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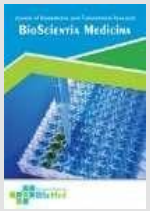

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A Distinctive Clinical Phenotype of Discoid Lupus Erythematosus in Papuanese Women: A 5-Year Analysis of Dyspigmentation, Scarring, and Malar Predilection

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A B S T R A C T

Background: Discoid lupus erythematosus (DLE), the most common form of chronic cutaneous lupus, exhibits significant clinical variability influenced by ethnicity. While disparities in presentation are recognized, data from unique indigenous populations such as the Papuanese in East Indonesia remain scarce. This study aimed to characterize the clinical and sociodemographic features of DLE in this specific cohort to identify its potentially distinctive phenotype. **Methods:** A five-year retrospective analysis of clinical databases was conducted at the Department of Dermatology and Venereology at a tertiary referral hospital in Jayapura, Papua, Indonesia. All patients clinically diagnosed with DLE by board-certified dermatovenereologists between January 2019 and December 2023 were included. Sociodemographic and clinical data, including lesion morphology, location, and management, were systematically collected and analyzed using descriptive statistics. **Results:** A total of 22 patients meeting the criteria were identified. The cohort demonstrated remarkable homogeneity; all patients were of Papuanese ethnicity and female (100.0%). The majority were in the 26-35 age group (40.9%), with a mean age of 29.4 years, and half were farmers (50.0%). Clinically, lesions were universally present on the nose and/or malar area (100.0%). The most common morphological triad was dyspigmentation, scarring, and telangiectasia, observed in 81.8% of patients. All patients reported photosensitivity and were managed with photoprotection and topical steroids. **Conclusion:** DLE I n Papuanese women presents as a distinctive, highly uniform clinical phenotype characterized by an exclusive female predilection, a strong association with sun exposure, and a universal malar distribution with a high propensity for disfiguring dyspigmentation and scarring. These findings underscore the necessity of culturally competent, early, and aggressive management strategies to mitigate long-term sequelae in this vulnerable population.

1. Introduction

Lupus erythematosus (LE) represents a complex spectrum of autoimmune disorders that primarily target the skin, although systemic involvement is a defining feature of its most severe form.[1] The cutaneous manifestations of LE (CLE) are diverse and are broadly classified into acute (ACLE), subacute (SCLE), and chronic (CCLE) subtypes. Among these, discoid lupus erythematosus (DLE), the cardinal presentation of CCLE, is the most frequently

encountered subtype in dermatological practice.[2] DLE can exist as a localized disease confined to the skin or, in a minority of cases, may be associated with or progress to systemic lupus erythematosus (SLE).[3]

Clinically, classic DLE is characterized by well-demarcated, erythematous-to-violaceous papules and plaques that evolve to exhibit an adherent scale, follicular plugging, and, ultimately, central atrophy, scarring, and pigmentary alteration.[4] These lesions show a strong predilection for sun-exposed areas, most notably the face, scalp, and ears, reflecting the critical role of ultraviolet (UV) radiation as a primary trigger in genetically susceptible individuals. The disease typically affects individuals between the ages of 20 and 40, with a well-documented and significant predilection for females over males.[5,6]

In recent years, the field of dermatology has increasingly recognized the profound impact of ethnicity and skin phototype on the manifestation, severity, and outcomes of numerous skin disorders. Conditions such as psoriasis, for instance, have been shown to present with different morphological characteristics and biopsychosocial impacts across various racial and ethnic groups, a realization that has spurred more inclusive and tailored therapeutic approaches.[7] This paradigm is particularly relevant to DLE, where pigmentary disturbances—both hyper- and hypopigmentation—are known to be more frequent, severe, and cosmetically disfiguring in patients with skin of color. Despite this knowledge, significant gaps persist in the medical literature concerning the specific clinical characteristics of DLE in many of the world's diverse indigenous populations.[8]

Indonesia, an archipelago of extraordinary ethnic and geographic diversity, presents a unique setting for studying these variations. However, published data on the clinical and demographic profile of DLE from this region, particularly its eastern provinces, are exceptionally limited. The Papuanese people of East Indonesia represent a distinct ethnic group with unique genetic and phenotypic traits. Understanding the presentation of autoimmune diseases like DLE in this population is essential for addressing health disparities and providing equitable dermatological care. The lack of baseline data has hindered the development of region-specific clinical guidelines and patient education strategies.[9]

This study represents one of the first detailed clinical characterizations of DLE within the indigenous Papuanese population of East Indonesia, a genetically and phenotypically distinct group. Its novelty lies in the systematic documentation and analysis of a homogenous patient cohort, which has revealed a uniquely uniform clinical phenotype previously undescribed in the literature.[10] Therefore, this study aimed to retrospectively analyze and characterize the sociodemographic profile, clinical presentation, and therapeutic management of DLE in Papuanese women over a five-year period to define its distinctive features, contextualize them within the global understanding of the disease, and inform culturally competent clinical practice.

2. Methods

This study was conducted as a retrospective, single-center, descriptive analysis of patient medical records. All data were collected from the clinical databases of the Department of Dermatology and Venereology, Faculty of Medicine, Universitas Cenderawasih/Abepura Regional General Hospital, located in Jayapura, Papua, Indonesia. This facility functions as a tertiary referral center for the province of Papua. To ensure patient confidentiality, all data were fully anonymized and de-identified prior to analysis. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

The study population included all patients who were referred to and managed at the outpatient dermatology clinic with a clinical diagnosis of DLE between January 1st, 2019, and December 31st, 2023, a period spanning five full years. Inclusion criteria stipulated a clinical diagnosis of DLE and an age of 12 years or older at the time of diagnosis. Patients with incomplete medical records regarding primary

demographic or clinical characteristics were excluded.

The clinical diagnosis of DLE was established by at least two board-certified dermatovenereologists based on a constellation of characteristic clinical findings. These defining features included the presence of one or more inflammatory, indurated erythematous plaques, often with evidence of central atrophy, follicular plugging, and significant pigmentary disturbances (hyper- or hypopigmentation) predominantly in sun-exposed regions with symmetrical distribution. The diagnosis was further supported by evidence of solar damage, such as lentigines or keratotic papules.

A standardized data collection form was utilized to extract relevant information from the anonymized medical records. The collected data were organized into three main categories: Sociodemographic Characteristics: Data were derived from the patient's official family certificate and direct history taking. This included gender, age, occupation, and self-declared ethnicity (race); Clinical Characterization: This comprised a detailed description of the dermatological findings at the time of presentation. It included patient-reported symptoms (notably photosensitivity), the anatomical location of the lesions, and the specific morphology of the lesions (such as dyspigmentation, scarring, telangiectasia, papules/plaques, discoid shape, hyperkeratosis, scaling, alopecia); Therapeutic Management: The prescribed treatments for each patient were recorded, categorizing them into photoprotection (sunscreen), topical steroids, and systemic steroids.

All extracted data were entered into a Microsoft Excel spreadsheet for organization and cleaning. Subsequent statistical analysis was performed using SPSS Version 25.0 (IBM Corp., Released 2022, Armonk, New York)27. Descriptive statistics were employed to analyze the data28. Categorical variables were presented as frequencies and percentages (n, %), while continuous variables like age were summarized using the mean and presented with age group classifications.

3. Results

During the five-year study period, 22 patients with a clinical diagnosis of DLE were identified and included in the analysis. The sociodemographic profile of this cohort, detailed in Table 1, was characterized by a remarkable uniformity. All 22 patients (100.0%) were of indigenous Papuanese ethnicity, and all were female. The mean age at diagnosis was 29.4 years. The largest proportion of patients fell into the 26–35 year age group, comprising 9 individuals (40.9%). Analysis of patient occupation revealed a strong link to outdoor activities; exactly half of the cohort (11 patients, 50.0%) worked as farmers, with housewives (6 patients, 27.3%) and students (5 patients, 22.7%) making up the remainder.

Table 1. Sociodemographic profile of DLE Patients in East Indonesia (n=22).

Characteristic	Sub-category	Frequency (n)	Percentage (%)
Age	Mean Age	29.4 years	N/A
	Age Group Distribution		
	12–16 years	1	4.5
	17–25 years	5	22.7
	26–35 years	9	40.9
	36–45 years	5	22.7
	46–55 years	2	9.1
Gender	Female	22	100.0
	Male	0	0.0
Occupation	Farmer	11	50.0
	Housewife	6	27.3
	Student	5	22.7
Ethnicity	Papuanese	22	100.0

The clinical features of DLE in this cohort are summarized in Table 2. The presentation was consistent across all patients. Photosensitivity was a universal symptom, reported by all 22 patients (100.0%). The anatomical location of the lesions showed a complete predilection for the face, with every patient (100.0%) exhibiting lesions on the nose and/or malar area. While the malar region was universally affected, a small number of patients also had lesions on the mucous membranes (13.6%), scalp (9.1%), ears (9.1%), and the V-area of the neck (9.1%).

The lesion morphology was defined by a dominant triad of chronic changes. Dyspigmentation, scarring, and telangiectasia were each prominently featured in 18 of the 22 patients, representing 81.8% of the cohort for each characteristic. Other common lesion types included inflammatory papules and/or plaques (72.7%) and classic discoid lesions (72.7%). Hyperkeratosis and adherent scaling were also frequent, each being present in 11 patients (50.0%). Alopecia was observed in the two patients with scalp involvement (9.1%).

Table 2. Clinical characterization of the DLE study population (n=22).

Clinical feature	Sub-category	Frequency (n)	Percentage (%)
Primary symptom	Photosensitivity	22	100.0
Anatomical location	Nose/ malar area	22	100.0
	Mucous membrane	3	13.6
	Scalp	2	9.1
	Ears	2	9.1
	V area of neck (front)	2	9.1
Lesion morphology	Dyspigmentation	18	81.8
	Scarring	18	81.8
	Telangiectasia	18	81.8
	Papule/ plaque	16	72.7
	Discoid	16	72.7
	Hyperkeratotic	11	50.0
	Scale	11	50.0
	Alopecia	2	9.1

The therapeutic approach for all patients, as detailed in Table 3, was centered on inflammation control and robust photoprotection. All 22 patients (100.0%) were prescribed broad-spectrum sunscreen and received education on sun-avoidance measures. As a first-line therapy, all patients (100.0%) also received topical corticosteroids to manage their inflammatory lesions. For a subset of patients with more severe or recalcitrant disease, systemic treatment was necessary; 7 patients (31.8%) were prescribed systemic corticosteroids during the study period.

Table 3. Therapeutic management of DLE patients (n=22).

Therapeutic category	Specific treatment	Frequency (n)	Percentage (%)
Foundation therapy			
Photoprotection	Sunscreen	22	100.0
Topical anti-inflammatory	Topical Corticosteroid	22	100.0
Second-line therapy			
Systemic anti-inflammatory	Systemic Corticosteroid	7	31.8

4. Discussion

This study provides a critical and granular characterization of DLE within a unique, homogenous cohort of Papuanese women in East Indonesia. The sociodemographic data derived from a five-year retrospective study of 22 patients with Discoid Lupus Erythematosus (DLE) in East Indonesia paints a remarkably clear and compelling portrait of the typical individual affected by this condition in this specific region. Far from being a random assortment of demographic variables, the data reveals a strikingly homogeneous cohort, providing profound insights into the potent interplay between genetic predisposition, hormonal factors, and environmental triggers that precipitate DLE.[11] This profile is not merely descriptive; it is foundational to understanding the unique clinical phenotype observed in this population and has significant implications for targeted public health initiatives and clinical practice.

The findings converge to delineate a distinctive clinical phenotype that is remarkably uniform and carries profound implications for understanding the interplay between genetics, environment, and autoimmunity.[12] The discussion of these findings will focus on three central pillars that define this phenotype: the exceptional demographic homogeneity, the undeniable nexus of sun exposure and lesion localization, and the severe, damage-oriented nature of the lesion morphology.

The most striking demographic feature is the absolute uniformity of the cohort: 100% of the patients were female, and 100% were of Papuanese ethnicity. While a female predilection in DLE is a globally recognized phenomenon, typically cited at ratios of 3:1 or 4:1, an exclusive female prevalence over a five-year period in a tertiary referral center is exceptional.[13] This finding strongly suggests that the factors driving female susceptibility to autoimmune diseases are exceptionally potent in this population. The underlying mechanisms for female predominance in autoimmunity are multifactorial, but two hypotheses are particularly relevant here. First, the role of sex hormones, specifically estrogen, which is known to influence the development, survival, and function of key immune cells like B cells and plasmacytoid dendritic cells, may be a critical factor. Second, the contribution of the X chromosome, which harbors a multitude of immune-related genes, is significant. The presence of two X chromosomes in females may lead to a higher baseline expression of these genes and an increased risk of antibody overproduction following immune triggers.[14] The fact that this absolute female predominance was observed in a genetically distinct and relatively isolated population like the Papuanese may hint at a founder effect, where specific X-chromosome polymorphisms that increase susceptibility to autoimmunity are more common.

The study identified the mean age of disease onset as 29.4 years, with the majority of patients (40.9%) falling within the 26-35 year age bracket. This finding is highly consistent with data from numerous international studies, which consistently report a peak incidence of DLE between the ages of 20 and 40.[15] This alignment demonstrates that while the "who" (Papuanese women) is unique, the "when" of disease onset in this cohort mirrors the global pattern. The clinical and psychosocial significance of this cannot be overstated. DLE emerges in these women during their prime productive and childbearing years. It is a time of establishing careers, building families, and fostering social relationships. The onset of a chronic, often disfiguring dermatological condition during this crucial life stage imposes a substantial burden, impacting self-esteem, social interactions, and economic productivity, particularly when the visible lesions of DLE can lead to social stigmatization.[16]

Perhaps the most functionally significant demographic characteristic identified is occupation. A remarkable 50.0% of the patients in this cohort were farmers. This single data point provides a powerful link between the patient's daily life and the pathophysiology of the disease. DLE is a quintessential photosensitive disorder, and ultraviolet (UV) light exposure is a well-established primary triggering factor in its development.[17] The occupation of a farmer in an equatorial region like Papua involves

intense, prolonged, and chronic sun exposure. This environmental pressure likely serves to unmask the latent autoimmune predisposition in these susceptible individuals.[18] The other listed occupations, housewife (27.3%) and student (22.7%), while not exclusively outdoors, also involve considerable time spent under direct sunlight in this geographic location. This strong occupational link reinforces the critical importance of photoprotection as a cornerstone of both prevention and management strategies. The data compellingly suggests that for this Papuanese cohort, DLE is, in many respects, an occupational disease, where the daily work environment is a direct catalyst for its clinical expression. This intersection of genetics and environment is key to understanding the disease's high prevalence and specific presentation in this group.

The clinical data presented in Table 2 move beyond identifying who is affected and delve into the critical question of how discoid lupus erythematosus (DLE) manifests in this unique Papuanese cohort. The findings are not merely a collection of signs and symptoms; they collaboratively sketch a highly consistent and severe clinical portrait. This portrait is defined by three core pillars: a universal photosensitive trigger, an absolute predilection for the central face, and a morphological signature dominated by permanent, disfiguring damage.[12] This clinical characterization provides a powerful narrative of the disease's pathophysiology and its profound impact on patients.

The second pillar of this phenotype is the powerful and undeniable link between UV radiation and disease manifestation. This study presents a compelling triad of evidence: half the patients were farmers, an occupation defined by intense, chronic sun exposure; all patients reported photosensitivity; and all patients displayed lesions on the maximally sun-exposed malar region of the face. UV radiation, particularly UVB, induces apoptosis in epidermal keratinocytes.[14] This programmed cell death leads to the externalization of intracellular autoantigens, which are normally hidden from the immune system. These antigens are then taken up by plasmacytoid dendritic cells, which in turn produce vast quantities of Type I interferons. This "interferon signature" is a central pathogenic axis in all forms of lupus, driving a vicious cycle of inflammation, photosensitivity, and further cell damage. The universal presentation on the malar area in this cohort is particularly noteworthy. While the "butterfly rash" is a classic sign of lupus, it is not universally present in all DLE populations. For instance, studies among Black patients have shown a clear predominance of severe, scarring DLE on the scalp. The complete absence of scalp predominance and the absolute malar predominance in our Papuanese cohort suggest a site-specific susceptibility that is phenotypically distinct. This could be related to local differences in skin immune response or simply reflect the overwhelming and direct nature of solar irradiation to the face in the occupational and lifestyle settings of these patients.

The data unequivocally establishes ultraviolet (UV) radiation as the primary driver of disease activity in this population. The finding that 100.0% of patients reported photosensitivity and 100.0% exhibited lesions on the nose and/or malar area is a perfect clinical correlation. This is not a mere tendency but an absolute feature, confirming the central role of sun exposure in triggering and exacerbating DLE. Pathophysiologically, UV light induces apoptosis in epidermal keratinocytes. This process causes the externalization of normally hidden intracellular autoantigens. These exposed antigens are then recognized by the immune system, particularly by plasmacytoid dendritic cells, which respond by producing massive amounts of Type I interferons. This "interferon signature" is a cornerstone of lupus pathogenesis, creating a self-amplifying cycle of photosensitivity, inflammation, and further skin damage.[15]

The absolute predilection for the malar region—the classic "butterfly" distribution—is exceptionally significant. While common in lupus, its 100% prevalence here contrasts with studies in other ethnic groups, such as Black patients, where the scalp is

often the most severely affected site.[17] This suggests a unique site-specific susceptibility in Papuanese women. The malar eminences and the bridge of the nose are the areas of the face that receive the highest and most direct dose of incident solar radiation, a factor magnified in an equatorial region like Papua and further intensified by the outdoor occupations of the patients. This anatomical precision provides a clear and direct link between the environmental trigger and the resulting clinical manifestation.

The most alarming aspect of the clinical phenotype is the lesion morphology. The dominant triad of dyspigmentation, scarring, and telangiectasia, each present in 81.8% of patients, represents the end-stage sequelae of a chronic and destructive inflammatory process. This suggests that patients are either presenting late in their disease course or that the disease itself is inherently more aggressive and damaging in this population. The high rate of pigmentary change is a hallmark of inflammatory dermatoses in skin of color. The chronic inflammation in DLE simultaneously stimulates melanocytes to produce excess melanin (leading to post-inflammatory hyperpigmentation) while also damaging the dermo-epidermal junction.[18] This damage allows melanin to fall into the dermis ("pigmentary incontinence") and can also lead to the outright destruction of melanocytes, resulting in permanent hypopigmentation or depigmentation. The resulting mottled appearance of light and dark patches on the face is a source of profound psychosocial distress and is often more concerning to the patient than the initial inflammatory lesions. The presence of scarring in over 80% of patients is a definitive sign of irreversible tissue destruction. The lymphocytic infiltrate in DLE attacks the basal layer of the epidermis and associated structures like hair follicles. This sustained attack leads to dermal atrophy, resulting in the characteristic depressed, "punched-out" scars. The high frequency of scarring in this cohort is a critical finding, as it indicates that the inflammation is not being controlled before permanent damage occurs. It serves as a powerful argument for the necessity of early diagnosis and the institution of aggressive, scar-preventing therapies. These dilated superficial blood vessels (telangiectasia) are another sign of chronic damage. They result from a combination of long-term inflammation and the thinning (atrophy) of the overlying epidermis, which makes the underlying vasculature more prominent.

The final, and perhaps most clinically critical, pillar is the morphological signature of the lesions, which were dominated by a triad of dyspigmentation, scarring, and telangiectasia in over 80% of patients.[19] This is a phenotype of chronic, destructive inflammation. The pronounced dyspigmentation is a hallmark of inflammatory dermatoses in skin of color. The inflammatory process in DLE stimulates melanocytes to increase melanin production (leading to hyperpigmentation) and can also damage the basal keratinocytes, causing melanin to drop into the dermis where it is engulfed by macrophages (a process known as pigmentary incontinence), leading to long-lasting, slate-grey discoloration. At the same time, the intense inflammation can be cytotoxic to melanocytes, resulting in areas of permanent hypopigmentation and depigmentation. This mixture of light and dark patches is exceptionally disfiguring and a source of significant psychosocial distress. The data also shows a high prevalence of features indicative of active inflammation, such as papules/plaques (72.7%), discoid lesions (72.7%), and hyperkeratotic scale (50.0%). The coexistence of these active lesions alongside the markers of permanent damage (scarring and dyspigmentation) suggests that patients are experiencing a chronically active or relapsing-remitting disease course. They are simultaneously forming new, inflamed lesions while bearing the scars of past inflammatory events. This clinical picture underscores the chronic, persistent nature of DLE and the challenge of achieving complete and lasting remission. The low prevalence of alopecia (9.1%) directly corresponds to the low rate of scalp involvement, reinforcing the face as the primary battlefield for the disease in this specific phenotype.

The high prevalence of scarring (81.8%) points to the severity of the inflammatory infiltrate in this phenotype. DLE is characterized by an interface dermatitis, where lymphocytes attack the dermo-epidermal junction.[10] This sustained attack destroys the basal cell layer and damages dermal appendages like hair follicles, leading to the characteristic atrophic, "punched-out" scars and permanent hair loss (cicatricial alopecia) when the scalp is involved. The fact that scarring was so common suggests that by the time these patients presented to the tertiary center, significant, irreversible damage had already occurred. This highlights a critical public health issue: the need for much earlier diagnosis and intervention. The therapeutic data support this, as nearly a third of patients required systemic corticosteroids, a treatment reserved for more severe or widespread disease, indicating that simple topical therapy was insufficient to control the destructive inflammation.

Synthesizing these pillars, the DLE phenotype in Papuanese women appears to be the result of a powerful interaction between a highly susceptible host and a potent environmental trigger. The host is defined by a potential genetic and hormonal predisposition that is exclusively female in this cohort. The trigger is the intense, chronic UV radiation of the equatorial sun, magnified by an outdoor agricultural lifestyle. This combination results in a highly focused, destructive autoimmune reaction on the most exposed part of the body—the face—leading to severe and permanent disfigurement.

The therapeutic management data presented in Table 3 reveal a highly logical, standardized, and guideline-concordant approach to treating discoid lupus erythematosus (DLE) within this specific patient cohort. The treatment strategy is clearly stratified into two tiers: a universal foundation of care provided to all patients and a second-line systemic therapy reserved for more severe or refractory cases. This structured approach reflects a deep understanding of DLE pathophysiology and addresses both the primary environmental trigger and the subsequent inflammatory cascade.

The cornerstone of management for every single patient was a dual strategy of photoprotection and topical anti-inflammatory therapy. The universal prescription of sunscreen (100.0%) is a direct and necessary response to the universal clinical finding of photosensitivity.[18] In a photosensitive disease like DLE, advising patients on sun protection measures is the most critical preventative intervention to minimize the risk of disease progression. By blocking or reflecting ultraviolet (UV) radiation, sunscreen helps to prevent the initial keratinocyte apoptosis that triggers the entire autoimmune cascade. This component of the treatment plan is therefore proactive, aiming to reduce the frequency and severity of disease flares.

Complementing this preventative measure, all patients (100.0%) also received topical corticosteroids. This aligns perfectly with established international guidelines, which position topical corticosteroids as the first-line treatment for managing active, localized DLE lesions. These agents act directly at the site of inflammation to suppress the local immune response, thereby reducing erythema, induration, and the progression towards irreversible scarring and dyspigmentation. The universal application of this dual foundation therapy indicates a consistent and high standard of care within the treating institution, ensuring every patient receives the essential tools to manage their chronic condition.[19]

However, the data also highlights the clinical challenges inherent in this disease. The fact that nearly one-third of the cohort (31.8%) required the addition of systemic corticosteroids provides a crucial, albeit indirect, measure of disease severity.[20] Systemic corticosteroids are typically reserved for flare-ups or for cases that are widespread, rapidly progressing, or unresponsive to topical treatments alone. The need to escalate to systemic therapy for a significant minority of patients suggests that their disease was too aggressive to be controlled by topical measures. This finding corroborates the clinical characterization of DLE in this population as a potentially severe phenotype with a high propensity for causing

significant damage, thus necessitating more potent, systemic immunosuppression in many instances to gain control and prevent further disfigurement.

5. Conclusion

This study provides compelling evidence that discoid lupus erythematosus in the indigenous Papuanese female population of East Indonesia manifests as a powerful and uniquely uniform clinical phenotype. This phenotype is unequivocally defined by an exclusive predilection for women, an absolute localization to the sun-exposed malar region, and a dominant morphological signature of severe dyspigmentation and permanent scarring. The findings paint a clear picture of a disease driven by a potent synergy between a susceptible host and an intense environmental trigger. From a clinical and public health perspective, these results are a call to action. They underscore the urgent need for heightened clinical suspicion of DLE in this population and emphasize the necessity of initiating aggressive, culturally-informed management at the earliest sign of disease to prevent the profound and irreversible cosmetic disfigurement that so clearly characterizes DLE in this unique patient cohort.

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