

Case Report

Recurrent erosive-crusted ear lesions in a middle-aged woman: a case of discoid lupus erythematosus

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ABSTRACT

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Keywords: Discoid lupus erythematosus Cutaneous lupus erythematosus Autoimmune skin disorders Discoid lupus erythematosus (DLE) is a chronic autoimmune skin condition characterized by erythematous scaly plaques, often resulting in scarring and pigmentary changes. It primarily affects sun-exposed areas and is more common in women aged 20-40. Although predominantly a skin condition, it can progress to systemic lupus erythematosus, warranting long-term monitoring. In this article, we e present the case of a 40-year-old woman with an 8-year history of recurrent erosive and crusted lesions on both earlobes, extending to the helix, with local atrophy. Biopsy revealed DLE. The patient responded well to topical corticosteroids, photoprotection, and remains under dermatological follow-up with no active lesions. This case underscores the importance of considering DLE in patients with chronic, scaly ear lesions. A tailored treatment approach, including topical therapy and photoprotection, can effectively manage localized disease while minimizing systemic exposure. Regular follow-up is essential to monitor for recurrence and systemic involvement.

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Lesiones erosivo-costrosas recurrentes en la orejas en una mujer de mediana edad: un caso de lupus eritematoso discoide

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RESUMEN

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Palabras clave: Lupus eritematoso discoide Lupus eritematoso cutáneo Trastornos autoinmunes de la piel El lupus eritematoso discoide (LED) es una enfermedad autoinmune crónica de la piel caracterizada por placas escamosas eritematosas que, a menudo, resultan en cicatrices y cambios pigmentarios. Afecta principalmente áreas expuestas al sol y es más común en mujeres de 20 a 40 años. Aunque es predominantemente una afección cutánea, puede progresar a lupus eritematoso sistémico, lo que requiere un seguimiento a largo plazo. En este artículo, presentamos el caso de una mujer de 40 años con una historia de 8 años de lesiones erosivas y costrosas recurrentes en ambos lóbulos de las orejas, que se extienden hasta el hélix, con atrofia local. La biopsia reveló LED. La paciente respondió bien a los corticosteroides tópicos y a la fotoprotección y, actualmente, se encuentra en seguimiento dermatológico sin lesiones activas. Este caso resalta la importancia de considerar el LED en pacientes con lesiones escamosas crónicas en las orejas. Un enfoque de tratamiento personalizado, que incluya terapia tópica y fotoprotección, puede controlar eficazmente la enfermedad localizada mientras se minimiza la exposición sistémica. El seguimiento regular es fundamental para detectar recurrencias y evaluar una posible progresión sistémica.

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1. INTRODUCTION

Discoid lupus erythematosus (DLE) is the most common type of chronic cutaneous lupus, an autoimmune skin condition on the lupus erythematosus spectrum. It is characterized by erythematous scaly plaques that often result in scarring and pigmentary changes [1, 2]. DLE accounts for approximately 50% to 85% of all chronic cutaneous lupus erythematosus cases [3]. The development of cutaneous lupus erythematosus results from a combination of genetic predisposition and environmental triggers. Key environmental factors include ultraviolet radiation, certain medications, cigarette smoking, and possibly viral infections. These triggers initiate an inflammatory response and contribute to an increased risk of developing DLE [1, 4].

The lesions predominantly occur on sun-exposed areas such as the face, scalp, ears, and upper torso. Affecting primarily women aged 20-40, DLE can lead to significant disfigurement, alopecia, and secondary infections. Although primarily a skin condition, 5-10% of DLE cases can progress to systemic lupus erythematosus, necessitating long-term monitoring for both systemic involvement and the risk of malignant transformation in chronic lesions. Rarely, squamous cell carcinoma can develop in longstanding discoid lesions, with an incidence rate of 2 to 3 percent and typically a poor prognosis [1, 2].

First-line treatment for DLE includes photoprotection, topical or intralesional corticosteroids, and topical calcineurin inhibitors [5]. Patients with chronic lesions unresponsive to topical treatments or extensive disease involvement may require systemic therapy. Antimalarials, known for their immunotherapeutic properties, are considered first-line systemic therapy for CLE. Treatment can be discontinued once lesions are erythema-free or scaleless, indicating disease inactivity. Generally, prognosis is favorable with appropriate treatment, although relapses are common [3, 6].

Contrary to the common presentations of DLE on the face and scalp, this case is noteworthy for its exclusive involvement of the earlobes, an uncommon site that can often lead to diagnostic delays and misdiagnosis.

2. CASE REPORT

A 40-year-old female presented to her family doctor with an 8-year history of recurrent erosive-crusted lesions on both ear lobes, extending to the helix, accompanied by some local atrophy, as shown in Figure 1. The lesions occurred throughout the year, mainly during winter.



Figure 1: Marked dermatoheliosis and erythematous, erosive-crusty lesions of the earlobes extending to the helix, some local atrophy.

Although a histopathology image is not included, the skin biopsy revealed mild acanthosis with focal vacuolization of the basal layer. The superficial and deep dermis exhibited a multinodular perivascular infiltrate of mild to moderate density, predominantly composed of lymphomononuclear cells and numerous plasma cells. Additionally, the infiltrate was associated with vasodilation and moderate solar elastosis, indicative of chronic sun exposure. These histopathological findings, in correlation with clinical presentation, support the diagnosis of DLE. The autoimmune workup showed negative serum antinuclear antibody (ANA) testing and complement levels (C3 and C4) were within normal limits, findings that do not exclude cutaneous lupus erythematosus but suggest a localized, rather than systemic, disease process.

Patient was started on topical corticosteroid therapy along with strict photoprotection measures, including regular use of broad-spectrum sunscreens and sun avoidance strategies. This approach resulted in significant clinical improvement, with resolution of active lesions. At the most recent followup visit, the patient remained lesion-free, highlighting the effectiveness of targeted therapy and long-term photoprotection in the management and prognosis of DLE. Regular dermatological follow-up is ongoing to monitor for disease recurrence or potential progression to systemic lupus erythematosus.

3. DISCUSSION

This case underscores the importance of early recognition and appropriate management of DLE to mitigate potential complications and improve the patient's quality of life. Early intervention is essential to reduce the risk of scarring, atrophy and dyspigmentation, leading to significant morbidity rather than mortality. For many patients with long-standing DLE, particularly those with visible lesions on their face or scalp, the psychosocial impact of disfiguring lesions should not be underestimated [6, 7].

Given the presentation of recurrent erosive and crusted lesions on the earlobes, the differential diagnosis is difficult and includes several conditions, as shown in Table 1.

Table 1: Differential diagnosis of discoid lupus erythematosus			
Differential diagnosis	Age group	Clinical Presentation	Etiology & common triggers
Discoid lupus erythematosus	20-40 years, mostly women	Erythematous, scaly plaques leading to atrophy and scarring, typically on sun- exposed areas (face, scalp).	Autoimmune disorder, often triggered by ultraviolet (UV) exposure, stress, infections.
Perniosis	All ages	Painful, violaceous plaques or nodules on cold-exposed areas (ears, fingers, toes). May ulcerate in severe cases.	Cold exposure in predisposed individuals; poor circulation
Chronic Eczematous Dermatitis	All ages, more in middle age	Pruritic, erythematous, scaling patches with lichenification. Can affect ears due to contact allergens.	Allergic or irritant dermatitis, triggered by allergens, irritants, stress.
Sarcoidosis	20-40 years	Reddish-brown plaques or papules, often involving the face, nose, and ears; may have nodular features.	Unknown, possibly immune dysregulation; genetic predisposition.
Actinic keratosis	40+ years	Pre-malignant rough, scaly lesions in sun- exposed areas.	Chronic UV exposure.
Basal cell carcinoma	50+ years	Pearly, flesh-colored nodules with telangiectasia may ulcerate. Occurs in sun- exposed areas (ears, face).	Chronic UV exposure.
Psoriasis	20-40 years	Well-demarcated erythematous plaques with silvery scales; often affects scalp, elbows, knee, but can affect ears.	Chronic immune-mediated disorder, triggered by stress, infections, trauma.
Cutaneous leishmaniasis	20-40 years	Ulcerated, crusted lesions, typically on exposed skin after sandfly bites.	Protozoan infection (Leishmania spp.) transmitted by sandfly bite

Moreover, this case enhances the necessity for long-term follow-up due to the potential risk of progression to systemic disease and the risk of malignant transformation in chronic lesions. A significant challenge in managing chronic DLE is prolonged disease activity, which increases the risk of squamous cell carcinoma due to persistent skin inflammation. This risk is particularly higher in patients with untreated or poorly controlled disease for an extended period [5, 8].

Although systemic antimalarial therapy, such as hydroxychloroquine, is commonly indicated for widespread or refractory cases of chronic DLE, the decision to withhold it in this case was based on the mild nature of the lesions, their limited distribution, and the patient's significant positive response to topical therapy and photoprotection. The absence of systemic lupus erythematosus features, coupled with a normal autoimmune workup, further supported the decision to avoid systemic treatment at this stage [3, 5, 6].

The prognosis for DLE is generally favorable with appropriate management, particularly when early intervention strategies—such as photoprotection and topical corticosteroids—are implemented [1, 3]. Given the chronic relapsing-remitting nature of DLE, which tends to flare during certain environmental triggers, such as sun exposure or stress, the patient remains under close follow-up [9]. Close monitoring allows for early identification of relapses and timely adjustments in treatment to prevent disease progression.

This individualized approach emphasizes the importance of

tailoring treatment plans based on disease severity, distribution, and patient response to initial therapies. Such a strategy not only improves the likelihood of treatment success but also minimizes the potential for unnecessary systemic treatments, which may carry significant side effects.

4. CONFLICT OF INTERESTS

The authors have no conflict of interest to declare. The authors declared that this study has received no financial support.

5. REFERENCES

1. McDaniel B, Sukumaran S, Koritala T, Tanner LS. Discoid Lupus Erythematosus. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.

 Tebbe B. Clinical course and prognosis of cutaneous lupus erythematosus. Clin Dermatol. 2004 Mar-;22(2):121-4. doi: 10.1016/j.clindermatol.2003.12.018.

3. Panjwani S. Early diagnosis and treatment of discoid lupus erythematosus. J Am Board Fam Med. 2009;22(2):206-13. doi: 10.3122/jabfm.2009.02.080075.

4. Miot HA, Bartoli Miot LD, Haddad GR. Association between discoid lupus erythematosus and cigarette smoking. Dermatology. 2005;211(2):118-22. doi: 10.1159/000086440.

5. Kuhn A, Aberer E, Bata-Csörgő Z, Caproni M, Dreher A, Frances C, et al. S2k guideline for treatment of cutaneous lupus erythematosus - guided by the European Dermatology Forum (EDF) in cooperation with the European Academy of Dermatology and Venereology (EADV). J Eur Acad Dermatol Venereol. 2017;31(3):389-404. doi: 10.1111/jdv.14053.

6. Company-Quiroga J, Alique-García S, Romero-Maté A. Current Insights Into The Management Of Discoid Lupus Erythematosus. Clin Cosmet Investig Dermatol. 2019;12:721-32. doi: 10.2147/CCID.S184824.

7. Drenkard C, Theis KA, Daugherty TT, Helmick CG, Dunlop-Thomas C, Bao G, et al. Depression, stigma and social isolation: the psychosocial trifecta of primary chronic cutaneous lupus erythematosus, a cross-sectional and path

analysis. Lupus Sci Med. 2022;9(1):e000697. doi: 10.1136/lupus-2022-000697.

8. Chong BF, Song J, Olsen NJ. Determining risk factors for developing systemic lupus erythematosus in patients with discoid lupus erythematosus. Br J Dermatol. 2012;166(1):29-35. doi: 10.1111/j.1365-2133.2011.10610.x.

9. Kuhn A, Ruland V, Bonsmann G. Photosensitivity, phototesting, and photoprotection in cutaneous lupus erythematosus. Lupus. 2010;19(9):1036-46. doi: 10.1177/0961203310370344.