

Sapna M. Gupta, MD
Jeffrey M. Weinberg, MD

CASE HISTORY

A 45-year-old woman presents to a dermatology clinic with a scalp lesion that has been increasing in size over the past 3 years. Physical examination demonstrates a hypopigmented, atrophic plaque with telangiectasia, depressed scarring, and alopecia (Figure 1). The lesion is 4 cm long. The patient denies pain and pruritus at the site of the lesion; systemic involvement is not evident. The patient has not previously sought medical attention.

WHAT IS YOUR DIAGNOSIS?

- A) Tinea capitis
- B) Discoid lupus erythematosus
- C) Sarcoidosis
- D) Lichen planus

WHAT IS THE APPROPRIATE TREATMENT?

- A) Prednisone
- B) Surgery
- C) Antifungals
- D) Antimalarials

ANSWERS

The correct answers are discoid lupus erythematosus (B) and antimalarials (D).

DISCUSSION

Discoid lupus erythematosus (DLE) is a chronic, indolent skin disease characterized by sharply margined, raised, erythematous lesions that spread slowly while the centers of the lesions heal with atrophy and scarring.¹ DLE usually occurs on the face, ears, and scalp.² Although not considered to be a rare disease, the prevalence of DLE is unknown.³ Most patients diagnosed with DLE are adults between the ages of 25 and 45 years, and DLE affects women twice as often as men. The cause of DLE is unknown, but various factors, (eg, sunlight, cold, physical trauma, menses) have been shown to induce the appearance of lesions in predisposed individuals. The development of squamous cell carcinoma is a rare complication of long-standing DLE lesions.⁴

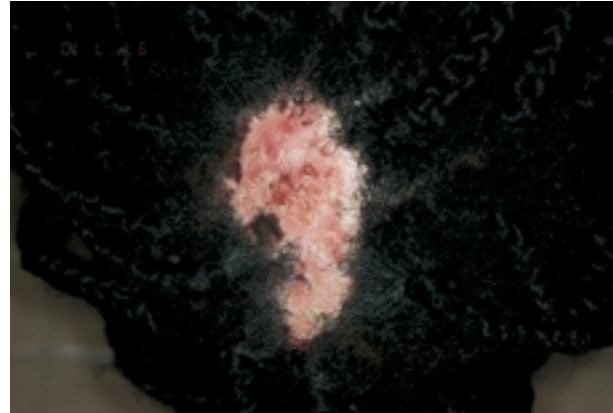


Figure 1. Hypopigmented, atrophic plaque of the scalp with telangiectasia, depressed scarring, and alopecia.

The lack of systemic symptoms (eg, arthritis, gastrointestinal symptoms, central nervous system symptoms), the absence of significant titers of antinuclear antibodies, and the normal serum levels of complement suggest that the pathogenesis of DLE differs from that of systemic lupus erythematosus. During the early nineteenth century, the term *lupus* was used to characterize a destructive disease consisting of spreading ulceration of the face. In 1851, Cazenave classified DLE as a variety of lupus because of the erythematous and atrophic nature of the disease. In 1872, Kaposi separated the chronic discoid form from the acute systemic form.

Diagnosis

Biopsy and immunofluorescence are necessary to establish a definitive diagnosis of DLE. Biopsy specimens of DLE lesions usually demonstrate follicular plugging, thickening of the epidermal basement membrane, a lymphocytic infiltrate in the dermis, and the presence of mucin.⁵ Direct immunofluorescence reveals immunoglobulin deposits at the dermoepidermal junction. A search for abnormal laboratory values is generally unrewarding. An antinuclear antibody is positive in approximately 5% of DLE cases.

Dr. Gupta is a Resident, Department of Dermatology, New York Medical College, New York, NY, and Dr. Weinberg is Assistant Professor of Dermatology, New York Medical College, and Clinical Director, Department of Dermatology, Metropolitan Hospital Center, New York.

Differential Diagnosis

Other disorders also cause raised erythematous lesions with central atrophy, telangiectasia, and scarring resulting in permanent alopecia. These entities include chronic suppurative folliculitis, tinea capitis caused by *Trichophyton tonsurans*, lichen planus of the scalp, and sarcoidosis. Pseudopelade of Brocq is a rare form of scarring alopecia in which destruction of hair follicles produces multiple, round cicatricial patches of hair loss of varying sizes. Pseudopelade generally produces a more scattered and patchy appearance than the well-defined plaques of DLE, and direct immunofluorescence is negative.⁶

Treatment

Active DLE lesions must be treated aggressively to prevent scarring. Topical corticosteroids can be used; however, the best treatment is local injection of corticosteroids into the lesion. If no response occurs, an antimalarial agent may be used; antimalarial drugs should only be used in cases of active disease. Chloroquine (250 mg) and hydroxychloroquine (200 mg) are given once daily or twice daily for 3 to 4 weeks and then once daily. A response is usually seen after 4 to 8 weeks. Relapses are frequent even after antimalarial treatment, and long-term therapy is frequently necessary. Patients should be observed for possible side effects, especially ocular toxicity.⁷ Avoidance of exacerbating factors, including sunlight, wind, cold, and physical trauma, is also advisable.

SUMMARY

DLE is a disorder with a unique presentation that may mimic other entities. Biopsy and immunofluorescence may be necessary for definitive diagnosis. Aggressive treatment is suggested to prevent scarring. Progression to systemic disease is rare, ranging from 1% to 5%. It is important to reassure DLE patients that the disease is local and self-limited. **HP**

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