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Cover Page Footnote

We would like to thank the patient and Department of Dermatology, Ospital ng Maynila Medical Center for facilitating the writing of this manuscript.

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Case Report

A 61-year-old Filipino man with lichen planus concomitant with cicatricial alopecia, mimicking discoid lupus erythematosus

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Abstract

Background: Lichen planus (LP) is an idiopathic inflammatory disease affecting the skin, mucous membranes, hair, and nails. Even though rare, LP may also present as cicatricial alopecia or a condition referred to as lichen planopilaris (LPP). On the other hand, lupus erythematosus (LE) is an autoimmune disorder with a possibility of systemic involvement. Classical discoid lupus erythematosus (DLE) is the most common form of LE and has a hallmark of scarring alopecia.
Case Illustration: A 61-year-old Filipino man presented with a 7-month history of persistent multiple erythematous hairless scarring plaques on the scalp with multiple erythematous–violaceous to hyperpigmented atrophic plaques on the face and distal upper extremities.
Discussion: The remarkable atrophic scarring alopecia on the scalp, along with the atrophic coin-shaped plaques on the face and extensor aspects of both forearms on this patient, brought DLE as the initial clinical impression. Besides cicatricial alopecia being a prevalent feature of DLE, the noticeable scarring alopecia on the scalp with the concomitant appearance of multiple atrophic skin lesions on sun-exposed areas supported this reasoning. Nevertheless, the skin punch biopsy of this patient showed numerous histopathological features of LP.
Conclusion: LP can present with several morphological cutaneous presentations, including atrophic LP, which may mimic cutaneous DLE. Even though LP is a non-scarring disease, a follicular variant of LP, LPP has a distinct clinical and histologic entity with associated scarring alopecia. The presence of atrophic cutaneous LP concomitant with scalp LPP may mimic DLE clinically.

Keywords*: cicatricial alopecia, discoid lupus erythematosus, lichen planus, scarring alopecia*

Background

Lichen planus (LP) is an idiopathic inflammatory skin disease that most commonly affects middle-aged adults. Classic LP is characteristically presented with “the four Ps,” which consist of purple, polygonal, pruritic, and papules. Few or multiple papules may coalesce, forming plaques.[1] However, besides the skin, LP may also affect the hair follicles, known as lichen planopilaris (LPP).[2,3] Classic LPP affects the vertex scalp and displays erythema plaques of individual keratotic follicular papules with associated scarring alopecia.[1] The prevalence of LPP is rare, which was 0.043% in the United States as of March 2022, with the average age at diagnosis being 62.4 years old and most prevalent in women.[4] On the other hand, discoid lupus erythematosus (DLE), the most common form of cutaneous lupus erythematosus, is more frequent, with an estimated incidence of 4.79 per 10,000 cases.[5] DLE typically presents as erythema to hyperpigmented plaques with atrophic central scarring, telangiectasia, and hypopigmentation.

The scalp is the most common site affected by DLE, occurs in about 30-50% of patients, and manifests as erythematous, atrophic, alopecic patches that continue to progress towards permanent hair loss and scarring. DLE is a common cause of scarring alopecia, accounting for

60% of all primary lymphocytic alopecias.[6] Classic LPP usually starts at the vertex and crown. It presents multifocal, coalescing alopecic patches with perifollicular erythema, follicular hyperkeratosis, and scaling. Compared to DLE, the areas of hair loss are often smaller in LPP, showing the irregularly shaped and interconnected, reticulated pattern of bald patches.[1] However, we presented an unusual case of a 61-year-old Filipino man with LP and multiple remarkable areas of atrophic scarring alopecia on the scalp, which made us clinically misdiagnose it as DLE initially.

Case Illustration

A 61-year-old Filipino man was referred with a clinical diagnosis of discoid LE to undergo a biopsy in our institution. The patient presented with a 7-month history of persistent, multiple erythematous to violaceous plaques on the extensor aspect of distal upper extremities, chest, and upper back, as well as multiple erythematous atrophic hairless plaques on the scalp. Lesions initially appeared on the distal area of the right arm and scalp, which gradually increased in size and number and have spread to the face, chest, upper back, and distal area of the left arm. The patient worked as a motorcycle courier, exposing him to sunlight every day.

The pruritic level of the lesions was described as 8/10 using the Peak Pruritus Numerical Rating Scale.[7] Scratching was done persistently, causing the lesion to develop erosions. Six months prior to the consult, due to the persistence of symptoms, the patient sought a consult with a private dermatologist and was prescribed clobetasol propionate 0.05% ointment for the extremities and an unrecalled scalp solution; both were applied twice daily inconsistently for four months and one month, respectively. The medications provided temporary relief of the pruritus. Another consult was done three months prior to the consult; the patient was prescribed cloxacillin 500 mg/tablet, which was taken inconsistently by the patient for three months, as well as clobetasol propionate 0.05% ointment, which he applied inconsistently for three months, and provided some relief of the pruritus. In the interim, the lesion was noted to be persisted. One day before the consult, due to the persistence of the lesions, the patient followed up with the previous private dermatologist and was advised to have a histopathology diagnostic test.

Dermatological examination revealed multiple, well-defined, erythematous–violaceous to hyperpigmented atrophic plaques, some topped with excoriations, erosions, adherent scales, and crusts, measuring from 0.5x0.5 cm to 2x2.5 cm located on the face, and both extensor aspects of forearms (Figure 1). The lesions on the scalp were noted as multiple erythematous to hypopigmented alopecic atrophic plaques topped with excoriations, erosions, and central scarring measuring from 1x1 cm to 2x3.5 cm located on the vertex and crown areas. Upon dermoscopic examination, no *Wickham striae* or telangiectasia and absence of follicular ostia were noted on the skin and scalp, respectively. No mucosal or nail involvement was noted.

Laboratory examinations, including complete blood counts and routine urine tests, were within normal limits except for mild elevation of alanine transaminase (40.48 U/L; normal limits 0-40 U/L). Antinuclear antibody was negative (antibody index: 0.456; values less than 0.90 are considered negative).

The patient was initially treated with cetirizine 10 mg/tablet as needed for pruritus, topical clobetasol propionate 0.05% ointment twice daily, and mupirocin 2% ointment twice daily for two weeks, in addition to emollients and sunblock with SPF greater than 50. After two weeks of treatment, it was observed that the lesions had improved, showing reduced erythema, erosions, scales, and crusts. The clobetasol propionate 0.05% ointment was continued for eight weeks and was switched to tacrolimus 0.1% ointment for one week. Upon follow-up (Figure 2), no new lesion was observed. Since this case was uncommon, we obtained permission and informed consent from the patient to make a case report and publish it for education.

A 4-mm skin punch biopsy was taken from the extensor aspect of the right forearm, which revealed flat and atrophic epidermis, hypergranulosis, few necrotic keratinocytes, focal vacuolar alteration at the dermo-epidermal junction (DEJ) and exocytosis of lymphocytes, sub-epidermal linear split, numerous melanophages beneath the epidermis and interstitial spaces, and lichenoid band of lymphocytes in papillary dermis (Figure 3). This result was compatible with histopathologic findings LP.

Discussion

We presented an interesting case of a 61-year-old Filipino man with cutaneous LP concomitant with cicatricial alopecia on the scalp, mimicking discoid lupus erythematosus. At a glance, the most remarkable lesion from the patient was the multiple erythematous atrophic scarring hairless plaques on the scalp accompanied by multiple atrophic

coin-shaped plaques on the face and extensor aspects of both forearms. The clinical characteristic of scarring alopecia and atrophic coin-shaped scarring plaques on the sun-exposed area brought DLE as the primary clinical impression.

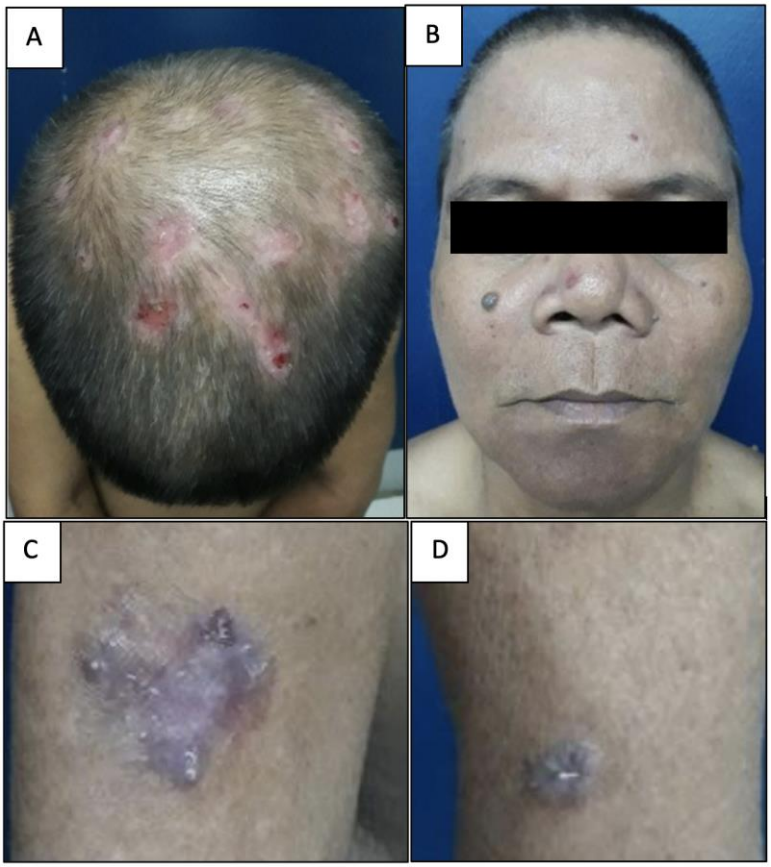

Figure 1. A. Multiple, well-defined, erythematous to hypopigmented atrophic plaques topped with excoriations, erosions, and central scarring on the scalp. B. There are few well-defined, erythematous to violaceous plaques on the face. C, D Multiple, well-defined, erythematous–violaceous to hyperpigmented atrophic plaques, some topped with excoriations, erosions, scales, and crusts on both extensor aspects of both forearms.

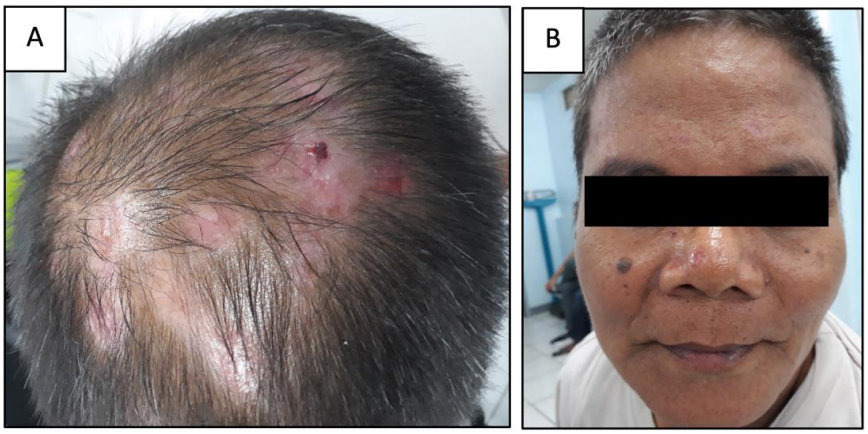

Figure 2. Upon Follow-Up, after Six Weeks of Treatment**.**
A. The excoriations and erosions were decreased, although there were still multiple slight erythematous to hypopigmented atrophic scarring plaques.
B. The previous erythematous to violaceous plaques on the face healed.

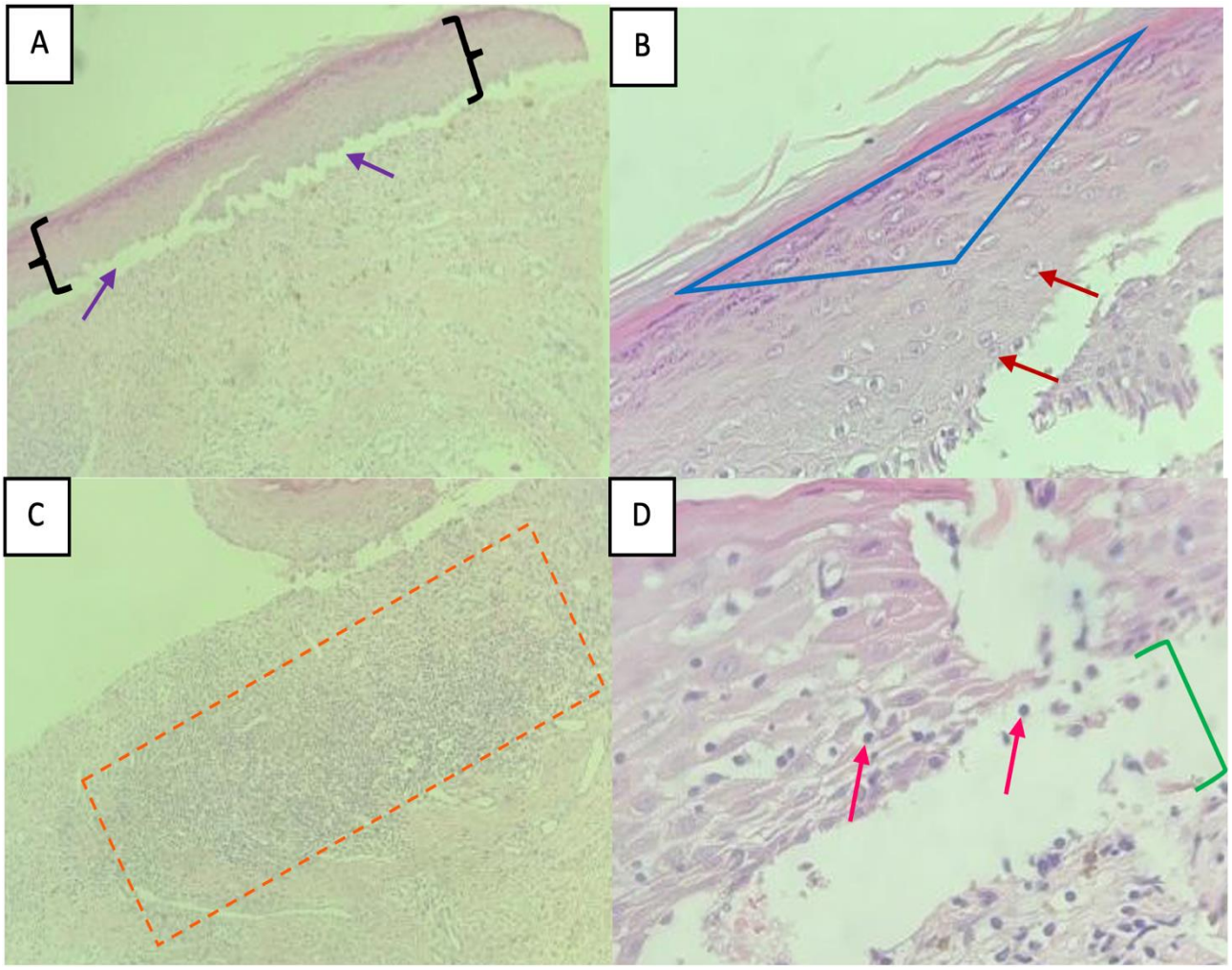

Figure 3. Histopathological Findings.
A. Atrophic epidermis (black curly brackets) and sub-epidermal linear split (purple arrows) resulting from interface vacuolar alteration.
B. Wedge-shaped hypergranulosis (the area inside the blue triangle) and necrotic keratinocytes (red arrows).
C. Lichenoid band of lymphocytes in the papillary dermis (area inside the orange dotted box).
D. Vacuolar alteration at the DEJ (green bracket) and exocytosis of lymphocytes (pink arrows).

Initially, we ruled out LP because, upon the examination, we did not find the hallmark of *Wickham striae* and oral involvement, which usually occurs in 60-70% of LP patients. In addition, the lesions were primarily found in the extensor aspects and sun-exposed area, which is just the opposite of flexural predilection in LP. Most of all, cutaneous LP is a non-scarring disease (except for LPP), contrary to the significant scarring lesions in this patient. We did the skin biopsy and requested some laboratory examinations. An antinuclear antibody (ANA) test was performed to exclude the possibility of SLE, and the result was negative. Nevertheless, the histopathologic result revealed some prominent features of LP, including hypergranulosis, few necrotic keratinocytes, vacuolar alteration at DEJ, and the lichenoid band of lymphocytes in the papillary dermis. The first-line treatment for limited cutaneous LP is high-potency topical corticosteroids, the same as the first-line therapy for LPP. We prescribed the patient cetirizine 10 mg/tablet od to help ease the intractable pruritus, topical clobetasol propionate 0.05% ointment bid, which was the first-line treatment for two weeks, mupirocin 2% ointment to be applied on the excoriations and erosions, as well as emollients and sunblock SPF greater than 50.

Mupirocin 2% ointment was given because there were significant excoriations and erosions due to the vigorous scratching. Not only to prevent

secondary bacterial infection, but mupirocin was also claimed to promote wound healing by stimulating keratinocyte proliferation, improving angiogenic activity, and enhancing the production of several growth factors.[8,9] The patient was advised to wear sunblock in the daytime since he worked as a motorcycle courier, exposing him to direct sunlight daily. Furthermore, most of the lesions were found in the sun-exposed area. It is known that a new lesion of LP may be triggered by the Koebner phenomenon, which one of the causes is sunburn.[10] Narrow-band UVB phototherapy is one of the treatment choices for LP. However, some literature does not recommend direct UV rays as a substitute for directed phototherapy.

Natural UV rays display all wavelengths across the spectrum, while phototherapy exhibits only selected particular wavelengths, filtering out the ineffective, even harmful, and burning rays found in the natural UV rays. After two weeks of the treatment, the lesions were improved, with less erythema, erosions, scales, and crusts. The clobetasol propionate 0.05% ointment was continued for another six weeks and was switched to tacrolimus 0.1% ointment for one week in the interim. Upon the follow-up, no new lesion was noted. This case report provided an unusual case with some interesting pictures; hopefully, it can give a new insight into recognizing other patients with similar cases. However, this case report was limited to one case; thus, the findings cannot be generalized.

Conclusion

LP can present with several morphological cutaneous presentations, including atrophic LP, which may mimic cutaneous DLE. Even though LP is a non-scarring disease, a follicular variant of LP, LPP has a distinct clinical and histologic entity with associated scarring alopecia. Atrophic cutaneous LP concomitant with scalp LPP may mimic DLE clinically and warrant further laboratory and histopathology examinations to establish the final diagnosis, considering that LP and LE have different courses of disease and treatment of choice.

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Author Contributions

All authors contribute equally for this case report.

Conflict of Interests

No conflict of interest.

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