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CASE REPORT

Isolated Actinic Prurigo Cheilitis in an Elderly Thai Female: An Atypical Presentation

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Abstract: Actinic prurigo (AP) is a rare photodermatosis with variable clinical presentations across different ethnic populations. While lip involvement is common in Latin American populations, isolated cheilitis as the sole manifestation in elderly Asian patients has not been previously documented. We report a 67-year-old Thai female presenting with chronic, recurrent painful ulcers on the lower lip persisting for six months. Despite multiple treatments at a private clinic, new lesions continued to develop. Dermatological examination revealed multiple erosions on an erythematous, edematous plaque at the vermilion border of the lower lip. Histopathological examination demonstrated dense lymphocytic infiltration with well-defined lymphoid follicles, demonstrating the histopathological pattern of follicular cheilitis, which is pathognomonic for actinic prurigo cheilitis. Interestingly, phototesting and photoprovocation yielded normal results across all UV spectra. Complete remission was achieved after two months of treatment with topical 1.25% hydrocortisone cream combined with strict sun protection measures. This case highlights an unusual phenotype of actinic prurigo in an Asian patient, characterized by isolated lip involvement in the absence of other cutaneous manifestations. It underscores the importance of considering AP in the differential diagnosis of chronic lip lesions, even in atypical demographics and with normal phototesting results. The favorable response to conservative management emphasizes the value of early recognition and appropriate treatment.

Keywords: cheilitis, follicular cheilitis, isolated lip lesion, photodermatosis, photo-induced dermatoses, solar prurigo

Introduction

Actinic prurigo (AP) is an idiopathic photodermatosis characterized by abnormal inflammatory responses to sunlight exposure.[1] The disease demonstrates marked geographic variation, with highest prevalence in Mestizo and Native American populations in high-altitude Latin American regions.[2] Emerging reports suggest more diverse clinical phenotypes, particularly in Asian populations.[3,4]

Lip involvement in AP, termed AP cheilitis, typically accompanies other sun-exposed skin lesions and represents a hallmark feature in Latin American cohorts.[5] Histologically, AP cheilitis exhibits well-organized lymphoid follicles within the dermis, a pattern termed follicular cheilitis, which is considered pathognomonic for this condition.[6] However, isolated lip lesions as the sole manifestation of AP remain exceptionally rare, particularly in elderly Asian patients. Herein, we present AP cheilitis in a 67-year-old Thai female, representing an atypical presentation that broadens our understanding of this photodermatosis.

Case Presentation

A 67-year-old Thai female presented with a six-month history of chronic, recurrent painful lower lip erosions. Despite treatment at multiple clinics with topical and oral medications, only partial improvement occurred, with new lesions continuing to develop. Her medical history included hypertension and hyperlipidemia, well-managed with amlodipine, simvastatin, and losartan for approximately 6 years. No recent exposure to new medications or cosmetic products was

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reported. She denied food or drug allergies but reported extensive sun exposure during younger years. No family history of photosensitivity disorders existed.

Dermatological examination revealed multiple erosions on an erythematous, edematous plaque localized to the lower lip vermilion border, without intraoral mucosal involvement (Figure 1). Other cutaneous examination including sun-exposed areas (ie, face, neck, dorsal hands, forearms) revealed no additional cutaneous lesions. Conjunctivae appeared normal bilaterally. Initial differential diagnoses included actinic cheilitis, discoid lupus erythematosus, erosive lichen planus, and contact cheilitis.

A 4-mm punch biopsy from lower lip demonstrated epidermal acanthosis, spongiosis and parakeratosis. The lamina propria exhibited a diffuse lymphoplasmacytic infiltrate with well-formed lymphoid follicles and congested blood vessels (Figure 2). These findings were consistent with follicular cheilitis, a characteristic feature of AP cheilitis.[6] Phototesting showed minimal erythema doses (MED) for UVA, UVB, and visible light within normal limits. Photoprovocation testing across the entire UV spectrum yielded negative results. The patient received topical 1.25% hydrocortisone cream twice daily with comprehensive photoprotection measures, including broad-spectrum sunscreen (SPF 50+ with UVA and UVB protection), photoprotective lip balm (SPF 30+, reapplied after eating/drinking), and wide-brimmed hats. At two-month follow-up, complete remission was documented (Figure 3). The patient reported no new lesions with good photoprotection adherence and remained asymptomatic at six-month follow-up.

Discussion

AP demonstrates remarkable geographic and ethnic variability. The disease predominantly affects individuals in high-altitude regions (>1000 meters above sea level) with intense UV exposure.[2] Prevalence of AP ranges from 0.003% - 8%,[7] with highest rates in Latin American Mestizo populations[5,8] and Native Americans (Amerindians).[9,10] While initially considered rare outside Latin America, increasing Asian and European case reports have expanded understanding of global distribution.[3,11–15] Classic cases typically manifest in the first decade with chronic, relapsing course and female predominance.[2,7,10] Adult-onset disease, as in our patient, represents an increasingly recognized variant.

Clinical manifestations of AP are polymorphic, characterized by intensely pruritic erythematous papules coalescing into plaques on sun-exposed areas,[1,5] with secondary excoriations, lichenification, and eczematization. Lesions typically affect the face, neck, anterior chest, extensor forearms, and dorsal hands symmetrically.[2,10] Lips and conjunctival involvement commonly occur in Mestizo and Native American populations.[5,8,16] Remarkably, lip lesions may

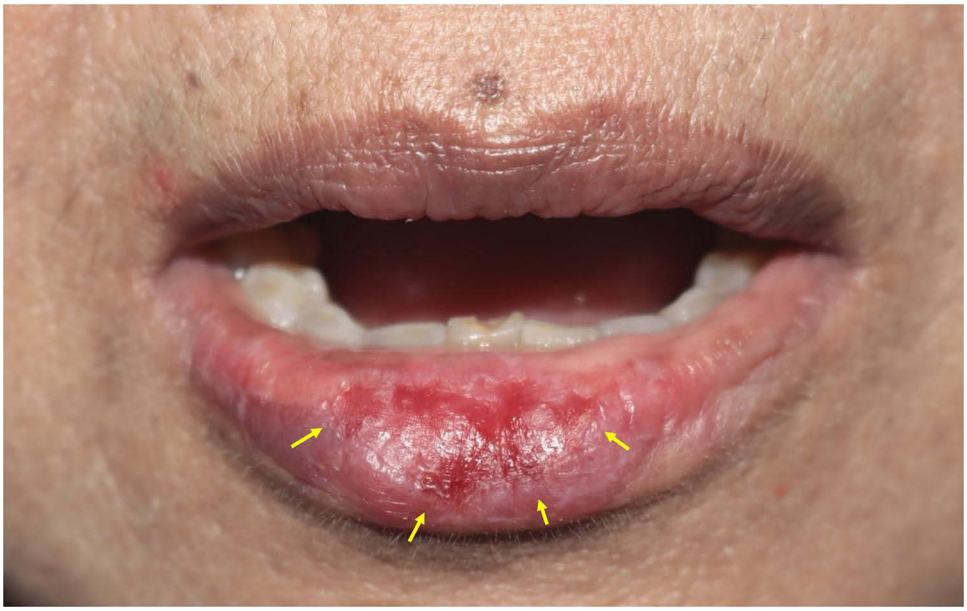

Figure 1 Clinical presentation at baseline showing multiple erosions on an erythematous, edematous plaque at the lower lip (arrows).

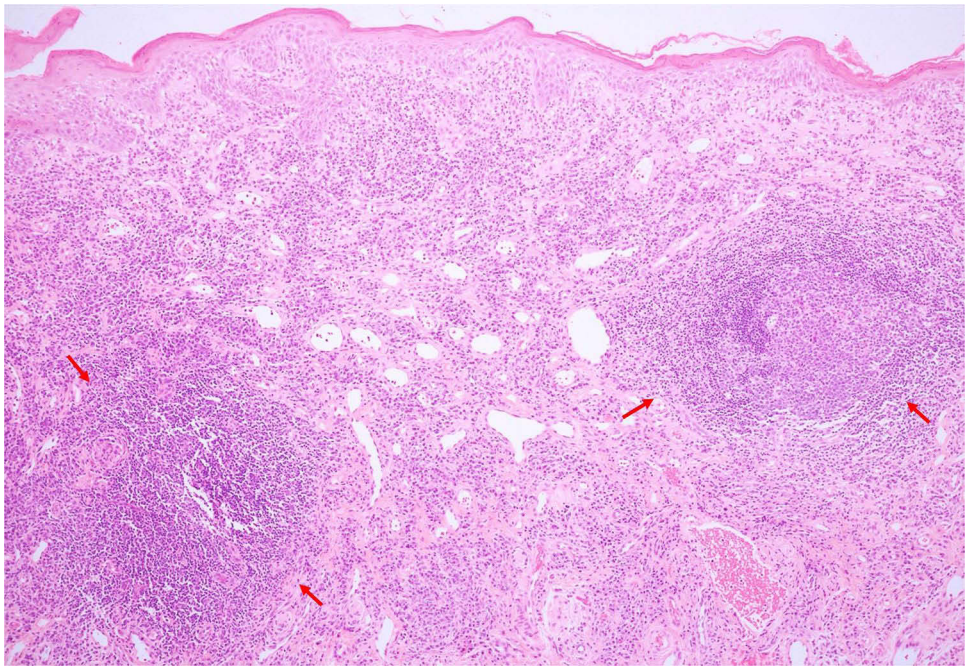

Figure 2 A 4-mm punch biopsy obtained from the lower lip demonstrated epidermal acanthosis, spongiosis, and parakeratosis. The underlying lamina propria showed a diffuse lymphoplasmacytic infiltrate with well-formed lymphoid follicles and congested blood vessels. (hematoxylin-eosin at 100x magnification). Arrows highlight the lymphoid follicles.

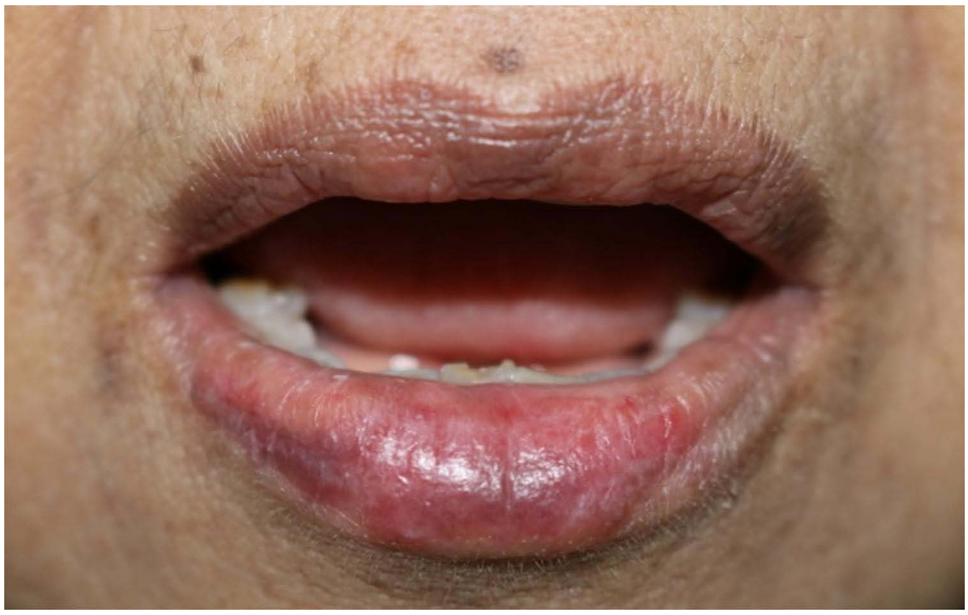

Figure 3 Two-month follow-up demonstrating complete resolution following treatment with topical 1.25% hydrocortisone cream and photoprotection measures.

occasionally represent the sole manifestation, as demonstrated in our case.[5,8,17,18] Asian populations demonstrate significant phenotypic differences from that observed in Latin American cohorts. Asian patients more frequently present with male predominance and later age of onset. Lip and conjunctival involvement are notably uncommon.[3,12–14] To our knowledge, our patient represents the first Asian case with isolated lip involvement. Table 1 summarizes previously reported cases of AP cheilitis.[5,6,8,17–19]

Table 1 Characteristics of Actinic Prurigo Cheilitis in Previous Literature

No.	Authors, Year	Population	Age at Onset	Sex Predominance	Number of Cases	Number of Cases with Isolated Lip Involvement
1	Mounsdon et al, 1988[17]	USA	61 and 69 years	Female	5	2 (40%)
2	Herrera-Geopfert and Magaña, 1995[6]	Mexico	8-66 years	Female	57	9 (15.7%)
3	Vega-Memije et al, 2002[8]	Mexico	9-82 years	Female	116	32 (27.6%)
4	Miranda et al, 2014[18]	Brazil	63 and 58 years	Female	2	2 (100%)
5	Plaza et al, 2016[5]	Multi-center	3-67 years	Female	75	42 (56%)
6	Acuña et al, 2018[19]		25 years	Female	1	1 (100%)
7	Current case	Thailand	67 years	Female	1	1 (100%)

AP cheilitis clinically presents with scaling, fissures, erythema, edema, and ulceration, mimicking actinic cheilitis.[19] Histologically, characteristic features include acanthosis with spongiosis, basal cell vacuolation, and dense lymphocytic infiltrate forming well-defined follicles.[6] These follicles exhibit T cells at the periphery and B cells within germinal centers. This pattern, designated follicular cheilitis, represents the pathognomonic feature of AP cheilitis.[6] Variable eosinophils and macrophages may be present at the epithelial-connective tissue interface, though sensitivity and specificity are only 75% and 36.4%, respectively.[5,6] Differential diagnoses for isolated lip lesions include actinic cheilitis, granulomatous cheilitis, contact cheilitis, erosive lichen planus, and discoid lupus erythematosus.[5] Actinic cheilitis shows chronic sun damage and lacks lymphoid follicles, while erosive lichen planus features Wickham striae and interface dermatitis. Discoid lupus erythematosus presents with interface dermatitis and mucin deposition. Contact cheilitis has an acute onset linked to allergens, and granulomatous cheilitis displays persistent edema with non-caseating granulomas. The presence of lymphoid follicles in the biopsy confirmed AP cheilitis, ruling out these alternatives. Key distinguishing characteristics of these conditions are summarized in Table 2.[20–22]

Table 2 Differential Diagnosis of Follicular Cheilitis and Disease Characteristics

Condition	Clinical Features	Histopathological Features	Key Distinguishing Features
Actinic prurigo cheilitis	Erosions, scaling, edema; recurrent; sun exposure history	Lymphoid follicles, spongiosis, plasma cells	Follicular cheilitis pattern; positive family history
Actinic cheilitis	Scaling, atrophy, white plaques; lower lip predominance	Solar elastosis, dysplasia (variable degrees)	Chronic sun damage; risk for malignant transformation
Erosive lichen planus	Wickham striae, erosions, pain; oral involvement common	Interface dermatitis, colloid bodies, band-like infiltrate	Reticular pattern; oral mucosal involvement
Discoid lupus erythematosus	Erythema, scaling, atrophy, hyperpigmentation	Interface dermatitis, deep perivascular infiltrate, mucin	Systemic features possible; positive ANA
Contact cheilitis	Acute onset, pruritus, history of allergen exposure	Spongiosis, superficial perivascular infiltrate	Clear temporal relationship with contactant
Granulomatous cheilitis	Persistent swelling, fissuring; may be part of Melkersson-Rosenthal syndrome	Non-caseating granulomas	Persistent edema; facial nerve involvement in Melkersson-Rosenthal syndrome

The pathogenesis of AP involves type IV hypersensitivity to UV radiation, with both Th1 and Th2 responses.[23] UV-stimulated keratinocytes produce tumor necrosis factor (TNF)-α, while Th1 cells release proinflammatory cytokines and Th2 cells contribute interleukin (IL)-4, IL-5, and recruit mast cells and eosinophils.[23–26] Consequently, the intrinsic apoptotic pathway implicates the final step of the reaction.[24,27] Torres-Alvarez et al reported persistent epidermal Langerhans cell counts following UV exposure in AP-affected individuals, contrasting with typical decreases in healthy controls, suggesting Langerhans cell activation.[28]

Several risk factors have been implicated in disease pathogenesis. While UV exposure remains the most extensively studied environmental trigger, genetic susceptibility plays a critical role.[29,30] Strong genetic associations exist with HLA-DR4 (particularly DRB1*0407) in Mexican populations,[31] HLA-DRB1*14 in Canadian populations,[9] and HLA-DRB1*03:01 in Asian cohorts.[4,12]

Phototesting in AP may yield variable results, with a reduced or normal MED.[1,14,32] Our patient demonstrated normal phototesting and photoprovocation across the entire UV spectrum, in contrast to typically positive photoprovocation in previous reports.[13,14] These findings suggest that AP should remain in the differential diagnosis even when phototesting is negative, provided the histopathologic features are characteristic.

Although individuals with skin of color possess higher baseline melanin levels providing inherent photoprotection, they remain susceptible to photodermatoses such as AP. Asian populations have an estimated natural SPF of 13.4, which is insufficient to prevent chronic UV-induced damage.[33–35] The lips present unique challenges due to lower melanin density, environmental exposure, and frequent disruption of applied photoprotective agents through eating and drinking, necessitating particular attention in AP cheilitis management. For our patient, we emphasized the critical importance of comprehensive photoprotection tailored to her skin type and specific anatomical involvement. We also counseled the patient that consistent photoprotection was essential to prevent disease recurrence, as UV radiation can trigger AP regardless of baseline skin pigmentation.[36]

Strict photoprotection constitutes the cornerstone of AP management, including protective clothing, wide-brimmed hats, sunglasses, and broad-spectrum sunscreen.[5] Thalidomide remains the most effective treatment due to selective TNF-α inhibition and suppression of Langerhans cell antigen-presenting capacity.[37–40] Notably, therapeutic response to thalidomide may serve as a diagnostic indicator for AP; however, its use is limited by significant teratogenicity and requires strict contraceptive measures and monitoring protocols. Topical and systemic corticosteroids effectively control pruritus and eczematous lesions during acute exacerbations.[2,5,41] Alternative therapeutic options include antimalarials, calcineurin inhibitors, janus kinase inhibitors, and phototherapy.[2,42]

AP typically follows a chronic, relapsing course with seasonal exacerbations.[2,7] While childhood-onset disease may improve after puberty in some patients, adult-onset cases tend to demonstrate a more persistent trajectory.[2,3,14] Lip involvement, as seen in our patient, presents particular therapeutic challenges due to the difficulty of achieving complete photoprotection in this anatomical site and the chronic nature of cheilitis.[8,43] However, with rigorous photoprotection and appropriate anti-inflammatory therapy, symptomatic control can be achieved. Long-term follow-up is recommended to monitor for recurrence and ensure continued adherence to photoprotective measures.[7]

Conclusion

Our patient represents a rare case of isolated AP cheilitis in a 67-year-old Asian female. This case highlights several atypical features: isolated labial involvement without other cutaneous lesions, adult onset in an elderly patient, occurrence in an Asian population where AP is less commonly reported, and negative phototesting despite characteristic histopathology. Reporting such cases enhances clinical awareness and underscores the importance of considering AP cheilitis in the differential diagnosis of chronic lip erosions, even when phototesting is negative and the patient's demographic profile is atypical.

Ethics Approval and Consent to Participate

This article was performed in accordance with the principles of Declaration of Helsinki. Ethical approval was exempted by the Ethics Committee of the Faculty of Medicine, Ramathibodi Hospital, Mahidol University. The patient provided explicit consent for the use of clinical photographs and histopathological images for educational and research purposes. Patient anonymity has been preserved throughout this paper, and no identifying information has been disclosed.

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References

1. Crouch R, Foley P, Baker C. Actinic prurigo: a retrospective analysis of 21 cases referred to an Australian photobiology clinic. *Australas J Dermatol*. 2002;43(2):128–132. doi:10.1046/j.1440-0960.2002.00574.x
2. Hojyo-Tomoka MT, Vega-Memije ME, Cortes-Franco R, Domínguez-Soto L. Diagnosis and treatment of actinic prurigo. *Dermatol Ther*. 2003;16(1):40–44. doi:10.1046/j.1529-8019.2003.01606.x
3. Ker KJ, Chong WS, Theng CT. Clinical characteristics of adult-onset actinic prurigo in Asians: a case series. *Indian J Dermatol Venereol Leprol*. 2013;79(6):783–788. doi:10.4103/0378-6323.120726
4. Chen Q, Shen M, Heng YK, et al. Actinic Prurigo in Singaporean Chinese: a positive association with HLA-DRB1*03:01. *Photochem Photobiol*. 2016;92(2):355–359. doi:10.1111/php.12569
5. Plaza JA, Toussaint S, Prieto VG, et al. Actinic Prurigo Cheilitis: a clinicopathologic review of 75 cases. *Am J Dermatopathol*. 2016;38(6):418–422. doi:10.1097/dad.0000000000000459
6. Herrera-Geopfert R, Magaña M. Follicular cheilitis. A distinctive histopathologic finding in actinic prurigo. *Am J Dermatopathol*. 1995;17(4):357–361. doi:10.1097/00000372-199508000-00009
7. Valbuena MC, Muvdi S, Lim HW. Actinic prurigo. *Dermatol Clin*. 2014;32(3):335–44,viii. doi:10.1016/j.det.2014.03.010
8. Vega-Memije ME, Mosqueda-Taylor A, Irigoyen-Camacho ME, Hojyo-Tomoka MT, Domínguez-Soto L. Actinic prurigo cheilitis: clinicopathologic analysis and therapeutic results in 116 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2002;94(1):83–91. doi:10.1067/moe.2002.123539
9. Wiseman MC, Orr PH, Macdonald SM, Schroeder ML, Toole JW. Actinic prurigo: clinical features and HLA associations in a Canadian Inuit population. *J Am Acad Dermatol*. 2001;44(6):952–956. doi:10.1067/mjd.2001.112579
10. Hojyo-Tomoka T, Vega-Memije E, Granados J, et al. Actinic prurigo: an update. *Int J Dermatol*. 1995;34(6):380–384. doi:10.1111/j.1365-4362.1995.tb04435.x
11. Rattanakaemakorn P, Suchonwanit P. Scalp pruritus: review of the pathogenesis, diagnosis, and management. *Biomed Res Int*. 2019;2019:1268430. doi:10.1155/2019/1268430
12. Chen YA, Yang CC, Ting SW, Lee JY, Chen W. Adult-onset actinic prurigo: report of 19 patients from Taiwan. *J Eur Acad Dermatol Venereol*. 2016;30(11):e140–e142. doi:10.1111/jdv.13450
13. Akaraphanth R, Sindhavananda J, Gritiyarangsan P. Adult-onset actinic prurigo in Thailand. *Photodermatol Photoimmunol Photomed*. 2007;23(6):234–237. doi:10.1111/j.1600-0781.2007.00316.x
14. Sangmala S, Wongpraparut C, Silpa-Archa N, Chaiyabutr C. Exploring adult-onset actinic prurigo in Thailand. *Dermatol Reports*. 2024;16(3):9810. doi:10.4081/dr.2023.9810
15. Chanprapaph K, Sutharaphan T, Suchonwanit P. Scalp biophysical characteristics in males with androgenetic alopecia: a comparative study with healthy controls. *Clin Interv Aging*. 2021;16:781–787. doi:10.2147/cia.S310178
16. Magaña M, Mendez Y, Rodriguez A, Mascott M. The conjunctivitis of solar (actinic) prurigo. *Pediatr Dermatol*. 2000;17(6):432–435. doi:10.1046/j.1525-1470.2000.01818.x
17. Mounsdon T, Kratochvil F, Auclair P, Neale J, Lee L. Actinic prurigo of the lower lip. Review of the literature and report of five cases. *Oral Surg Oral Med Oral Pathol*. 1988;65(3):327–332. doi:10.1016/0030-4220(88)90117-x
18. Miranda AM, Ferrari TM, Werneck JT, Junior AS, Cunha KS, Dias EP. Actinic prurigo of the lip: two case reports. *World J Clin Cases*. 2014;2(8):385–390. doi:10.12998/wjcc.v2.i8.385
19. Acuña PH, Pardo AC, Roa MA. Recurrent painful lesions on the lower lip. *Indian J Dermatol Venereol Leprol*. 2018;84(2):179–181. doi:10.4103/ijdvl.IJDVL_430_16
20. Chanprapaph K, Pomsoong C, Tankunakorn J, Eden C, Suchonwanit P, Rutnin S. Comparative analyses of clinical features, histopathology, and CD123 immunohistochemistry of oral lupus erythematosus, lichen planus, and other lichenoid lesions. *Dermatology*. 2022;238(3):464–475. doi:10.1159/000517971
21. Narayanan D, Rogge M. Cheilitis: a diagnostic algorithm and review of underlying etiologies. *Dermatitis*. 2024;35(5):431–442. doi:10.1089/derm.2023.0276
22. Phuwaraks K, Rutnin S, Suchonwanit P. Lichen sclerosus of the labial mucosa: a case report and literature review. *Clin Cosmet Invest Dermatol*. 2024;17:253–258. doi:10.2147/ccid.S448367
23. Vega Memije ME, Cuevas Gonzalez JC, Hojyo-Tomoka MT, Rodríguez Lobato E. Actinic prurigo as a hypersensitivity reaction type 4. *Int J Dermatol*. 2017;56(6):e135–e136. doi:10.1111/ijd.13550
24. Cuevas-González JC, Vega-Memíje ME, García-Vázquez FJ, Rodríguez-Lobato E, Farfán-Morales JE. Apoptosis and apoptotic pathway in actinic prurigo by immunohistochemistry. *Med Oral Patol Oral Cir Bucal*. 2016;21(1):e53–8. doi:10.4317/medoral.20765
25. Chanprapaph K, Mahasaksiri T, Kositkuljorn C, Leerunyakul K, Suchonwanit P. Prevalence and risk factors associated with the occurrence of autoimmune diseases in patients with alopecia areata. *J Inflamm Res*. 2021;14:4881–4891. doi:10.2147/jir.S331579
26. Rattananukrom T, Suchonwanit P. Are drug treatment strategies really effective against alopecia areata? *Expert Opin Pharmacother*. 2021;22(3):257–260. doi:10.1080/14656566.2020.1854728

27. Suchonwanit P, Leerunyakul K, Kositkuljorn C. Diagnostic and prognostic values of cutaneous manifestations in COVID-19. *Dermatol Ther*. 2020;33(4):e13650. doi:10.1111/dth.13650
28. Torres-Alvarez B, Baranda L, Fuentes C, et al. An immunohistochemical study of UV-induced skin lesions in actinic prurigo. Resistance of Langerhans cells to UV light. *Eur J Dermatol*. 1998;8(1):24–28.
29. Cuevas-Gonzalez JC, Vega-Memíje ME, Borges-Yáñez SA, Rodríguez-Lobato E. Risk factors associated with actinic prurigo: a case control study. *An Bras Dermatol*. 2017;92(6):774–778. doi:10.1590/abd1806-4841.20175105
30. Vera Izaguirre DS, Zuloaga Salcedo S, González Sánchez PC, et al. Actinic prurigo: a case-control study of risk factors. *Int J Dermatol*. 2014;53(9):1080–1085. doi:10.1111/ijd.12168
31. Grabczynska SA, McGregor JM, Kondeatis E, Vaughan RW, Hawk JLM. Actinic prurigo and polymorphic light eruption: common pathogenesis and the importance of HLA-DR4/DRB1*0407. *Br J Dermatol*. 1999;140(2):232–236. doi:10.1046/j.1365-2133.1999.02654.x
32. Grabczynska SA, Hawk JL. What is actinic prurigo in Britain? *Photodermatol Photoimmunol Photomed*. 1997;13(3):85–86. doi:10.1111/j.1600-0781.1997.tb00119.x
33. Lim HW, Arellano-Mendoza MI, Stengel F. Current challenges in photoprotection. *J Am Acad Dermatol*. 2017;76(3 Suppl 1):S91–S99. doi:10.1016/j.jaad.2016.09.040
34. Krutmann J, Bouloc A, Sore G, Bernard BA, Passeron T. The skin aging exposome. *J Dermatol Sci*. 2017;85(3):152–161. doi:10.1016/j.jdermsci.2016.09.015
35. Brenner M, Hearing VJ. The protective role of melanin against UV damage in human skin. *Photochem Photobiol*. 2008;84(3):539–549. doi:10.1111/j.1751-1097.2007.00226.x
36. Agbai ON, Buster K, Sanchez M, et al. Skin cancer and photoprotection in people of color: a review and recommendations for physicians and the public. *J Am Acad Dermatol*. 2014;70(4):748–762. doi:10.1016/j.jaad.2013.11.038
37. Deng L, Ding W, Granstein RD. Thalidomide inhibits tumor necrosis factor-alpha production and antigen presentation by Langerhans cells. *J Invest Dermatol*. 2003;121(5):1060–1065. doi:10.1046/j.1523-1747.2003.12565.x
38. Suchonwanit P, Pakornphadungsit K, Leerunyakul K, Khunkhet S, Sriphojanart T, Rojhirunsakool S. Frontal fibrosing alopecia in Asians: a retrospective clinical study. *Int J Dermatol*. 2020;59(2):184–190. doi:10.1111/ijd.14672
39. Sampaio EP, Sarno EN, Galilly R, Cohn ZA, Kaplan G. Thalidomide selectively inhibits tumor necrosis factor alpha production by stimulated human monocytes. *J Exp Med*. 1991;173(3):699–703. doi:10.1084/jem.173.3.699
40. Londoño F. Thalidomide in the treatment of actinic prurigo. *Int J Dermatol*. 1973;12(5):326–328. doi:10.1111/j.1365-4362.1973.tb00066.x
41. Suchonwanit P, Kositkuljorn C, Mahasaksiri T, Leerunyakul K. A comparison of the efficacy and tolerability of three corticosteroid treatment regimens in patients with alopecia areata. *J Dermatol Treat*. 2022;33(2):756–761. doi:10.1080/09546634.2020.1773384
42. Morissette F, Coulombe J. Refractory pediatric actinic prurigo successfully treated with upadacitinib: a case report. *SAGE Open Med Case Rep*. 2025;13:2050313x251341558. doi:10.1177/2050313x251341558
43. Thadanipon K, Suchonwanit P. Measuring patient quality of life following treatment for alopecia. *Patient Prefer Adherence*. 2021;15:1601–1610. doi:10.2147/ppa.S282399