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Authors

Halim, P Anthony
Rizky, Luddwi Achmad
Fitri, Eyleney Meisyah
et al.

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Case Presentation

Unilateral discoid lupus erythematosus: two atypical cases with distinct presentations

P Anthony Halim, MD¹, Luddwi Achmad Rizky, MD¹, Eylene Meisyah Fitri, MD¹, Endi Novianto, MD, PhD¹, Maria Angela Putri Maharani, MD², Sondang P Sirait, MD, PhD¹, Windy Keumala Budianti, MD, PhD^{1a}

¹ Department of Dermatology and Venereology, Faculty of Medicine Universitas Indonesia, Dr Cipto Mangunkusumo Hospital, Jakarta, Indonesia, ² Department of Anatomical Pathology, Faculty of Medicine Universitas Indonesia, Dr Cipto Mangunkusumo Hospital, Jakarta, Indonesia

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Abstract

Discoid lupus erythematosus can present in various atypical forms, posing significant diagnostic challenges as it can mimic other infectious or neoplastic dermatoses. Herein, we detail two cases of strictly unilateral, multifocal discoid lupus erythematosus and compare them with previously reported cases. This report contributes to the limited literature on this unusual presentation, particularly in individuals with skin of color.

Introduction

Cutaneous lupus erythematosus (CLE) represents a diverse group of autoimmune diseases with varied skin manifestations.¹ Specific CLE is traditionally classified into acute, subacute, and chronic forms, each linked to a different risk for developing systemic lupus erythematosus. Discoid lupus erythematosus (DLE) is a common subtype of chronic CLE, classically presenting as erythematous scaly plaques in photodistributed areas that later develop atrophic scarring and dyspigmentation.¹ DLE can manifest in a myriad of atypical forms,² posing significant diagnostic challenges as these can mimic various dermatoses. It is imperative to accurately diagnose and promptly treat DLE patients, as the damage it causes is often irreversible and can impair quality of life. DLE typically presents with bilaterally distributed lesions, and cases showing a preference for one body side are rare. Among these, linear DLE, characterized by unilateral lesions along the Blaschko lines, is well documented in the medical literature.³ Our cases, described herein, detail unusual presentations of strictly unilateral, non-Blaschkoid, yet multifocal distribution of DLE on the face.

Case Synopsis

Case 1

A 40-year-old, heavy-smoking man presented with an 8-year history of pruritic, red-purplish plaques on the left side of his face, with no history of preceding trauma. He reported frequent unprotected solar exposure as an outdoor worker. Further probing revealed no history of repeated unilateral solar exposure or prior trauma or dermatoses on his left face. His other medical and family histories were unremarkable. Previous topical treatment yielded no improvement. On examination, scaly erythema-violaceous plaques with atrophic scarring were observed on his left face (**Figure 1A-B**). Dermoscopy showed follicular keratotic plugs, white scales, and white structureless areas (**Figure 1C**). A head-to-toe physical examination did not reveal any other lesions in other body parts. Non-tender lymph node enlargement was palpated on the left submental and submandibular regions, with a previous fine-needle aspiration biopsy suggesting reactive lymphadenopathy. The initial differential diagnosis included DLE, lupus vulgaris, and mycosis fungoides.

Mycobacterial culture and polymerase chain reaction from skin biopsies were negative. Histopathological examination (**Figure 2A-C**) revealed features indicative of DLE, including follicular plugging, interface dermatitis, and basal membrane thickening. However, dense perifollicular lymphocytic infiltrates forming germinal center-like structures with some atypia raised the possibility of folliculotropic mycosis fungoides. Immunohistochemical analysis (**Figure 2D-F**) confirmed the lesion as non-neoplastic, characterized by mixed B and T-cell lymphoid aggregates with a proliferation rate within the expected range. Collectively, the clinicopathological findings supported a diagnosis of unilateral DLE.

^a Corresponding Author: Windy Keumala Budianti MD PhD, Department of Dermatology and Venereology, Faculty of Medicine Universitas Indonesia, Dr Cipto Mangunkusumo Hospital, 71st Diponegoro Street, Jakarta 10430, Indonesia, Tel: (+62) 021-31930373, Email: wkeumala@gmail.com

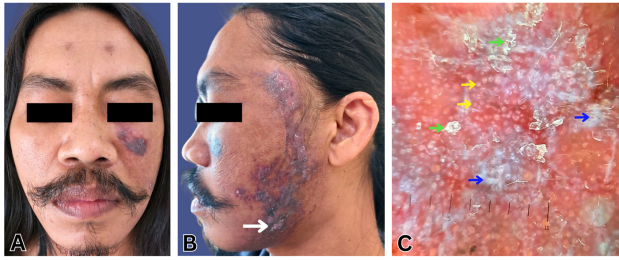


Figure 1. Case 1: Unilateral discoid lupus erythematosus. **A-B)** Scaly violaceous plaques with areas of atrophic scarring (white arrow) were noted on the left face. **C)** Dermoscopy demonstrated follicular plugging (yellow arrows), white scales (green arrows), and white structureless areas (blue arrows).

The patient did not meet the 2019 European League Against Rheumatism/American College of Rheumatology criteria for systemic lupus erythematosus, despite laboratory tests showing positive antinuclear antibodies (ANA) with a titer of 1:100 and decreased C3 level of 75mg/dl (reference range 90-180mg/dl). The patient was counseled to quit smoking and adhere to strict photoprotection. Treatment with 0.25% desoximetasone ointment for one month was followed by 1% pimecrolimus cream. After three months, a partial improvement was noted. Although systemic therapy with hydroxychloroquine was considered, the patient did not return for follow-up.

Case 2

A 51-year-old smoking man presented with a 15-year history of asymptomatic erythematous plaques on the left lower face. He denied any preceding trauma, skin conditions, or lesional bleeding. Although he disclosed history of extensive solar exposure as an outdoor worker, predominantly left-sided sun irradiation was denied. Examination revealed scaly erythematous plaques with atrophic scarring and violaceous borders on the left supralabial and mandibular regions, measuring 1.6x3.5cm and 3.9x2.8cm, respectively (Figure 3A-B). The supralabial lesion was verrucous and partly firm on palpation. Dermoscopy displayed white structureless areas, pigmentation, white scales, and telangiectasias, with more pronounced follicular plugging in the mandibular plaque (Figure 3C). A full-body skin examination confirmed the absence of additional lesions elsewhere. The initial differential included DLE, cutaneous mycobacterial infection, morphea, and cutaneous malignancies.

Mycobacterial culture and polymerase chain reaction from skin biopsies were negative. Histologically, both lesions showed findings of typical DLE, which included follicular plugs, interface dermatitis, periadnexal infiltrates, and thickened basement membrane with increased dermal mucin (Figure 4A-E). The supralabial lesion also exhibited hyperkeratosis and acanthosis, consistent with hypertrophic DLE. These findings established a diagnosis

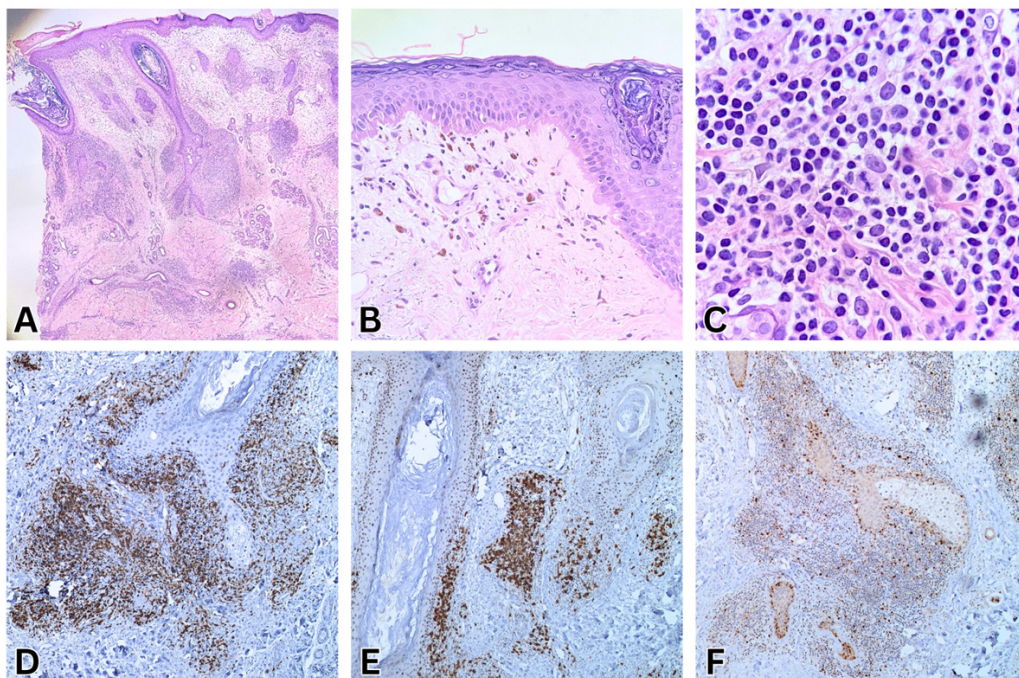


Figure 2. Case 1: Histopathological findings. **A)** Follicular plugging and germinal center (GC)-like perifollicular infiltrates (H&E, x40). **B)** Minimal vacuolar alteration, thickened basement membrane, sparse interface and perivascular lymphohistiocytic infiltrates, along with pigment incontinence, were noted (H&E, x400). **C)** The GC-like infiltrates primarily consisted of lymphocytes and histiocytes, with some of the former exhibiting atypical changes and mitotic figures (H&E, x1000). Immunohistochemical staining (x100) for **D)** CD3 and **E)** CD20 revealed mixed CD3+ T cells and CD20+ B cells infiltration, with a proliferation rate of 5-10% as indicated by **F)** Ki67 staining.

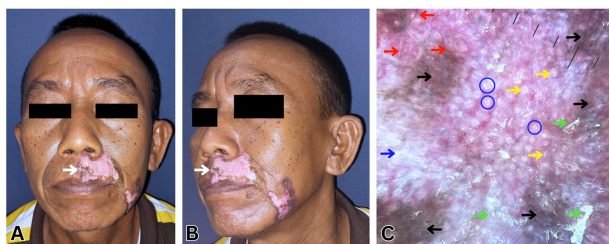


Figure 3. Case 2: Unilateral discoid lupus erythematosus with hypertrophic and classical lesions. **A-B)** Erythematous scaly plaques with violaceous to hyperpigmented borders and atrophic scarring were seen on the left supralabial and mandibular region. Note the more verrucous and indurated supralabial lesion (white arrow). **C)** Dermoscopy of the mandibular lesion revealed rosettes (blue circles), follicular plugs (yellow arrow), white scales (green arrow), pigmentation (black arrows), telangiectasias (red arrows), and white structureless areas (blue arrow).

of unilateral DLE with both hypertrophic and classical lesions.

Laboratory tests revealed a positive ANA at a titer of 1:320 and a weakly positive lupus anticoagulant, but the patient did not fulfill the European League Against Rheumatism/American College of Rheumatology classification criteria for systemic lupus erythematosus. Smoking cessation and strict photoprotection were recom-

mended. Treatment with 0.1% mometasone cream and additional intralesional injections of 10mg/ml triamcinolone acetonide for the hypertrophic plaque resulted in significant improvement after six weeks.

Case Discussion

The cases presented here exemplify two unusual presentations of multifocal DLE manifesting unilaterally. These two cases are morphologically distinct and clinically may resemble other infectious or neoplastic skin diseases. Dermoscopy is a helpful diagnostic tool for DLE, revealing characteristic signs such as keratotic plugs, scales, telangiectasias, and white structureless areas. However, for atypical cases, histopathological confirmation is paramount.

In the first case, the presence of lymphadenopathy initially suggested possible infection or neoplasm. However, infectious etiologies were ruled out based on histopathological and microbiological findings, whereas immunohistochemical analysis excluded neoplastic processes. The second case was notable for the coexistence of hypertrophic and classical DLE lesions in a unilateral pattern, an exceptionally rare presentation.

DLE cases with a strictly unilateral distribution have been documented, particularly those with linear lesions along the Blaschko lines, often classified as linear CLE.³ This subtype, usually seen in children, is thought to result from epidermal mosaicism.³ Unilateral eyelid DLE cases

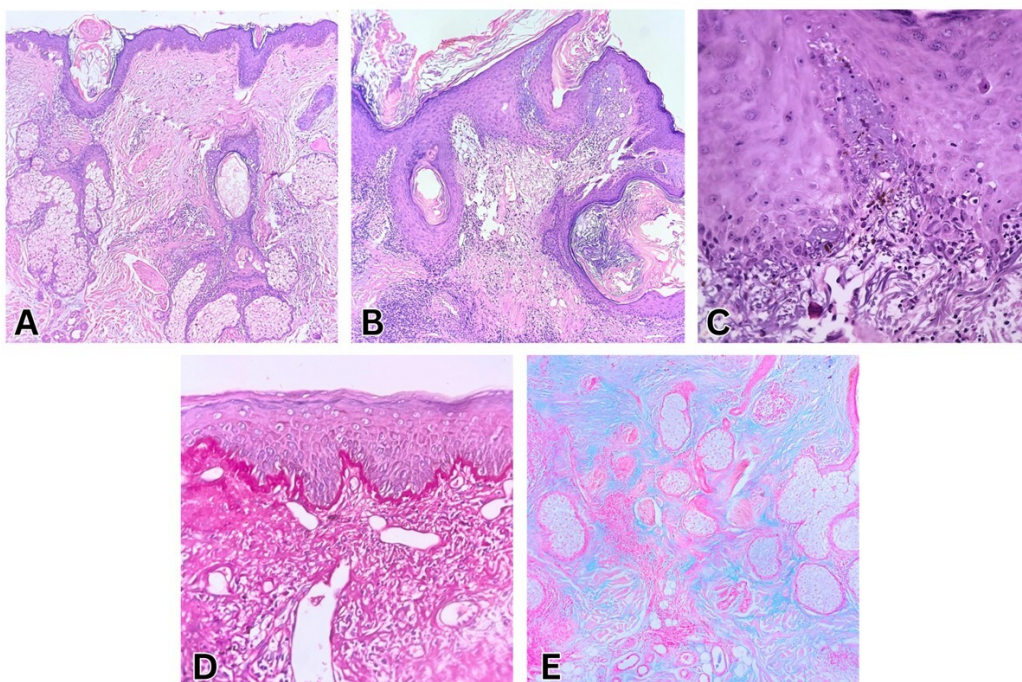


Figure 4. Case 2: Histopathological findings. **A)** Biopsy of the mandibular lesion demonstrated keratotic follicular plugging along with features of interface and periadnexal lymphohistiocytic infiltration (H&E, x100). **B)** The supralabial specimen showed more prominent follicular plugging, in addition to hyperkeratosis and irregular acanthosis (H&E, x100). **C)** Under higher magnification, dyskeratotic cells, vacuolar changes, lymphohistiocytic infiltrates along the dermo-epidermal junction, and pigment incontinence were noted (H&E, x400). Additional histochemical staining highlighted **D)** basal membrane thickening (Periodic acid-Schiff, x400) and **E)** dermal mucin deposition (Alcian blue, x100).

have also been reported, as detailed by Wang et al.⁴ However, our cases, along with several other unilateral DLE cases previously reported (Table 1), do not fit into either category.⁵⁻¹³ These cases uniformly showed facial involvement and were predominantly adults. Intriguingly, a male predominance (8 males to 5 females) was noted, deviating from the typical female dominance seen in lupus erythematosus.

Although no obvious cause for the unilateral presentation could be substantiated in our cases, some earlier reports attributed their cases to the Wolf's isotopic response, secondary to infection,^{5,8} trauma,^{6,7,9} or radiation.¹² In particular, Ruocco introduced the concept of an "immunocompromised cutaneous district",⁶ in which a prior insult alters the local cellular interactions within the skin, including the lymphatic circulation of immunologic cells and the neuroinflammatory pathways.¹⁴ These changes lead to the development of a vulnerable body area, or locus minoris resistentiae, which is more prone to various dermatosis, including sarcoidosis, leprosy, DLE, and neoplasms.^{9,14} Although a clear history of previous skin injury was not reported, the patients presented might have had a left-sided facial injury that they failed to recall, consistent with a report by Ruocco et al⁹ describing a case of unilateral DLE after a decades-long history of bruising and swelling without cuts or scars.

Another possible explanation for this peculiar unilateral presentation is asymmetric sun exposure, as considered by two previous authors.^{10,11} However, both our

patients were outdoor workers who reported uniform sun irradiation. In Indonesia, where driving is on the left, significant sun exposure would typically occur on the passenger side. This is unlikely to be a factor in our cases, as both patients primarily used motorcycles. Moreover, we observed similar signs of photoaging on both sides of the face in both patients, suggesting uniform chronic sun exposure.

Nevertheless, unilateral localized DLE cases generally show favorable responses to treatment and do not exhibit systemic disease at initial presentation, highlighting the importance of accurate diagnosis and prompt management. Typical first line treatments include potent topical and intralesional corticosteroids and antimalarials such as hydroxychloroquine.¹⁵

Conclusion

These two cases highlight a rare clinical presentation of discoid lupus erythematosus as strictly unilateral, multifocal facial lesions. Recognizing such atypical manifestations is essential for timely diagnosis and treatment to prevent irreversible skin damage.

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Potential conflicts of interest

The authors declare no conflicts of interest.

Table 1. Previous Reports of Non-Blaschkoid Unilateral Discoid Lupus Erythematosus

No	Author (Year)	Ref	Sex	Age	Lesion Side	Duration (months)	Treatment	Outcome	Remarks
1	Bardazzi et al. (2010)	5	F	38	Left	±18	PP, HCQ, TCS, topical tretinoin	Full resolution	On the location of healed cutaneous leishmaniasis
2	Baroni et al. (2010)	6	F	68	Right	±48	NR	NR	On the location of burn injury scar
3	Ko et al. (2011)	7	M	35	Right	±24	HCQ, TCS	Significant improvement	On the location of healed abrasion wound
4	Balevi et al. (2013)	8	F	62	Right	NR	NR	NR	On the location of healed herpes zoster
5	Ruocco et al. (2013)	9	M	71	Right	5	TCS	Full resolution	On the location of previous injury
6	Verma et al. (2017)	10	F	23	Left	4	PP, HCQ, SCS	Significant improvement	–
7	Yadav et al. (2018)	11	F	13	Right	6	PP, HCQ, TCS	Significant improvement	–
8	Carlesimo et al. (2020)	12	M	55	Right	NR	TCS	Partial improvement	On the location of prior radiotherapy
9	Liang et al. (2020)	13	M	33	Left	±36	Intralesional betamethasone	Significant improvement	Minimal response with topical and systemic therapy
10			M	38	Right	±24			
11			M	43	Right	±12			
12	Case 1		M	40	Left	±96	PP, TCS, TCI	Partial improvement	Lost to follow-up before HCQ initiation
13	Case 2		M	51	Left	±180	PP, TCS, intralesional triamcinolone (on hypertrophic lesion)	Significant improvement	Partially hypertrophic lesion

Note: Ref, reference; F, female; M, male; NR, not reported; HCQ, hydroxychloroquine; SCS, systemic corticosteroids; PP, photoprotection; TCI, topical calcineurin inhibitor; TCS, topical corticosteroid

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