A progressive scalp lesion

Our patient's case of "ringworm" on the scalp had been treated with 6 weeks of griseofulvin. But 13 years later, she wanted to know why the lesions had never gone away.

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A 47-YEAR-OLD AFRICAN AMERICAN WOMAN sought care at our clinic for multiple progressive scalp lesions. She said that she first noticed the lesions 13 years ago when her children were diagnosed with ringworm on the scalp. At that time, her physician thought that she, too, had tinea capitis, and she was treated with 6 weeks of griseofulvin. The lesions persisted, however.

She told us that the lesions were nonpruritic and that she didn't have any other symptoms. The patient did not have a history of trauma or exposure of chemicals to the scalp, and she was not taking any prescription or over-the-counter medications.

Examination of her scalp revealed scattered irregularly shaped, nontender lesions that were centrally hypopigmented and peripherally hyperpigmented. She also had scarring and hair loss (**FIGURE 1**). She had no other lesions on her body.

- WHAT IS YOUR DIAGNOSIS?
- HOW WOULD YOU MANAGE THIS CONDITION?

FIGURE 1
Irregularly shaped scalp lesions

PHOTOS COURTESY OF: AHUNNA AHIARAH, MD

Our 47-year-old patient had multiple scattered scalp lesions that were nontender and centrally hypopigmented. Scarring, alopecia, and surrounding areas of hyperpigmentation were also visible.

Diagnosis: Discoid lupus erythematosus

A punch biopsy and tissue pathology confirmed that our patient had discoid lupus erythematosus (DLE).

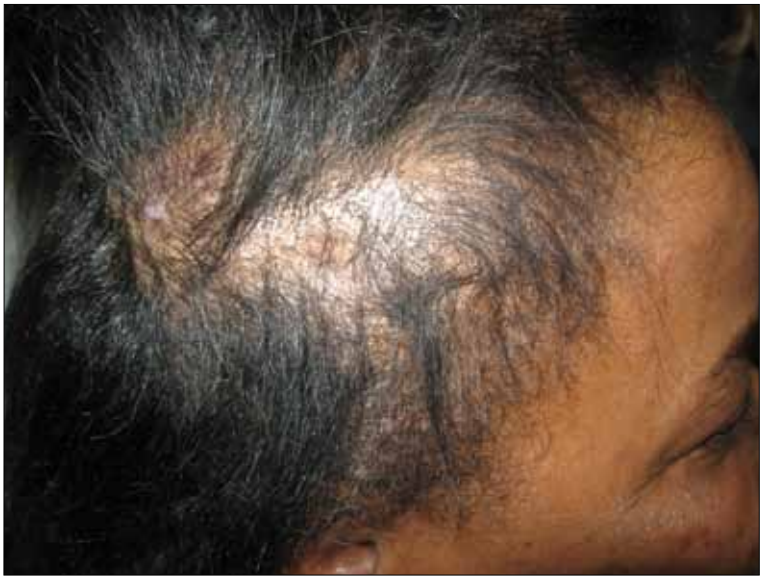
DLE is a common type of cutaneous lupus that is chronic and is typically associated with atrophy and scarring of the skin. The primary discoid lesion is a discrete erythematous papule or plaque with adherent scaling, follicular plugging, atrophic scarring, central hypopigmentation, and hyperpigmented borders.

DLE is a dermatosis that is localized in 80% of patients and occurs mainly on sun-exposed areas of the skin, such as the scalp, face, and ears. In 20% of cases it occurs on the extremities and upper trunk. Women are affected more than men, and it can affect any age group, although it is more common in individuals between the ages of 20 and 40.[1]

The etiology of DLE is unknown. What we do know is that less than 5% of patients with DLE eventually end up with systemic lupus erythematosus (SLE), while up to 25% of patients with SLE go on to develop chronic discoid lesions.[2]

Abnormalities in serology are not common in DLE. About 20% of patients with DLE show positive antinuclear antibody titers when tested. This is the case in the presence of widespread disease more so than in localized DLE. The presence of antinative (antidouble-stranded) or anti-Smith antibodies is usually suggestive of systemic symptoms and occurs in 5% of cases.[3]

FIGURE 2
A visible improvement



After 4 months of treatment with hydroxychloroquine, the patient's scalp lesions improved and there was evidence of hair growth.

Several conditions mimic DLE

The differential diagnosis for DLE includes the following:

Lichen plano pilaris. This condition has a predilection to the crown of the head and the frontal central region of the scalp; it is also associated with bilateral eyebrow hair loss. Patients may complain of pruritus, localized tenderness, and a burning sensation. The etiology is unknown; it is most often seen in middle-aged women with a chronic, progressive clinical course.[4]

Alopecia areata. Patients suddenly lose hair in patches. The hair grows back, and then falls out again. This asymptomatic condition is a tissue-restricted autoimmune disease of the hair follicle that most commonly occurs among children and young adults.[5]

Dissecting scalp cellulitis. This is a suppurating and cicatrizing disease of the scalp of unknown etiology. It typically affects the scalp vertex and occipital region, and is most common among young black men. The erythematous papules eventually discharge seropurulent material and form underlying intercommunicating sinuses with eventual scarring.[6] Patients are likely to complain of pain and pruritus.

Tinea capitis. As noted earlier, this is commonly referred to as "ringworm" and is of a fungal, infectious etiology that typically affects children. Permanent scarring and alopecia are common in affected areas, and patients complain of itching and burning.

Diagnosis can be made on clinical grounds

While a clinical diagnosis of DLE can be made, a tissue biopsy of a new inflamed site is confirmatory. Histopathologic findings show hyperkeratosis, follicular plugging, thickening of the basement membrane, atrophic epidermis, and dermal perifollicular and periappendageal lymphocytic inflammatory infiltrate.

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Direct immunofluorescence of lesions shows granular immunoglobulin and complement deposition at the dermal-epidermal junction.[7,8]

Early treatment is key

Early treatment may be helpful in preventing permanent scarring. Therapeutic options commonly used are oral antimalarials (strength of recommendation [SOR]: **A**) and topical (SOR: **A**) or intralesional (SOR: **B**) corticosteroids.[9] Other topical agents, such as calcineurin inhibitors, retinoids, and imiquimod, have been found to be helpful in some cases.

Alternative systemic agents that appear to be useful include methotrexate, azathioprine, thalidomide, dapsone, and mycophenolate mofetil. Patients should be advised to avoid the sun and wear broad-spectrum sunscreen.[7,10]

Finally, the patient sees some improvement

We discussed the risks and benefits of the various treatments, and our patient elected to start hydroxychloroquine (Plaquenil), 200 to 400 mg/day orally (not to exceed 6.5 mg/kg per day). We referred her for a baseline ophthalmological exam and stressed that she needed a repeat exam every 6 to 12 months while she remained on the hydroxychloroquine. We also referred her to a rheumatologist.

After 4 months of treatment, she showed some improvement (**FIGURE 2**), with no side effects from the medication. The patient was subsequently lost to follow-up. JFP

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Strength of recommendation (SOR)

- **A** Good-quality patient-oriented evidence
- **B** Inconsistent or limited-quality patient-oriented evidence
- **C** Consensus, usual practice, opinion, disease-oriented evidence, case series

Treatment options include oral antimalarials and topical or intralesional corticosteroids.

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