double-stranded (ds) DNA antibodies. 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. 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.
ORAL MANIFESTATIONS:

According to Andreasan, 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. According to Sugarman, variation of oral lesions exists and stimulate other diseases such as leukoplakia and lichen planus. Lesions usually affects the palate, buccal mucosa and gingiva. Salivary gland involvement may occur leading to secondary Sjogren’s syndrome, and severe xerostomia in 30% of SLE patients.

![Systemic Lupus Erythematosus](image)

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

Commonly used blood tests in the diagnosis of SLE are:

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:

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:

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<tr>
<th>Class of Drugs</th>
<th>Agents and dosages</th>
<th>Adverse effects</th>
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<tbody>
<tr>
<td>NSAIDs (including salicylates)</td>
<td>Various agents and dosages</td>
<td>Gastrointestinal irritation and bleeding, renal toxicity, hepatic toxicity, hypertension</td>
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<tr>
<td>Antimalarials</td>
<td>Hydroxychloroquine PO 200–400 mg daily</td>
<td>Macular damage, muscle weakness</td>
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TREATMENT:
Systemic Lupus Erythematosus is a lifelong illness and patients should be monitored indefinitely. It is a high risk disease with end organ damage possibilities. The damage can affect severely the function of organs and quality of life.

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.

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. 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.

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\textsuperscript{9,16,17,18}.

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 DLEs to improve the patient’s appearance, to control existing lesions and limit scarring, and to prevent the development of further lesions\textsuperscript{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:

15. Brianna McDaniel; Sukesh Sukumaran; Laura S. Tanner: Discoid Lupus Erythematosus