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Linear Pigmented Discoid Lupus Erythematosus on the Scalp of a 52-Year-Old Male, Filipino Treated with Pico Laser: A Case Report

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Abstract

Linear cutaneous lupus erythematosus is an exceptionally rare variant of cutaneous lupus erythematosus (CLE) characterized by linear erythematous plaques along the lines of Blaschko. This case report describes a unique presentation of linear pigmented discoid lupus erythematosus (DLE) in a 52-year-old, male, Filipino with a history of intense sun exposure. The patient presented with a progressive hyperpigmented linear plaque on the left frontal and left parietal area, without systemic symptoms. The patient demonstrated improvement following a treatment of super high potency topical corticosteroid then topical calcineurin inhibitor with strict photoprotection, and adjunctive measures. The lesions decreased in thickness, hyperpigmentation, and scaling. The Cutaneous Lupus Erythematosus Disease Area and Severity Index showed a decrease in the activity score from six to two, indicating a positive treatment response. Hyperpigmentation of the plaques decreased after two treatment sessions and showed further improvement at one-year follow-up. This is the first reported case of linear DLE on the scalp among local journals in the Philippines, highlighting the importance of considering CLE in the differential diagnosis of linear hyperpigmented plaques, even in the absence of systemic symptoms. This is also the first known case to use pico laser as treatment for DLE, based from our knowledge and from published literature.

Introduction

Lupus erythematosus, a multisystem disorder affecting the skin, involves complex interactions between immunologic, genetic, and environmental factors.[1] In the Philippines, it occurs in 30 to 50 per 100,000 people, compared to 20 to 150 per 100,000 in the United States [2]. Linear cutaneous lupus erythematosus (LCLE) is a rare variant, characterized by linear erythematous plaques along Blaschko lines, with the first case reported in 1998.[3] Among reported LCLE cases, 39.2% were diagnosed as DLE. Nine cases of adult-onset linear CLE on the forehead have been documented.[4] Due to LCLE's rarity and limited data, further research and case studies are needed to enhance our understanding of its pathogenesis, clinical features, and optimal management.

Discoid lupus erythematosus is a chronic form of cutaneous lupus, primarily affects the skin characterized by round or disk-shaped lesions that may be scaly, red, and inflamed. It can cause changes in skin pigmentation and may lead to scarring or hair loss. It occurs more frequently in women during their fourth and fifth decades of life. The prevalence of lupus can vary between different populations and regions.[2]

Linear cutaneous lupus erythematosus primarily affects children and young adults, with a mean age of onset of 22 years. It shows no gender predilection and involves single or multiple asymptomatic linear erythematous plaques following the lines of Blaschko. The most affected areas include the head, neck, trunk, then extremities.[4]

As far as the available records indicate, only 16 cases of LCLE involving the scalp have been reported in the English language as of 2022.[5] Only 23 patients reported patients were from Asia. [3,6,7] In the Philippines, there has been 724 cases of discoid lupus erythematosus with no reported case of being linear in pattern from 2011 to 2022.[8]

It is a connective tissue disease with a complex mix of immunologic, genetic, and environmental factors. Possible triggers include trauma, viral infections, irritation, or exposure to agents like ultraviolet (UV) light, drugs, pesticides, metals, and more. Among these, UV radiation is particularly linked to lupus-specific skin issues in this patient. Photosensitivity and antinuclear antibody (ANA) test results are usually negative or weakly positive in linear cutaneous lupus erythematosus.[9] Histologically, it shows features like hyperkeratosis, follicular plugging, epidermal atrophy, and basal layer degeneration with dense lymphocyte infiltration around blood vessels and adnexal structures, along with dermal pigmentary changes.[10]

It has been observed that 5% to 15% of patients with DLE have the potential to progress and develop SLE.[9] The timeframe between the initial diagnosis of DLE and the onset of SLE vary significantly with intervals ranging from four months to 34 years.[11] From our knowledge and from published literature, no current data of linear DLE has developed to SLE.

Case Report

A 52-year-old, male, Filipino who is a retired medical technologist from Cavite presented with a 3-month history of solitary, hyperpigmented linear plaque on the left frontal area extending to the forehead. There were no pain, tenderness, pruritus, scales, hair loss, malar rash, oral ulcers, fever, dyspnea, chest pain, bubbly urine, joint

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pains, muscle pains, seizures, dizziness and pallor. No topical agents used prior. He went to the beach with no sun protection one month prior to the appearance of lesion. No medications were taken nor applied. Interval history showed increase in size and pigmentation of the lesion extending in a linear, downward fashion and appearance of fine, whitish, adherent scales. Persistence of the linear plaque prompted consultation.

He has no comorbidities and no known allergy. He has had no prior surgeries, injuries and accidents and did not receive blood transfusion or any of its components. He has no record of taking any medications and supplements. He has no history of prolonged exposure to ionizing radiation. He has no history of herpes zoster infection. He has a family history of hypertension, diabetes mellitus, psoriasis and rheumatoid arthritis. He is a 20 pack-year smoker and occasional alcoholic beverage drinker.

He presented with a solitary, well demarcated, linear violaceous to hyperpigmented plaque measuring 8 cm in length and 2 cm in width diameter with flat hyperpigmented borders and central hypopigmented area with scanty, fine adherent scale in the left frontal area extending to the forehead (Figure 1A) and a solitary, well demarcated, irregularly shaped, slightly hyperpigmented plaque measuring 3 cm by 2 cm with scanty, fine, adherent whitish scale with hair loss on the left parietal area (Figure 1B). There was a positive carpet tack sign. No hair loss but with hair thinning was noted.

There were no lesions on the neck, chest and abdomen, back, axilla, both upper and lower extremities, palms and soles. No nail changes were seen.

Ancillaries showed leukopenia and eosinophilia. Chemistry panel of blood urea nitrogen, creatinine, fasting blood sugar, hemoglobin A1C, aspartate aminotransferase, alanine aminotransferase and blood uric acid were all normal. Lipid profile showed elevated low-density lipoprotein. No casts, no hematuria, no proteinuria were seen on urinalysis. C-reactive protein and antinuclear antibody were both negative. Chest X-ray showed essentially unremarkable findings.

Dermoscopy on the left frontal area showed follicular keratotic plugs with perifollicular white halos, rosettes, yellow dots and irregular linear vessels. There is presence of white structureless areas and pink white background and speckled brown pigmentation (Figure 2).

Dermoscopy on the left parietal area of the scalp revealed perifollicular white yellowish scales and follicular keratotic plugs (Figure 3).

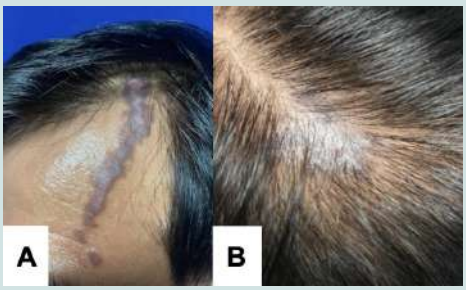

Figure 1: Initial photo of the patient showing a plaque on left frontal area (A) and left parietal area (B)

There were no proximal nailfold capillary changes seen in capillaroscopy.

Biopsy was done on the left frontal area. Stratum corneum showed marked parakeratosis. The epidermis was atrophic with focal areas of hyperplasia (Figure 4).

There was extensive basal vacuolar alteration with presence of melanophages in the papillary dermis (Figure 5A) with presence of some necrotic keratinocytes (Figure 5B). In the dermis are lichenoid, superficial and deep patchy dense perivascular and periadnexal infiltrates of lymphohistiocytes (Figure 5C). The blood vessels were telangiectatic in the upper dermis (Figure 5D).

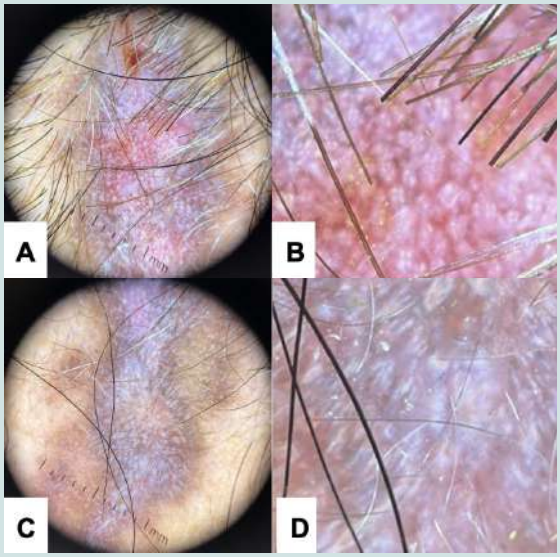

Figure 2: Dermoscopy with polarizing light of left frontal area (A, C) and a closer view (B, D)

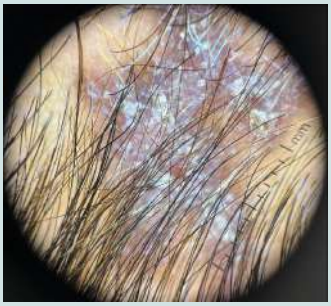

Figure 3: Dermoscopy with polarizing light of left parietal area

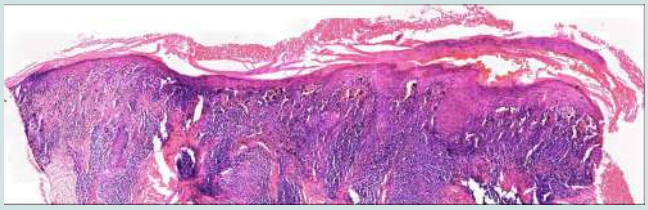

Figure 4: Hematoxylin-Eosin-stained section with 10x magnification

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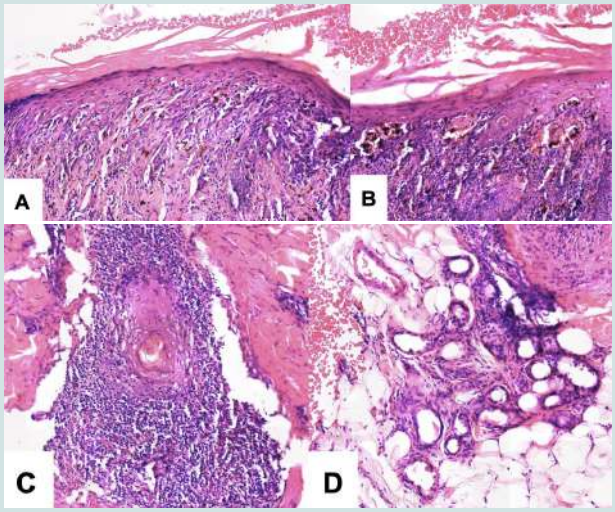

Figure 5: Hematoxylin-Eosin-stained section with 40x magnification (A. vacuolar interface, B. necrotic keratinocytes, C. patchy dense infiltrates, D. telangiectatic blood vessels)

Alcian blue staining was strongly positive for interstitial mucin deposition with enhancement of the blue color both in the dermis and around the perivascular and periadnexal structures (Figure 6).

Patient was treated with super high potency topical corticosteroid, clobetasol propionate 0.05% ointment twice a day for two weeks then once a day for the next two weeks. He was advised with strict photoprotection, mild soap and emollients. He was encouraged to discontinue cigarette smoking and to follow-up after two weeks. There was decrease in the thickness, hyperpigmentation, scaling of plaques on forehead and scalp on follow-up. In week four, medication was shifted to topical calcineurin inhibitor, tacrolimus 0.1% ointment applied twice a day to lesions for 20 weeks. Further decrease in the thickness, hyperpigmentation and scaling of plaques on forehead and scalp was seen (Figure 7) (Figure 8). Dermoscopy showed disappearance of the follicular plugs, perifollicular white halos, rosettes, yellow dots, and telangiectatic vessels from the baseline. Only brown pigmentation and few white structureless areas remained (Figure 9).

Cutaneous Lupus Erythematosus Disease Area and Severity Index (CLASI) was measured and activity score decreased from six to two suggesting a positive response to the treatment.

Thereafter, the patient had pico laser treatment. The treatment parameters of Nd:YAG picosecond laser were 1,064 nm, 3-mm spot size, fluences 0.8 J/cm^2, 1 Hz with one pass and whitish gray spot as the end point. Regular follow-up appointments of every four weeks were scheduled and showed adverse effects of pruritus. There was decrease in hyperpigmentation on the plaques after two sessions. Furthermore, there was decrease in hyperpigmentation after one-year of follow-up with constant sun protection.

Discussion

From 1978 to 2022, 72 cases of linear DLE were reported in 45 global journals. The patients had an average age of 24.47 years, and there was nearly an equal distribution between genders, with 35 males and 37 females (Table 1).

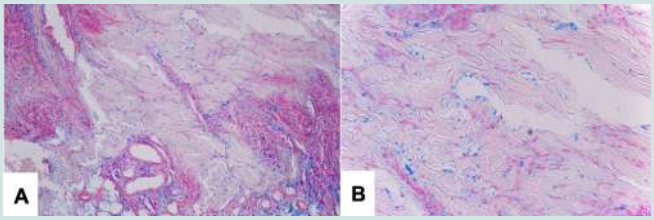

Figure 6: Alcian blue stained section with 20x magnification (A) and 40x magnification (B)

Figure 7: Post treatment on left frontal area

Figure 8: Post treatment on left parietal area

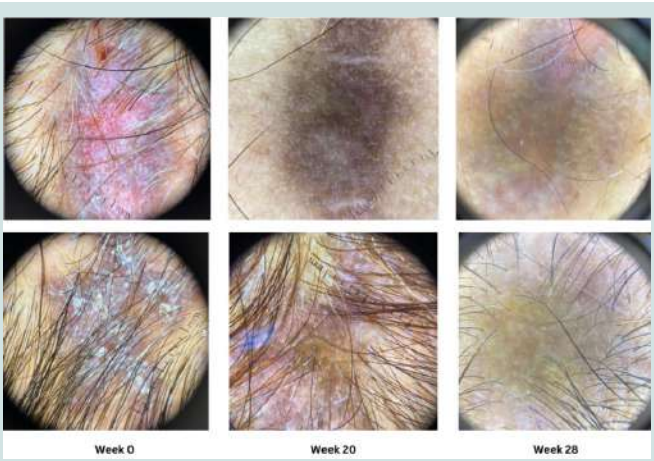

Figure 9: Dermoscopy of post treatment on left frontal area and left parietal area

The most affected location was the face (72%) followed by upper extremities (21%), neck (19%), trunk (7%), lower extremities (5.5%), scalp (4.1%), back (3%), and buttock (1.3%) (Table 1). The average duration from onset to presentation was approximately 4.5 years.

The initial diagnoses included linear cutaneous lupus erythematosus (24%), lichen planus (18%), discoid lupus erythematosus (12%), morphea (7%), lichen striatus (6%), epidermal nevus (2%), and unspecified diagnosis (31%). Among the patients, 22% had positive ANA tests, and 4.6% experienced photosensitivity symptoms (Table 1). Treatment strategies included photoprotection, topical or intralesional corticosteroids, and calcineurin inhibitors. Some used hydroxychloroquine alongside strict sun protection. While some patients improved, others had persistent post-hyperpigmentation despite treatment.

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Table 1: Summary journals of linear DLE worldwide and their clinical characteristics

Year	Reference	Age	Sex	Location	Duration from Onset	Initial Diagnosis	ANA	Associated symptoms
1978	Umbert P and Winkelmann R [12]	7	F	Arm	1 year	DLE	-	-
1998	Abe M et al [3]	11	F	Face, neck	3 years	N/A	-	-
1998	Abe M et al [3]	3	F	Right cheek	11 months	DLE	+	-
1999	Bouzit N et al[13]	29	F	Forehead	3 years	Lichen striatus or inflammatory linear verrucous epidermal nevus	-	N/A
1999	Green J et al[14]	7	M	Bilateral cheeks, nasolabial fold, occiput, right parietal scalp., trunk	1 year	N/A	+	-
1999	Green J et al[14]	8	M	Face, trunk	3 months	DLE	+	-
2000	Abe et al [3]	23	M	Cheek	N/A	N/A	-	N/A
2001	Davies MG and Newman P[15]	18	F	Left cheek	2 years	N/A	-	-
2001	Choi JC et al[16]	6	F	Nose	6 months	N/A	-	-
2001	Lee MW et al[17]	4	M	Infraorbital area; cheek	6 months	N/A	-	-
2006	Sabat et al[18]	19	M	Epicanthal area and nose	5 years	N/A	+	-
2006	Rockman H[19]	42	F	Right trunk, leg	N/A	N/A	N/A	-
2007	Engelman DE et al[20]	6	F	Trunk	6 years	N/A	-	-
2008	Julia M et al[21]	13	F	Arm, buttock	9 years	N/A	-	-
2009	Gaitanis et al[22]	21	M	Supraorbital and infraorbital area, perioral	1 year	CLE	-	N/A
2009	Thind et al[23]	22	F	Perioral, chin	1 year	CLE	-	-
2010	Kim et al[24]	33	M	Forehead, nose	2 months	LCLE	-	-
2011	Alcantara-Gonzalez et al[25]	45	M	Scalp	N/A	N/A	-	-
2011	Daldon PEC and Lage R[26]	15	M	Arm	2 years	N/A	+	-
2011	Kawachi Y et al[27]	6	F	Mandibular area, neck	N/A	N/A	N/A	-
2011	Imhof L et al[29]	15	F	Cheek, perioral area	3 years	LCLE	-	-
2011	Alcantara-Gonzalez et al[25]	64	M	Neck	7 months	N/A	-	-
2011	Kawachi Y et al[26]	6	F	Cheek, auricle, neck	2 years	LCLE	-	-
2012	Verma et al[26]	31	F	Cheek, upper back and extremities	6 years	DLE	+	N/A
2013	Aiyama A et al[30]	11	M	Arm	11 years	DLE	+	-
2013	Aiyama A et al[30]	22	M	Arm	11 years	LCLE	+	-
2015	Ma H et al[31]	13	F	Mandible, auricle, neck	7 years	LCLE	-	-
2015	Frances K et al[32]	15	F	Arm	2 years	Lichen striatus	-	-
2015	Frances L et al[32]	19m	F	Right arm	2 months	LS	N/A	N/A
2015	Mao QX et al[10]	32	F	Mandible, neck	N/A	N/A	+	N/A
2016	Jin H et al[33]	14	M	Forehead, nose, cheek, thigh	2 years	CLE	+	Photosensitivity
2016	Jin H et al[33]	12	F	Angle of mandible, neck	3 months	CLE	+	-
2016	Jin H et al[33]	12	M	Forehead, cheek, preauricular area	3 years	CLE	-	-
2016	Marinho AK et al[34]	9	F	Upper and lower limb	5 years	N/A	+	-
2017	Saha et al[35]	45	M	Left forearm	2 years	N/A	N/A	N/A
2017	Mao XM et al[10]	31	F	Mandible, neck	1 month	LCLE	-	-
2017	Campos-Munos L et al[36]	11	F	Forehead, neck	N/A	DLE	-	Photosensitivity
2018	Meena M et al[37]	57	M	Right face	5 years	N/A	-	-
2019	Sindhusen S et al[4]	28	F	Forehead	3 month	Morphea	-	-
2019	Hassan M et al[38]	24	M	Scalp to nasal tip	N/A	Morphea	N/A	N/A
2020	Toyama et al[7]	7	M	Arm	4 years	LCLE	-	-
2020	Yadav D et al[39]	6	M	Cheek, limb, trunk	1 year	DLE	+	-
2020	Niki M et al[40]	12	F	Cheek	3 years	LCLE	+	+

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2020	Perez-Bernal J et al[41]	15	M	Arm	8 years	Lichen striatus	-	-
2020	Liu W et al[6]	12	F	Right forehead	1 year	N/A	-	-
2020	Liu W et al[6]	16	M	Perioral, neck	3 years	N/A	-	-
2020	Lim D et al[42]	15	M	Thigh	3 years	N/A	-	-
2020	Lim D et al[42]	12	F	Thigh	6 years	N/A	-	-
2020	Liu W et al[6]	16	M	Right lower lip to neck	12 years	Lichen planus	-	-
2020	Liu W et al[6]	50	M	Left forehead and left nasal alar	6 years	Lichen planus	-	-
2020	Milosalvjevic K et al[43]	55	M	Left arm	3 month	LCLE	-	N/A
2020	Liu W et al[6]	42	F	Left nasal dorsum)	6 years	Linear morphea	-	-
2020	Liu W et al[6]	54	M	Right jaw to neck	3 years	LCLE	-	-
2020	Liu W et al[6]	47	M	Right lower lip to neck	7 years	Lichen planus	-	-
2020	Liu W et al[6]	12	F	Left forehead, left lateral eyelid, left jaw	8 years	Linear morphea	-	-
2020	Liu W et al[6]	49	F	Left outer canthus	36 years	Lichen planus	+	-
2020	Liu W et al[6]	27	F	Right postaurem	36 years	LCLE	-	-
2020	Liu W et al[6]	36	M	Left nasal dorsum	3 years	Lichen planus	-	-
2020	Liu W et al[6]	31	M	Right jaw to neck	1 year	LCLE	-	-
2020	Liu W et al[6]	40	F	Right forehead to nasal dorsum	2 years	Lichen planus	-	-
2020	Liu W et al[6]	45	M	Left forehead	1 year	LCLE	-	-
2020	Liu W et al[6]	46	F	Right jaw	3 years	LCLE	-	-
2020	Liu W et al[6]	40	F	Lateral forehead and neck	12 years	Lichen planus	-	-
2020	Liu W et al[6]	31	M	Left jaw	24 years	Lichen planus	-	-
2020	Choudhary et al[44]	37	F	Cheek	1 ½ year	Lichen planus	-	-
2020	Liu W et al[6]	43	M	Left jaw	2 years	LCLE	+	-
2020	Liu W et al[6]	60	M	Right jaw to neck	3 years	Lichen planus	-	-
2020	Liu W et al[6]	46	F	Right forehead	10 days	Lichen planus	-	-
2021	Kumar P et al[45]	32	F	Forehead, left side of nose	2 months	Lichen planus	+	-
2021	Wu V et al[46]	29	M	Back, shoulder, right arm	5 years	N/A	N/A	N/A
2022	Gorai S et al[47]	48	F	Left breast	2 years	DLE	-	-

Acute exacerbations of DLE are typically treated with high potency topical corticosteroids to reduce inflammation and promote healing. Improvement is often seen within two weeks. Once lesions show signs of inactivity, treatment can stop. Prolonged use of topical corticosteroids may cause skin thinning. Alternative therapies can be considered if there is no response to topical corticosteroids. Topical calcineurin inhibitors like tacrolimus or pimecrolimus are options, especially for facial involvement. They modulate the skin's immune response without causing atrophy. Physical treatments like laser therapy, cryotherapy, and dermabrasion can also manage CLE.[9]

Previous research found that combining intense pulsed light (IPL) and Q-switched 1,064-nm Nd:YAG laser effectively treated DLE scarring lesions without worsening them.[48]

In a separate study, a 1,064-nm long-pulse Nd:YAG laser improved DLE lesions in three sessions, three weeks apart.[49]

Another study used pulsed dye laser (PDL) on 12 DLE patients three times, six weeks apart, resulting in decreased erythema and scaling/hypertrophy scores after six weeks, but no significant change in damage CLASI scores.[50]

In a different study, flashlamp PDL and long PDL showed an average improvement in over 60% of treated lesions, with some side effects like transient hyperpigmentation, mild scarring, and a few relapses after more than a year of treatment.[51]

Erbium-doped yttrium-aluminum-garnet (Er:YAG) laser treatment involved two sessions with six to ten passes. After one week, treated areas reepithelialized smoothly with minimal residual erythema and no scarring. There was no disease reactivation observed in both treated and untreated areas at a two-year follow-up.[52]

Pico laser, or picosecond Nd: YAG laser, delivers ultra-short laser pulses, approximately 1/1000 of a nanosecond, compared to traditional lasers. It targets skin concerns like tattoo removal, wrinkle reduction, and pigmentation removal efficiently and precisely. Its rapid energy delivery breaks down pigment or tissue without harming surrounding skin, reducing burns and pigmentation changes. Pico laser's faster rate breaks up ink or pigmentation into smaller particles, potentially speeding up and enhancing removal safely.[53] Based from our knowledge and from published literature, no linear DLE has been treated with pico laser. For this patient, pico laser was used for two sessions and regular follow-up appointments of every four weeks were scheduled. It showed adverse effects of pruritus. Hyperpigmentation of the plaques decreased after two treatment sessions and showed further improvement at one-year follow-up.

An additional consideration is whether the patient had a prior history of herpes zoster. Discoid lupus erythematosus has been reported to occur at sites of healed herpes zoster, often interpreted as an example of Wolf's isotopic response. [54-57] Although this history was not present in our patient, including such detail may

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provide important clinical context in similar cases. Notably, herpes zoster has also been observed on the scalp following Gam-COVID-Vac vaccination,[58] underscoring the potential interplay between viral infections, immune modulation, and cutaneous autoimmune manifestations.

Conclusion

This is the first reported case of linear pigmented DLE on the scalp in the Philippines. It is also the first known case to use pico laser as treatment for DLE, based from our knowledge and from published literature. The case involves a 52-year-old Filipino male with a rare presentation of linear pigmented DLE. He had a hyperpigmented linear plaque on the left frontal and parietal areas, preceded by intense sun exposure. No systemic symptoms were reported, and the patient had no significant medical history or known allergies.

This case emphasizes considering CLE as a potential diagnosis in patients with linear hyperpigmented plaques and sun exposure history, even without systemic symptoms. Treatment included a super high potency topical corticosteroid, strict photoprotection, and adjunctive measures, leading to decreased thickness, hyperpigmentation, and scaling. Topical calcineurin inhibitor was applied twice daily for 20 weeks, further improving the lesions. Dermoscopy revealed significant improvement, with only brown pigmentation and a few white areas left after 20 weeks. The CLASI score decreased from six to two, indicating a positive response to treatment. Hyperpigmentation of the plaques decreased after two treatment sessions and showed further improvement at one-year follow-up.

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