



Herpes zoster infection after topical steroid use in the setting of tumid lupus erythematosus

Rachel Powell, MD,^a Grace Hile, BS,^b Lori Lowe, MD,^{c,d} and J. Michelle Kahlenberg, MD, PhD^c
Ann Arbor, Michigan, and Springfield, Illinois

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INTRODUCTION

Topical corticosteroids are commonly used in the treatment of a wide range of skin manifestations of systemic lupus erythematosus (SLE). Patients with SLE are also known to have an increased risk of herpes zoster (HZ). Here we present a rare case of recurrent HZ induced by topical corticosteroid use for the treatment of tumid lupus in the background of systemic lupus. We also review the literature on topical corticosteroid use and varicella zoster infection. For the literature review, search terms both as keywords and subject headings, included *topical corticosteroids*, *topical steroids*, *corticosteroids* AND *systemic lupus erythematosus*, *lupus*, *cutaneous lupus* AND *herpes zoster*, *herpes virus*, *varicella zoster*, *shingles*. HZ is a rare complication of topical corticosteroid use. Providers should use caution in the treatment of cutaneous lupus erythematosus (CLE) with topical steroids, as these patients are at an increased risk for HZ infection.

CASE PRESENTATION

A 50-year-old man with a 4-year history of SLE, diagnosed by fulfilling 1997 American College of Rheumatology criteria¹ (antinuclear antibody positive at 1:1280, positive double-stranded DNA, serositis, thrombocytopenia) presented with a 2- to 3-week history of an enlarging erythematous plaque on his right cheek after acute sun exposure (Fig 1, A).

Abbreviations used:

CLE: cutaneous lupus erythematosus
HZ: herpes zoster
SLE: systemic lupus erythematosus
TLE: tumid lupus erythematosus
VZV: varicella zoster virus

The lesion was asymptomatic, and the patient denied antecedent trauma/arthropod bite or similar eruption in the past. He typically avoided sun exposure, was compliant with sunscreen use, and had not noted any recent malar rash. He had excellent control of his SLE on hydroxychloroquine monotherapy for the last 3 years. Review of systems was negative for fevers, malaise, oral sores, or arthralgia. Physical examination found a unilateral 2.5- × 1.3-cm, erythematous-to-violaceous, edematous plaque without surface change on the right inferiolateral orbit encroaching on right lower eyelid (Fig 1, A). A 3-mm punch biopsy was performed that showed a superficial and deep, perivascular and focally interstitial, lymphocytic infiltrate with increased dermal mucin and a near-normal epidermis. The histologic features were interpreted as most consistent with tumid lupus erythematosus (TLE) (Fig 1, B). He was prescribed desoximetasone 0.05% cream to be applied topically twice daily. After 2 weeks of using the topical steroid cream, tingling developed on the right side of his face. The steroid

From the Departments of Internal Medicine,^a Dermatology,^c Pathology,^d and Internal Medicine, Division of Rheumatology,^e University of Michigan, and Southern Illinois University School of Medicine.^b

Authors Powell and Hile contributed equally to this article.

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Correspondence to: J. Michelle Kahlenberg, MD, PhD, 5570A MSRB II 1150 E Medical Center Drive, Ann Arbor, MI 48109-5678. E-mail: mkahlenb@med.umich.edu.

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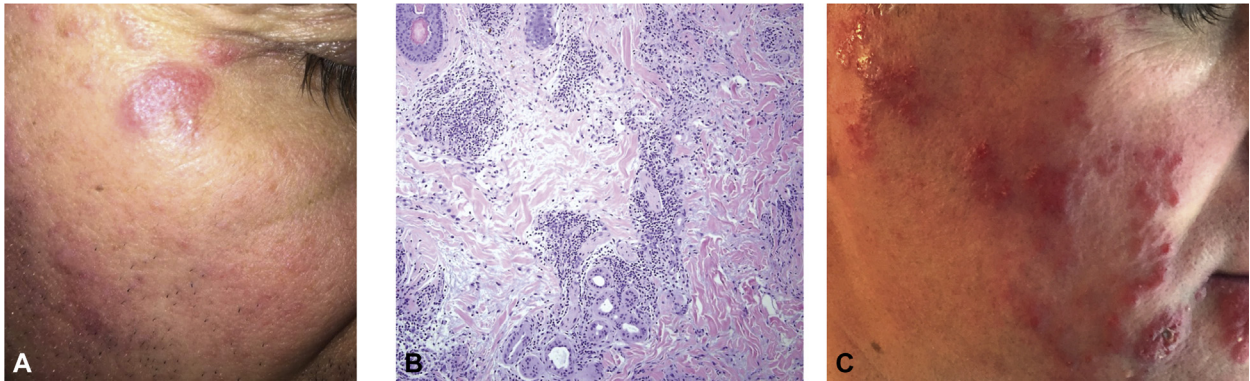


Fig 1. **A**, TLE near right lateral eye consisting of erythematous and edematous plaques. **B**, Skin biopsy of tumid lupus lesion shows a superficial and deep perivascular and focally interstitial lymphocytic infiltrate. The areas of pallor correspond to increased dermal mucin. **C**, Varicella zoster infection in the distribution of cranial nerve V³. (**B**, Hematoxylin-eosin stain; original magnification: ×100.)

cream was discontinued; however, the paresthesia worsened, and an erythematous rash with vesiculopapules developed on his right cheek and buccal mucosa with sharp demarcation at the midline of his face (Fig 1, C). Both facial and buccal lesions were positive for varicella zoster virus (VZV) by polymerase chain reaction. The eruption improved with a 7-day course of valacyclovir. Review of the original biopsy specimen did not show any histologic evidence of VZV infection, and immunohistochemical staining for VZV was negative.

One month after resolution of the herpes zoster, he experienced a recurrence of his tumid lupus. He was started on suppressive valacyclovir, 1 g daily, in preparation for intralesional triamcinolone injection. One dose was given before triamcinolone injection. One week after he received the intralesional steroids, the vesicular rash reoccurred and subsequently resolved after valacyclovir dose was increased to 3 g daily.

DISCUSSION

Herpes zoster infection is caused by reactivation of VZV and typically presents as a unilateral vesicular and painful rash in a dermatomal distribution. Patients with CLE have an increased risk for HZ with a reported incidence of 29.4 cases per 1000 person-years.² SLE patients are also at high risk for HZ, with an estimated 2 to 10-fold increased incidence compared with the general population.^{3,4} In fact, SLE is a stronger risk factor for HZ development than other autoimmune and noninflammatory musculoskeletal disorders, including rheumatoid arthritis.⁵⁻⁷ This increased risk is independent of immunosuppression, although systemic immunosuppressive therapy

is an additional risk factor for the development of HZ.⁵

TLE is a rare form of CLE. Clinically, TLE appears as erythematous papules and plaques and is characterized histologically by a superficial and deep perivascular lymphocytic infiltrate and increased dermal mucin. TLE differs from conventional discoid or subacute CLE by having no epidermal interface involvement. Topical corticosteroids, alone or in combination with systemic immune modulating therapy such as hydroxychloroquine, are the mainstay of treatment for CLE and TLE.⁸

Wolf's isotopic response, a phenomenon in which a new skin disorder occurs at the site of a previously healed lesion, could be considered an explanation for this case. However, most reports for this response start with HZ or herpes simplex followed by a secondary lesion, such as skin cancer.⁹ This patient had HZ reactivation subsequent to a tumid lupus lesion; no case reports exist describing this sequence for Wolf's isotopic response.

Although systemic corticosteroids are known to increase HZ risk, especially in patients with SLE, little is known about the risk of topical medications in reactivation of VZV. One case report linked the application of topical tacrolimus to the onset of zoster, suggesting topical preparations may be of concern.¹⁰ However, the risk of topical corticosteroids in contributing to reactivation of VZV is not known.

Corticosteroids decrease cell-mediated immunity, thus raising concern about the reactivation of latent VZV in terminal ganglia. Smith et al¹¹ argue that relatively large doses of corticosteroids are required for this to occur. Others have noted that reactivation from topical steroid preparations is unlikely, citing

evidence of few side effects from use of oral injections of triamcinolone in patients with herpes labialis and stomatitis.¹² Topical corticosteroids are also commonly used and generally considered safe and effective treatment for the cutaneous symptoms of herpes zoster and herpes simplex infections. However, our case suggests that in SLE patients, who are at an increased risk of HZ even without immunosuppressive therapy, topical steroids should be used with caution. Further, as vaccination for herpes zoster can decrease rates of virus reactivation in immunosuppressed people,¹³ administration of the vaccine before topical steroid treatment may be warranted in SLE patients.

This case presents a nonimmunosuppressed SLE patient who developed HZ in the distribution of topical steroid use followed by HZ recurrence while on suppressive valacyclovir therapy after an intradermal steroid injection. Topical and intradermal steroids should be considered potential risk factors for HZ reactivation in SLE patients.

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