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CUTANEOUS LUPUS ERYTHEMATOSUS

General Medicine

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KEYWORDS

INTRODUCTION

SLE is an autoimmune disease with multi-organ involvement. Cutaneous involvement i.e. Cutaneous lupus erythematosus (CLE) is the second most commonly involved site. Cutaneous involvement occurs in 70-85% of all individuals over the course of the disease. It is the presenting symptom in 25% of the patients. CLE has been classified into various types.

The patients with CLE can have a bad emotional component. It has an incidence of 3-4/100,000 and a prevalence of 70/100,000 in the US. Four of the 11 criteria in the 1997 American College of Rheumatology (ACR) diagnostic criteria for SLE are cutaneous manifestations including malar rash, discoid rash, photosensitivity and oral ulcers. The 2019 the European alliance of association for Rheumatology EULAR/ACR classification criteria for SLE include positive ANA followed by additive weighted criteria in 7 clinical and 3 immunological domains. Patients accumulating >10 points are classified as having SLE.[1]

Mucocutaneous is one of the seven clinical fields and includes alopecia, oral ulcers, subacute CLE, DLE and acute CLE. The Systemic Lupus International Collaborating clinics (SLICC) criteria classify a patient as having SLE if they have biopsy proven nephritis with positive ANA or anti-ds-DNA antibody or at least 4 out of 17 criteria including at least 1 immunological criteria and 1 clinical criterion. Four of the clinical criteria are mucocutaneous in nature-ACLE, CCLE, oral ulcers and nonscarring alopecias.

CLE is divided into LE-specific and LE-nonspecific skin conditions. LE-specific skin conditions include CCLE, SCLE and ACLE and their subtypes, ICLE.[2]

Histologic Features

The histologic features of CLE includes- lichenoid interface dermatitis, with basal layer vacuolization, apoptotic keratinocytes, periadnexal and perivascular mononuclear cell infiltrate, epidermal atrophy, and basement membrane thickening. Interface dermatitis is not typically seen. Biopsy is recommended to confirm the diagnosis of CLE. It is possible that patients have more than one form of CLE. The patients with localized DLE, hypertrophic LE, LEP and LET are more likely to have skin limited LE.

The cutaneous lupus erythematosus disease severity index (CLASI) is a validated instrument that has separate scores to measure activity and damage of CLE. Scores are based on the extent of erythema, scale/hypertrophy, mucus membrane involvement, acute hair loss and non-scarring alopecia. Damage scores are based on dyspigmentation and scarring including scarring alopecia. New variables like edema/infiltration and subcutaneous nodules/plaques have been added.[3]

Treatment

The treatment of CLE includes to prevent and treat the skin activity to minimize the damage. Sun protective measures like protective clothing, avoiding exposure during peak sunlight hours and daily use of SPF70 or higher UV sunscreens. Vitamin-D supplementation in all the patients. Smoking cessation should be advised.

Topical and intralesional steroids can be used in the limited cutaneous disease or as adjunctive therapy with systemic agents. An initial regimen of medium strength (Class-III) topical corticosteroids such as triamcinolone acetonide 0.1% applied daily to lesional skin can be tried on areas off the face. A more potent topical steroid such as clobetasol propionate 0.05% or betamethasone dipropionate 0.05% (Class I) should be considered. Chances of developing cutaneous atrophy from longer term therapy can be prevented by rotating the topical corticosteroids every 2 weeks with a topical calcineurin inhibitor such as pimecrolimus cream or tacrolimus ointment.

Anti-malarials (hydroxychloroquine, chloroquine and quinacrine) are first line therapies for cutaneous disease in SLE. Disease refractory to antimalarials may be treated with immunosuppressive agents like methotrexate, mycophenolate mofetil or azathioprine. Thalidomide is recommended in the treatment of refractory CLE. Dapsone can be effective in treatment of bullous LE, LEP and some cases of SCLE and DLE. ACLE can respond to Rituximab. Oral retinoid is an alternative therapy if antimalarials do not work. Acitretin has been shown to be effective in half of CLE patients in a randomized controlled trial.[4]

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