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A CASE OF SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) PRESENTING AS CUTANEOUS LUPUS

General Medicine

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ABSTRACT

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. Here we present a case of a patient who presented with the typical skin involvements and was found to have SLE.

KEYWORDS

INTRODUCTION

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.

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.[2]

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]

CASE REPORT

A 30 years old female presented to the Medicine OPD with chief complaints of

Skin eruptions for the past 1 month
Itching for the past 1 month
Fever for the past 1 month

The history of the present illness showed that the patient was alright 1 month back when she started experiencing skin eruptions over the face and back. These skin changes were erythematous in nature and scaly. The skin eruptions later progressed and merged together over days. The lesions were more prominent on exposure to the sunlight. The patient also complained of itching in the skin lesions which also started 1 month back. The itching increased on exposure to the sunlight. The patient also complained of fever for the past 1 month. It was of low grade and continuous in nature. There was no diurnal variation.

There was no history of joint pains, anuria, or oliguria or haematuria.

In the past history, there was nothing significant and the patient was not suffering from any disease in the past. There was no significant family history. The patient was not on any treatment in the past. The patient was a vegetarian and the bowel and bladder function was normal. The patient was having any addiction. The menstrual history of the patient was normal.

On general physical examination, the patient was consious, cooperative and sitting comfortably in the bed. The BMI of the patient was 20.9. The patient had a temperature of 100.5° F, pulse rate of 102/min, regularly regular, normal volume, normal character, no radiofemoral delay, condition of the vessel wall normal, all peripheral pulses well felt. Blood pressure was 128/84 mmHg. The respiratory rate was 18/min, thoracoabdominal. Pallor was present. There was no icterus, cyanosis, clubbing, lymphadenopathy or edema. JVP was not raised. An erythematous and scaly rash was present over the back and the chest. There was also presence of malar rash over the face.

On systemic examination, all the major systems were normal. A provisional diagnosis of SLE was kept along with a DD of Infectious disease.

The investigations done showed a Hb of 9 gm/dl, MCV of 86 fl, Platelet count of 1 lakh/mm^3. LFT and RFT were normal. RBS was 110 mg/dl. Chest X-ray was normal. ESR was 90 mm Ist hr, CRP was positive. ANA, ds-DNA, anti-SSA/Ro and SSB/La antibody tests were positive. The skin biopsy done showed basal cell damage (interface dermatitis), lymphohistiocytic inflammatory infiltrates. The lupus band test was positive. Based on the results of the investigations, a diagnosis of cutaneous lupus form of SLE was made.

Treatment was started and patient was given advice regarding wearing of wide brimmed hats, sun protective clothing and applying broad spectrum sunscreens (SPF 50 or more) was given. Patient was also started on topical steroid creams and oral steroids in the form of Prednisolone. Calcineurin inhibitors tacrolimus and pimecrolimus topical application was also given. Hydroxychloroquine, Methotrexate, Azathioprine were also given. Vitamin-D supplementation was also given.

Advice regarding the healthy diet, exercise, stress management, sufficient sleep was given. The skin lesion of the patient improved and the patient was discharged in good general condition.

DISCUSSION

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]

CONCLUSION

The SLE presenting predominantly as a cutaneous disease is a rare disease and requires a high index of suspicion for it's diagnosis. The presentation in the patient with a classical signs and symptoms of Skin involvement and antibodies positive for SLE and further response to the treatment given for the SLE confirms the diagnosis of CLE.

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