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ORIGINAL ARTICLE

Demographics and clinical presentations of 844 patients with light and dark skin types with polymorphous light eruption and chronic actinic dermatitis evaluated over 23 years

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Abstract

Introduction: Polymorphous light eruption (PMLE) and chronic actinic dermatitis (CAD) have been classically described in White individuals, although recent studies have reported higher prevalence in patients with dark skin types, particularly African Americans.

Objective: To evaluate for differences in demographic, and clinical features between persons with light and dark skin types who have PMLE and CAD.

Methods: Retrospective review of patients with PMLE and CAD who were diagnosed from January 1, 1998, through November 31, 2021, at a single academic dermatology center.

Results/Discussion: A total of 844 patients (725 [85.9%] female; mean [SD] age of onset: 41.7 [16.9] years) were diagnosed with PMLE, and 60 patients (22 [36.6%] female; mean age, [SD]: 60.6 [10.6] years) of age at presentation, disease duration of 8.2 [7.3] years were diagnosed with CAD. Although just over 50% of the general clinic population was White, the prevalence of PMLE and CAD was significantly higher in dark-skinned individuals compared to light-skinned individuals (PMLE: 625 [74.0%] vs. 219 [25.9%], p value $< .001$; CAD: 43 [71.6%] vs. 17 [28.3%], p value $= .003$) respectively. The pinpoint papular variant of PMLE (PP-PMLE) was predominantly seen in dark-skinned individuals.

Conclusion: A substantial proportion of PMLE and CAD cases are present in dark-skinned individuals. PP-PMLE can be mistaken for lichen nitidus. As such, recognition of this entity is important for adequate evaluation and management of patients with PMLE.

1 | INTRODUCTION

Photodermatoses represent a heterogeneous group of skin disorders that are provoked by ultraviolet radiation (UVR) or visible light exposure.[1] Polymorphous light eruption (PMLE) is the most common photosensitivity disorder, and previously was more commonly reported in women with light skin phototypes aged 20–30 years.[2] Clinically, PMLE is characterized by recurrent, delayed reactions to sunlight, ranging from erythematous papules, papulovesicles, and plaques to erythema multiforme-like lesions on sun-exposed surfaces. The same morphology tends to occur in the same individual. Although the estimated worldwide prevalence ranges from 10% to 20%, with higher rates reported at higher altitudes and in western countries, the occurrence of PMLE in dark-skinned individuals has now been

commonly reported.[2,3] Patients with PMLE are less susceptible to UVR-induced immunosuppression, resulting in the development of lesions following UV exposure.[4]

Following PMLE, chronic actinic dermatitis (CAD) is the second most common immunologically-mediated photosensitivity disorder classically described in older white men.[5] Chronic actinic dermatitis is a delayed-type hypersensitivity reaction to endogenous photoallergens and exogenous allergens.[5] The condition is characterized by a persistent dermatitis and/or lesions mimicking cutaneous T-cell lymphoma affecting predominantly sun-exposed sites. Diagnosis can be confirmed with phototesting, which reveals reduced minimal erythemal doses (MEDs), especially in the ultraviolet B (UVB) and shorter ultraviolet-A (UVA) wavelengths.[6] In some cases, phototesting can demonstrate an abnormal response to visible light (VL).[7] While the exact prevalence is unknown, newly emerged studies have reported an increase in the frequency of CAD in the skin of color population, particularly among African Americans (AAs).[8]

The aim of this study was to evaluate patients diagnosed with PMLE and CAD during a 23-year period in our photomedicine center, and investigate differences in demographic, clinical, and photobiological features between patients with light and dark skin types.

2 | METHODS

A retrospective record review of patients with PMLE and CAD diagnosed in the Photodermatology Unit, Department of Dermatology at Henry Ford Health (Detroit, MI, USA), from January 1, 1998, through November 31, 2021 was performed. All cases were diagnosed by clinical assessment, and in some, confirmed by histology. Diagnosis of CAD was confirmed through phototesting in some patients. The latter consisted of phototesting to broadband UVB, UVA, and visible light. The phototesting methods have been described previously.[1]

2.1 | Clinical Assessment

A detailed history was obtained, including age of onset; disease duration; gender; ethnicity; skin phototype (SPT); distribution and natural history of skin condition; seasonal variation; and whether lesions improved or worsened with topical and/or systemic treatment. Morphologic features and distribution of skin lesions were also recorded.

2.2 | Other Relevant Investigations

Routine assessment, in some but not in all patients, included plasma porphyrin assessments, serum levels of antinuclear antibody (ANA), and 25-hydroxyvitamin D levels.

2.3 | Statistical Methods

The data were analyzed using analysis of variance (SAS version 9.4). Data are presented as mean (SD). A χ^2 test was performed when making comparisons among light-skinned and dark-skinned groups. The prevalence of ANA among patients with PMLE was compared to the prevalence of ANA among healthy individuals as published in the study conducted by Satoh and colleagues.[9] Statistical significance was defined at the $p < .05$ level.

3 | RESULTS

3.1 | Polymorphous light eruption

3.1.1 | Demographic characteristics

A total of 844 patients (725 [85.9%] female; 119 [14.1%] male) were diagnosed with PMLE. Patients were aged 9–88 years at diagnosis, with a mean (SD) age of onset of 41.7 (16.9) years and a mean (SD) duration of disease of 11.5 (4.78) years (Table 1). Six hundred twenty-five patients had dark skin types (SPT: IV-VI), and 219 patients had light skin types (SPT: I-III). The population consisted of 533 African Americans, 188 Whites, 42 South Asians, 42 Middle Eastern patients, 18 non-White Hispanics, 10 East Asians, and six patients of mixed ethnicity.

3.1.2 | Clinical assessment

Polymorphous light eruption occurred more frequently in dark-skinned individuals than light-skinned individuals (625 [74%] vs. 219 [25.9%], p value $< .001$).

Dark-skinned individuals were more likely to develop PMLE on the neck (147 [23.5%] vs. 32 [14.6%], p value = .0096) and were more likely to present with pinpoint erythematous papules (346 [41%] vs. 46 [21%], p value $< .0001$), and hyperpigmented lichenified coalesced papules (50 [8%] vs. 4 [1.82%], p value = .0013), compared to light-skinned individuals.

Compared to dark-skinned individuals, light-skinned individuals were more likely to develop PMLE on the lower limbs (thighs and legs) (32 [14.6%] vs. 39 [6.24%], p value = .00012), and were more likely to present with erythematous papules and papulovesicles (66 [30.2%] vs. 22 [3.52%], $p < .0001$), Table 1.

With regards to treatment, a combination of topical corticosteroids and sunscreen was the most frequently prescribed regimen to patients with PMLE (686 [81.3%]), with higher reported use among dark-skinned individuals (524 [83.9%] vs. 162 [73.9%], p value = .0013), compared to light-skinned individuals.

Light-skinned individuals were more likely to be prescribed systemic agents (e.g., oral corticosteroids, methotrexate, 20 [9.2%] vs. 23 [3.4%], p value = .0016), and narrow-band UVB hardening therapy (10 [4.5%] vs. 12 [1.9%], p value = .034), and were more likely to experience lesion resolution (61/67 [91%] vs. 149/209 [71.2%], p value = .022),

TABLE 1 Demographic and clinical characteristics of patients with polymorphous light eruption

Clinical and photobiologic differences	Patient group by Fitzpatrick skin type			
	All ($n = 844$)	(I–III) ($n = 219$)	(IV–VI) ($n = 625$)	p-Value
Age of onset, mean (SD), year	41.7 (16.9)	40.1 (18.8)	42.45 (16.1)	.10
Disease duration, mean (SD), year	11.5 (4.78)	11.7 (4.83)	11.4 (4.2)	.45
Female *N* (%)	725 (85.9)	181 (82.6)	544 (87.0)	.60
Treatment type				
Topical corticosteroids and sunscreen	686 (81.3)	162 (73.9)	524 (83.8)	.0013*
Topical corticosteroids only	78 (9.3)	24 (10.9)	54 (8.64)	.31
Sunscreen only	61 (7.22)	22 (10.0)	39 (6.24)	.79
Treatment success				
Systemic therapy (e.g., corticosteroids, hydroxychloroquine)	43 (5.1)	20 (9.2)	23 (3.7)	.0016*
NB-UVB hardening therapy	22 (2.6)	10 (4.5)	12 (1.9)	.034*
Lesion resolution following one course of treatment, *N* (%)	210/276 (76)	61/67(91)	149/209 (71.3)	.022*
Recurrence following one course of treatment, *N* (%)	93/165 (56.3)	22/44 (50)	71/121 (58.6)	.43
Positive ANA, *N* (%)	112/400 (28)	25/95 (26.3)	87/305 (28.5)	.67
Systemic symptoms				
Systemic pruritus	94 (11.2)	35 (16.0)	59 (9.44)	.33
Arthralgia	23 (2.72)	4 (0.9)	19 (4.0)	.11

Abbreviations: ANA, antinuclear antibody; NB-UVB, Narrow band- ultraviolet B; SD, standard deviation.

*Denotes statistical significance.

compared to dark-skinned individuals. However, there was no significant difference in the recurrence rate between the two groups.

3.1.3 | Laboratory tests

Of 844 patients, 400 (47.35%) had an ANA test, of which 112 had a positive ANA (titers ranging from 1:80 to 1:640). The point prevalence of ANA positivity was 28% (95% CI: 23.6–32.4), significantly higher than the percentage described in healthy US population (13.4%, p value < .001). While the prevalence of positive ANA was higher in dark-skinned individuals compared to light-skinned individuals, this difference was not significant (28.5% vs. 26.3%, p value = .93). Although none of the ANA-positive patients were diagnosed with an autoimmune disorder, 23 (2.72%) patients reported experiencing arthralgias.

3.2 | Chronic actinic dermatitis

3.2.1 | Demographic characteristics

A total of 60 patients (22 [36.6%] female; mean age, SD: 60.6 [10.6] years of age at presentation; disease duration 8.2 [7.3] years) were diagnosed with CAD. Patients ranged in age from 49 to 83 years at diagnosis, with a mean (SD) age at onset of 55.54 (16.2) years, and duration of disease of 6.3 (3.7) years. Forty-three patients had dark skin types and 17 patients had light skin types. This cohort of CAD consisted of 40 African Americans, 16 Whites, two American Indians, one East Asian, and one South Asian. There was no significant difference in the onset (mean [SD] age, 61.7 [13.4] vs. 59.5 [11.4] years, $p > .05$) and duration (8.6 [6.2] vs. 7.9 [3.4] years, $p > .05$) of photosensitivity between dark-skinned and light-skinned individuals respectively.

Atopic dermatitis was the most common comorbidity identified in patients with CAD (22 [36.6%]). Although AD prevalence was found to be higher in light-skinned individuals compared to dark-skinned individuals, this difference was not significant (7 [41%] vs. 15 [34.8%], p value = .46).

3.2.2 | Clinical assessment

Chronic actinic dermatitis is typically manifested as photodistributed eczematous patches and sometimes erythematous and lichenified lesions involving sun-exposed areas (Table 2). Chronic actinic dermatitis was more prevalent in dark-skinned individuals than light-skinned individuals (43 [71.6%] vs. 17 [28.3%], p value = 0.003).

Dark-skinned individuals were more likely to present with CAD on the neck (33 [76.4%] vs. 3 [14.3%], p value = 0.0008) and were more likely to develop lichenified plaques (25 [58.1%] vs. 1 [5.9%], p value = 0.003), compared to light-skinned individuals.

Light-skinned individuals were more likely to present with reticulated erythematous patches (11 [64.7%] vs. 2 [4.65%], p value <0.001), compared to dark-skinned individuals.

Similar to PMLE, combination therapy (sunscreens and topical corticosteroids) was the most commonly prescribed regimen to patients with CAD. No significant difference was observed in the improvement and recurrence rates between light-skinned and dark-skinned individuals (Table 2). Although light-skinned individuals were more likely to be prescribed systemic agents (e.g., mycophenolate mofetil, methotrexate, dupilumab), compared to dark-skinned individuals, this difference was not significant (7/17 [41.2%] vs. 15/43 [34.8%], p value = 0.64).

More than half of patients (40 [66.6%]) reported experiencing moderate to severe pruritus, followed by leg edema (7 [11.6%]), arthralgia and fatigue (4 [1.84%]). No significant difference was noted between the two groups.

3.2.3 | Phototesting

Of the 60 patients with CAD, 22 (36.6%) underwent phototesting with UVA, broadband UVB, and VL. Twenty-two patients had

TABLE 2 Demographic and clinical characteristics of patients with chronic actinic dermatitis

Clinical and photobiologic differences	Patient group by Fitzpatrick skin type			
	All (n = 60)	(I-III) (n = 17)	(IV-VI) (n = 43)	p Value
Age at presentation, mean (SD), year	60.6 (10.6)	59.5 (11.4)	61.7 (13.4)	.14
Onset of photosensitivity, mean (SD), year	55.5 (17.3)	55.5 (17.2)	55.6 (15.4)	.91
Disease duration, mean (SD), year	8.2 (7.3)	7.9 (3.4)	8.6 (6.2)	.83
Duration of onset of photosensitivity, mean (SD), year	6.3 (3.7)	6.4 (3.9)	6.12 (4.2)	.94
Female N (%)	22 (36.6)	3 (17.6)	19 (44.1)	.054
Treatment type				
Use of corticosteroids and sunscreen	37 (61.6)	15 (88.2)	22 (51.2)	.008*
Use of corticosteroids only	1 (2.3)	1(5.9)	0 (0)	.11
Use of sunscreen only	1 (1.66)	0 (0)	1(2.3)	.89
Systemic agents (oral corticosteroids, MTX)	22 (36.6)	7 (41.2)	15 (34.8)	.64
Treatment success				
Lesion resolution following one course of treatment, N (%)	49/55 (89)	17/18 (94.4)	32/37 (86.4)	.37
Recurrence following one course of treatment, N (%)	23/50 (46)	9/18 (50)	14/32 (43.7)	.66
Positive phototesting	22 (36.6)	7 (41.2)	15 (34.8)	.64
Positive for UVA	12 (60)	4 (23.5)	8 (18.6)	.15
Positive for UVB	10 (16.6)	3 (17.6)	7 (16.3)	.17
Positive ANA N (%)	9/22 (36.6)	3/22 (13.6)	6/22 (27.2)	.64
Systemic symptoms, N (%)				
Pruritus	40 (66.6)	14 (82.3)	26 (60.4)	.62
Leg edema	7 (11.6)	3 (17.6)	4 (9.3)	.36
Arthralgia	4 (6.66)	1 (5.9)	3 (6.97)	.33
History of Atopic eczema	22 (36.6)	7 (41.2)	15 (34.8)	.64

Abbreviations: ANA, antinuclear antibody; MTX, methotrexate; SD, standard deviation; UVA, ultraviolet-A; UVB, ultraviolet-B.

*Denotes statistical significance.

a reduced MED value for either UVB (MED-B, $n = 3$, SPT: I-III; $n = 7$, SPT: IV-VI) and/or UVA (MED-A) ($n = 4$, SPT: I-III; $n = 8$, SPT: IV-VI), (Figures 1 and 2). The range of MED-B was between 6 mJ/cm^2 and 110 mJ/cm^2 (Figure 1), while the range of MED-A was 3 J/cm^2 to 18 J/cm^2 (Figure 2). The mean MED-A was similar for dark and light skin types (Figure 1). Visible light phototesting was negative in all patients.

Although dark-skinned individuals were more likely to have a reduced MED-B compared to light-skinned individuals, this difference was not significant (p value = .43).

3.2.4 | Laboratory findings

Plasma and urine porphyrin levels were evaluated in 20 out of 60 patients. None of these patients had elevations in plasma and/or urine porphyrins. Vitamin D status was assessed in 20 (33.3%) patients, of which two had low levels of vitamin D (13 ng/ml in one light-skinned patient, and 22 ng/ml in one dark-skinned individual).

ANA testing was performed in 22 out of 60 (47.35%) patients. Nine had a positive ANA (3 [17.6%] with skin types I-III; 6 [13.9%] with skin types IV-VI, p value = .64), with titers ranging from 1:160 to 1:320.

4 | DISCUSSION

In this large population cohort study seen over 23 years, we highlighted the differences in demographic distribution and clinical features in patients with light and dark skin types who presented with PMLE and CAD.

Although skin phototype has historically been associated with the likelihood of developing PMLE, with type I posing the highest risk and type IV (or higher) posing the lowest risk, we found that PMLE occurs significantly more frequently among dark-skinned individuals (SPT IV–VI) than light-skinned individuals (SPT I–III). This finding is consistent with recently published reports highlighting the increased prevalence of PMLE in dark-skinned individuals.[3,6,10]

Of the total dermatology clinic patients seen in our department between 2013 and 2022 (total of 257,952 patients) with self-identified race/ethnicity, over half of patients (57.4%) were White, 32.3% were Black, and 10.3% were of other races/ethnicity (e.g., East Asian, South Asian, Hispanic, multiracial, and Middle Eastern). Although more than half of the general clinic population was White, the percentages of skin of color patients with PMLE (74.0%) and CAD (71.6%) were significantly higher, supporting the increased prevalence in dark-skinned individuals, particularly African Americans.

While PMLE shows high variability in its clinical characteristics and severity, the pinpoint-papular variant of PMLE (PP-PMLE) was the most common morphology identified in dark-skinned individuals, accounting for 35.6% of all cases. PP-PMLE is considered a distinct entity that has been primarily described in individuals with skin types IV-VI.[11] Clinical manifestation of PP-PMLE may vary based on disease stage. During the acute phase, lesions manifest as pinpoint papules, and less commonly, vesicles, on exposed areas, occurring within a few hours of sun exposure.[10] Patients frequently present with shiny pinhead macules as the acute lesions resolve. In contrast, papular or urticarial lesions in light-skinned PMLE patients resolve without any residual lesions. Although PP-PMLE may be mistaken for lichen nitidus, the latter is not associated with sun exposure, usually asymptomatic, and tends to be persistent. Unlike lichen nitidus, PMLE is a seasonal photodermatosis occurring in spring and early summer.

Previous studies have shown elevated titers of ANA in 2.9%–19% of patients with PMLE.[12–16] In our cohort, the prevalence of ANA positivity was 28%, with higher rates observed in dark-skinned individuals. Due to some shared immune features with SLE (e.g., high levels of interleukin-1, IL-1, and TNF alpha), it has been suggested that PMLE may precede the development of SLE. However, despite the increased prevalence of ANA, and a follow-up period of at least 2 years, none of the ANA-positive patients identified in our cohort developed SLE. Comparable findings were observed in a retrospective study involving 55 PMLE patients who had a positive ANA test. After a median follow-up of 8 years, none of the patients developed SLE.[16]

PMLE remains a challenging disease to manage. It is noteworthy that in this study, light-skinned individuals were more likely to experience lesion resolution compared to dark-skinned individuals. Although we did not control for several confounders, the variability in this finding may be attributed to the higher number of light-skinned individuals being prescribed systemic therapy compared to dark-skinned individuals. The underuse of systemic therapies, including NB-UVB, among dark-skinned individuals suggest that health disparities (e.g., insurance coverage, accessibility to transportation, ability to take time off for clinic visits, phototherapy, etc.) may exist in the care of patients with PMLE. As such, it is important to understand the reasons for these potential disparities and implement interventions to ensure equitable care for patients with PMLE.

Despite light-skinned individuals having higher rates of lesion resolution, most patients experienced lesion recurrence, with no significant difference in the recurrence rate between the two groups. The prolonged and relapsing course of this condition has been highlighted in a recent study involving 97 PMLE patients. Though a majority of these patients experienced improvement, it took 25 years until one third of patients had resolution of PMLE.[17]

Compared to PMLE, fewer studies have examined racial differences in CAD epidemiology. In our study, we found that the prevalence of CAD was higher in dark-skinned individuals than light-skinned individuals. This finding is consistent with a recently published study highlighting the increased prevalence of CAD in the skin of color population.[8] Other clinical and photobiological characteristics in our patients with CAD were consistent with what has been reported in the literature: most of our patients were older men, presented with eczematous eruptions in sun-exposed areas, and had reduced MEDs to UVB and UVA.

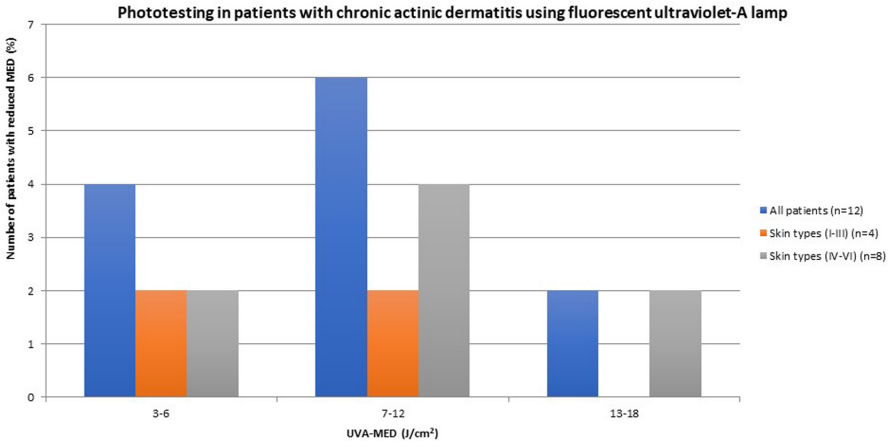

FIGURE 1 Phototesting in patients with chronic actinic dermatitis using fluorescent ultraviolet A lamp

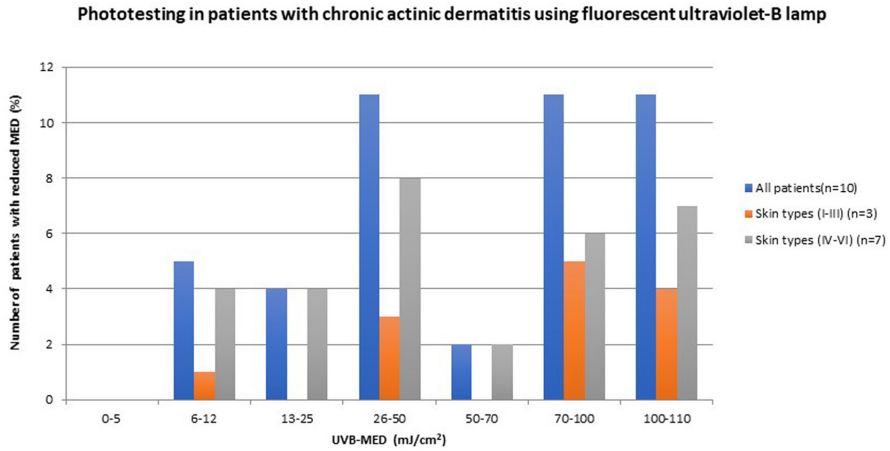

FIGURE 2 Phototesting in patients with chronic actinic dermatitis using fluorescent ultraviolet B lamp

Similar to PMLE, a third of our cohort of CAD patients were seropositive for ANA. While pruritus, joint pain, and joint swelling were commonly reported, none of the ANA-positive patients developed SLE over a follow-up period of 9 years. With regards to treatment, most patients experienced partial to complete lesion resolution following the use of sunscreens and topical corticosteroids, but nearly half experienced recurrence.

Although the exact mechanism remains unclear, exposure to UVR plays a critical role in the pathogenesis of PMLE and CAD.[18] The failure of normal UVR-induced immunosuppression appears to be the main immunological abnormality in PMLE. This is supported by the persistence of epidermal Langerhans cells following UVB irradiation.[19] In addition, PMLE is a delayed-type hypersensitivity reaction to an unknown antigen.[20] It has been suggested that UVR exposure, in genetically susceptible individuals, induces a cutaneous reaction that leads to the formation of a photoantigen, which triggers a robust immune response.[20]

Like PMLE, delayed type hypersensitivity reactions to UVR-induced skin photoantigen may play a role in CAD pathogenesis.[5] An immunologic response to contact allergens (e.g., colophony, fragrances, sunscreen) has also been shown to play a role in disease pathogenesis, particularly in dark-skinned individuals.[21–23] In previously published reports, positive photopatch test reactions to avobenzone and oxybenzone (organic UVR filters) were commonly observed in dark-skinned patients with CAD.[22,23]

5 | LIMITATIONS

Limitations include the retrospective nature of this study. Given that this study was conducted at a single-center institution, these results may not be generalizable. Not all patients with suspected CAD underwent phototesting; most commonly due to lack of insurance coverage. As such, the diagnosis of CAD was largely made by clinical and histologic assessments. Although a diagnosis of PMLE is generally based on the clinical examination and medical history, phototesting, especially photoprovocation testing, can be helpful in confirming the diagnosis, and determining the action spectrum of the disease.

6 | CONCLUSION

Our retrospective study of 844 patients seen over 23 years found that a substantial proportion of PMLE and CAD cases present in dark-skinned individuals, and that PMLE predominantly occurs in younger female patients with dark skin. PP-PMLE represents a unique variant of PMLE that appears to primarily affect dark-skinned individuals, particularly African Americans. As PP-PMLE can be mistaken for lichen nitidus, recognition of this variant is important for appropriate evaluation and management of these patients. In this analysis, we also demonstrated that both PMLE and CAD follow a chronic course with high rates of recurrence following lesion resolution. Given that

these two conditions pose therapeutic challenges, there is a need to improve our understanding of the molecular and immunologic basis of PMLE and CAD to develop effective and targeted therapies.

IRB APPROVAL

Study was approved by the Henry Ford Health Institutional Review Board.

CONFLICT OF INTEREST

Dr. Mohammad is an investigator for Clinuvel, Incyte Corporation, Pfizer, Avita, Arcutis, Pierre Fabre, Estee Lauder, Unigen Inc., Ferndale Healthcare Inc., and Allergan. Dr. Lim has served as an investigator for Incyte, L'Oreal, Pfizer, and PCORI; he has also served as a consultant for Pierre Fabre, ISDIN, Ferndale Pharma Group, La Roche-Posay, and Beiersdorf, and has participated as a speaker in an educational session for Pierre Fabre, La Roche-Posay, and Bioderma. JM and LM have no relevant conflicts to disclose.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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REFERENCES

1. Yashar SS, Lim HW. Classification and evaluation of photodermatoses. *Dermatol Ther*. 2003;16(1):1-7.
2. Lew R, Jacob J. Polymorphous light eruption: a common skin disease uncommonly recognized in the Hispanic population. *Oxf med Case Reports*. 2014;2014(8):145-147.
3. Hamel R, Mohammad TF, Chahine A, et al. Comparison of racial distribution of photodermatoses in USA academic dermatology clinics: a multicenter retrospective analysis of 1080 patients over a 10-year period. *Photodermatol Photoimmunol Photomed*. 2020;36(3):233-240.
4. Gruber-Wackernagel A, Byrne SN, Wolf P. Polymorphous light eruption: clinic aspects and pathogenesis. *Dermatol Clin*. 2014;32(3):315-334. viii.
5. Dawe RS, Ferguson J. Diagnosis and treatment of chronic actinic dermatitis. *Dermatol Ther*. 2003;16(1):45-51.
6. Dawe RS, Crombie IK, Ferguson J. The natural history of chronic actinic dermatitis. *Arch Dermatol*. 2000;136(10):1215-1220.
7. Lim H, Hawk J, Rosen C. Photodermatoses. In: Bolognia J, Schaffer J, Cerroni L, eds. *Dermatology*. Elsevier; 2018:1548-1568.
8. Tan K-W, Haylett AK, Ling TC, Rhodes LE. Comparison of demographic and Photobiological features of chronic actinic dermatitis in patients with lighter vs darker skin types. *JAMA Dermatol*. 2017;153(5):427-435.
9. Satoh M, Chan EKL, Ho LA, et al. Prevalence and sociodemographic correlates of antinuclear antibodies in the United States. *Arthritis Rheum*. 2012;64(7):2319-2327.
10. Kontos AP, Cusack CA, Chaffins M, Lim HW. Polymorphous light eruption in African Americans: pinpoint papular variant. *Photodermatol Photoimmunol Photomed*. 2002;18(6):303-306.
11. Bansal I, Kerr H, Janiga JJ, et al. Pinpoint papular variant of polymorphous light eruption: clinical and pathological correlation. *J Eur Acad Dermatol Venereol*. 2006;20(4):406-410.
12. Epstein JH. Polymorphous light eruption. *Dermatol Clin*. 1986;4(2):243-251.
13. Jansén CT, Karvonen J. Polymorphous light eruption: a seven-year follow-up evaluation of 114 patients. *Arch Dermatol*. 1984;120(7):862-865.
14. Mastalier U, Kerl H, Wolf P. Clinical, laboratory, phototest and phototherapy findings in polymorphic light eruptions: a retrospective study of 133 patients. *Eur J Dermatol*. 1998;8(8):554-559.
15. Hasan T, Ranki A, Jansen CT, Karvonen J. Disease associations in polymorphous light eruption. A long-term follow-up study of 94 patients. *Arch Dermatol*. 1998;134(9):1081-1085.
16. Tzaneva S, Volc-Platzer B, Kittler H, Hönigsmann H, Tanew A. Antinuclear antibodies in patients with polymorphic light eruption: a long-term follow-up study. *Br J Dermatol*. 2008;158(5):1050-1054.
17. Gruber-Wackernagel A, Schug T, Graier T, et al. Long-term course of polymorphic light eruption: a registry analysis. *Front med*. 2021;8:694281.
18. Lee CN, Chen TY, Wong TW. The Immunogenetics of photodermatoses. *Adv Exp med Biol*. 2022;1367:369-381.
19. Kölgen W, Both H, Van Weelden H, et al. Epidermal Langerhans cell depletion after artificial ultraviolet B irradiation of human skin In vivo: apoptosis versus migration. *J Invest Dermatol*. 2002;118(5):812-817.
20. Guarrera M. Polymorphous light eruption. *Adv Exp med Biol*. 2017;996:61-70.
21. Hawk JL. Chronic actinic dermatitis. *Photodermatol Photoimmunol Photomed*. 2004;20(6):312-314.
22. Que SK, Brauer JA, Soter NA, Cohen DE. Chronic actinic dermatitis: an analysis at a single institution over 25 years. *Dermatitis*. 2011;22(3):147-154.
23. Russo JP, Ipiña A, Palazzolo JF, Cannavó AB, Piacentini RD, Niklasson B. Photoallergic contact dermatitis to sunscreens containing Oxybenzone in La Plata, Argentina. *Actas Dermosifiliogr (Engl Ed)*. 2018;109(6):521-528.

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