

Overview of diagnosis and treatment options of autoimmune disease: cutaneous lupus

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ABSTRACT

Foundational lupus erythematosus is a persistent provocative condition which influences dominantly ladies in their 30s. Skin lesions that fall under the categories of acute cutaneous lupus erythematosus, subacute clinical manifestations include cutaneous lupus erythematosus and chronic cutaneous lupus erythematosus. It is recommended to treat cutaneous lupus in a variety of ways.

Keywords: Autoimmune Disease; Lupus; Skin lesions; Clinical manifestations

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INTRODUCTION

Systemic lupus erythematosus is an autoimmune condition that can affect the skin and/or mucous membranes as well as multiple organ systems in the body. Over 80% of SLE patients may be affected by these. There are skin lesions that are unique to lupus and skin lesions that are not. Sunlight frequently causes cutaneous lupus erythematosus, which typically affects women between the ages of 20 and 50. It is broken down into three main groups: chronic cutaneous lupus erythematosus, acute cutaneous lupus erythematosus, and subacute CLE. The latter constitutes 75% of all CLE cases, making it the most prevalent subtype. Because skin involvement and systemic involvement do not always correlate, it is essential to properly assess both. Patients with CLE have a risk of developing SLE of up to 28%, and earlier diagnosis and treatment may delay the onset of SLE [1].

As per the new Foundational Lupus Global Working together Facilities rules distributed in 2012, ACLE sores, CLE sores, oral and nasal ulcers, and no scarring alopecia structure four of the 11 clinical measures for the analysis of SLE. Malar rash, discoid rash, photosensitivity, and oral ulcers are four of the 11 rules from the American School of Rheumatology-supported SLE analysis measures. SLE and CLE both have similar incidence rates. CLE is multiple times more normal than SLE in men. Around 33% of patients with Subacute CLE have sores that are connected with the utilization of medications including terbinafine, thiazides, proton siphon inhibitors, and statins. As a result, controlling the condition necessitates quitting these medications [2].

Common diagnosis guidelines

The treatment of CLE ought to be isolated into no pharmacological and pharmacological methodologies. In the non-pharmacological approach, patient adherence must be addressed at each visit. Sunscreen application and physical protection, such as broad-brimmed hats and sun-protective clothing, are essential. Plasmacytoid dendritic cells are more responsive to toll-like receptor 9 and produce more IFN type 1 when smokers smoke. Additionally, tobacco causes photo toxicity and increases metalloproteinase 1 through 8 expression. As a result, quitting smoking is of the utmost importance. In addition, vitamin D supplementation has been found to improve medication response [3].

The use of calcineurin inhibitors and topical corticosteroids as part of the pharmacological therapy approach is widely accepted. Localized areas could benefit from intralesional

CSs. When a patient with CLE does not respond to topical treatment and/or the cutaneous disease is widespread, systemic therapy should be considered. Finding the suitable treatment for a patient could challenge. It is common knowledge that antimalarial treatment will be effective in 66% of CLE patients. One more gathering would answer suitably when immunomodulatory or immunosuppressive prescriptions are added to the routine; however, 10% of patients have recalcitrant lesions or are intolerant [4].

New advances in fundamental and translational examination in autoimmunity have been critical in the improvement of these new choices, which incorporate organic specialists. Key inflammatory pathways are the focus of these new drugs, which are crucial to the disease's pathogenesis. The next obstacle is accurately predicting an individual's response to these new medications that has CLE. The next objective is to tailor treatment to each patient's particular disease. The treatment aims to reduce scarring, prevent new skin lesions, and enhance the skin's appearance. The various medical treatments available to patients with CLE are briefly discussed in this article [5].

Treatments

The most commonly used topical treatments are calcineurin inhibitors and CSs. The most widely used types of topical CSs are fluorinated and non-fluorinated. They are first-line agents, but if used for a long time and on a large body surface area, they can have local and sometimes systemic side effects. The effectiveness of calcineurin inhibitors applied topically is the same as that of steroids applied topically. Because they do not affect endothelial cells or skin fibroblasts, they are especially helpful for children and facial lesions that avoid telangiectasias and atrophy. Most ACLE and CLE sores answer well to treatment, yet just minor impacts are noted in sores of Subacute CLE and tumid lupus. R-salbutamol is an agonist of the 2 adrenergic receptor that prevents the production of IL-2 and IFN-. Scaling/hypertrophy, pain, itching, and ulceration in CLE patients have significantly improved with twice-daily application of a 0.5 percent cream [6].

Antimalarial

Antimalarial were the oral first-line therapy in cutaneous lupus, introduced in 1953. Hydroxychloroquine, chloroquine, and quinacrine are the three drugs that belong to this class. The first case series of chloroquine sulfate in CLE was published in the British Medical Journal in 1955 by Lewis. Mechanism of action is proposed to be immunomodulation effects with influencing antigen presentation, stabilizing lysosomes, and suppression of toll-like receptor signaling, and reducing plasmacytoid dendritic cell production of IFN by preventing nucleic acids from acting on toll-like receptors. Dosage recommendations are as follows: hydroxychloroquine, 6.0–6.5 mg/kg of ideal body weight; chloroquine, 3.5–4 mg/kg of ideal body weight; quinacrine, 100 mg/day. For low-weight patients, use of actual weight for dosage is recommended. Adverse

effects include nausea, vomiting, irreversible retinopathy, and yellow discoloration of the skin/mucous membranes. Response rates of 75–95% are seen [7].

Methotrexate

Methotrexate, which was first utilized in 1965, is viewed as a second-line treatment, particularly in ACLE and CLE. In addition, it is utilized in headstrong antimalarial wound care and as a CS-saving specialist. The component of activity relies on activity on adenosine, a purine nucleoside with significant mitigating properties. Subsequently, CD4+ Immune system microorganisms go through apoptosis. Methotrexate significantly reduced autoantibodies in lupus patients compared to the control group in a study. Orally, intravenously, or subcutaneously, the recommended dosage is between 7.5 and 25 mg once per week. Gastrointestinal complaints are among the ominous effects, which can be eased up by taking folic destructive either already or following taking methotrexate; hepatotoxicity; nephrotoxicity; as well as the inhibition of bone marrow. Interstitial pneumonitis can be lethal, in spite of the way that it is very phenomenal [8].

Retinoids

According to the recommendations of the American Academy of Dermatology, retinoids, which were introduced in 1983, are also regarded as second-line treatments for CLE. Retinoids have been particularly useful in hypertrophic/verruccous CLE, where the sores of discoid lupus have shown a stronger response. Retinoids repress the statement of the proinflammatory cytokines, MRP-8, and IFN- γ . They are additionally calming and antiproliferative and they standardize keratinocyte separation in the epithelium. Suggested doses of acitretin and isotretinoin are 0.2-1.0 mg/kg/body weight every day. Cheilitis, hair loss, and an increase in triglyceride levels are some of the adverse effects. Due to the prevalence of birth defects, isotretinoin contraception for one month and acitretin contraception for three years are strictly enforced. Additionally, drug-induced hepatitis must be monitored. 13 patients treated with acitretin and 15 patients treated with hydroxychloroquine experienced improvements in the one and only randomized, double-blind, multicenter trial comparing the efficacy of acitretin and hydroxychloroquine in 28 and 30 CLE patients, respectively [9, 10].

CONCLUSION

Topical or intralesional therapy may be effective for the initial single CLE lesion. For patients with serious, safe, or repetitive injuries, it is vital to forestall scarring and post incendiary changes. Only open-label, nonrandomized studies have been published in the literature at this time, and very few randomized clinical trials have compared the various long-term medications. As a result, expert advice and observational studies constitute the majority of our patients' recommendations at this time.

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