VOLUME 6
ISSUE 3
2025

ISSN 2563-7673 (PRINT)
ISSN 2563-7681 (ONLINE)

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Ebglyss™ in moderate-to-severe atopic dermatitis (AD)

Ebglyss (lebrikizumab injection) is indicated for the treatment of moderate-to-severe atopic dermatitis in adults and adolescents 12 years of age and older with a body weight of at least 40 kg, whose disease is not adequately controlled with topical prescription therapies or when those therapies are not advisable.

Ebglyss can be used with or without topical corticosteroids.

EFFICACY DATA (week 16)

In ADvocate 1:

- The percentage of EASI 75 responders* and IGA (0,1) responders through week 16 was **>3 times higher with Ebglyss** vs placebo (EASI 75: 59% [n=283] vs 16% [n=141], respectively; IGA (0,1): 43% [n=283] vs 13% [n=141], respectively; *P*<0.001 for both; co-primary endpoints)[1,2]
- The percentage of patients with ≥4-point improvement in Pruritus NRS score at week 16 was **>3 times higher with Ebglyss** vs placebo (46% [n=263] vs 13% [n=130], respectively; *P*<0.001 ; secondary endpoint)[1,2]
- The percentage of EASI 90 responders* through week 16 was **>4 times higher with Ebglyss** vs placebo (38% [n=283] vs 9% [n=141], respectively; *P*<0.001; secondary endpoint)[1,2]

MAINTENANCE OF RESPONSE (week 16 to 52)

ADvocate 1 and 2 pooled data with Ebglyss 250 mg Q4W:

- 291 Ebglyss patients achieving EASI 75 or IGA 0,1 at week 16 without having received any rescue therapy were re-randomized to either Ebglyss 250 mg Q2W, Ebglyss 250 mg Q4W, or matching placebo (Ebglyss withdrawal) up to 52 weeks[1]
- 118 patients were re-randomized to Ebglyss 250 mg Q4W[1]

81.7% (94/115) of patients who were EASI 75 responders at week 16 maintained their response through week 52.[1,3]

76.9% (59/77) of patients who were IGA 0,1 responders at week 16 maintained their response through week 52.[1,3]

Visit ca.lilly.com/en/ebglyss/hcp or scan the QR code to learn more about Ebglyss

Clinical use:

No data are available to Health Canada; therefore, Health Canada has not authorized an indication for pediatric use in patients <12 years of age and in adolescents who weigh <40 kg.

Relevant warnings and precautions:

- Hypersensitivity: Hypersensitivity reactions have been reported following the use of Ebglyss. If a systemic hypersensitivity reaction (immediate or delayed) occurs, Ebglyss should be discontinued immediately, and appropriate therapy initiated.
- Helminth Infections: It is unknown if Ebglyss will influence the immune response against helminth infections by inhibiting IL-13 signalling. Treat patients with the pre-existing helminth infections before initiating treatment with Ebglyss.
- Conjunctivitis and Keratitis: Advise patients to report new onset or worsening eye symptoms to their health professional.
- Vaccinations: Avoid concurrent use of live vaccines in patients treated with Ebglyss.
- Pregnancy: It is preferable to avoid the use of Ebglyss during pregnancy. Women of reproductive potential should be advised to use effective contraception.
- Breast-feeding: A decision must be made to either discontinue breast-feeding or discontinue Ebglyss, considering the benefit of breast-feeding for the child and the benefit of therapy for the woman.

For more information:

Please consult the Product Monograph at https://pi.lilly.com/ca/ebglyss-ca-pm.pdf for important information relating to adverse reactions, drug interactions, and dosing information that have not been discussed in this piece. The Product Monograph is also available by calling 1-888-545-5972.

ADvocate 1 and ADvocate 2: Two identically designed, 52-week, randomized, double-blind, placebo-controlled, Phase 3 trials in adolescents and adults 12 years of age and older with moderate-to-severe AD not adequately controlled by topical medication(s) and who were candidates for systemic therapy. Patients received either Ebglyss 250 mg (with a loading dose of 500 mg at baseline and week 2, n=564) or placebo (n=287) Q2W up to week 16. Patients who responded at the end of this 16-week induction period were re-randomized to receive Ebglyss 250 mg Q2W (n=113), Ebglyss 250 mg Q4W (n=118), or placebo Q2W (n=60) for 36 additional weeks (maintenance period).
*Responder was defined as a subject with a 75% reduction in EASI, or an IGA 0 or 1 ("clear" or "almost clear") and a reduction of ≥2 points on a 0-4 IGA scale, from baseline to week 16, respectively.

EASI=Eczema Area and Severity Index; IGA=Investigator's Global Assessment; NRS=Numeric Rating Scale; Q2W=every 2 weeks; Q4W=every 4 weeks.

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doi.org/10.58931/cdt.2025.63144

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Off-Label Isotretinoin in Dermatologic Conditions: Doses, Duration, and Data

Fiona E. Lovegrove, MD, PhD, FRCPC, DERM

Introduction

Isotretinoin, also known as 13-cis-retinoic acid, has been a mainstay in dermatologic practice since its introduction in the early 1980s. Approved by Health Canada for the treatment of severe acne vulgaris, including recalcitrant nodular acne and acne conglobata,[1] isotretinoin has since been repurposed for a wide variety of other skin conditions. Early clinical trials using an average maximum dose of 1.2 mg/kg/day over a 16 week period demonstrated significant efficacy, with 95% of patients showing clinical improvement and 84% achieving complete clearance.[2]

Traditional isotretinoin regimens involved an initial dose of 0.5 mg/kg/day for 2–4 weeks, followed by maintenance therapy ranging from 0.1–1 mg/kg/day (up to a maximum of 2 mg/kg/day) over a total course of 12–16 weeks. However, findings from a larger dose-ranging study highlighted that side effects such as xerosis were dose-dependent, yet recurrence rates reached 42% in patients treated with 0.1 mg/kg/day,[3] suggesting the need to carefully balance efficacy and side-effect risks when prescribing isotretinoin.

More recently, micronized isotretinoin formulations and a better understanding of its pharmacodynamics have led to more widespread use of low-dose regimens. These offer similar efficacy with fewer adverse effects, better tolerability, and improved adherence.

Isotretinoin exerts its effects through a multimodal mechanism by reducing sebum production, downregulating toll-like receptor expression (thus diminishing inflammation), and normalizing keratinocyte differentiation. These actions make it a versatile therapeutic option for

conditions such as follicular disorders, inflammatory dermatoses, and those with keratinocyte hyperproliferation as a pathology hallmark. Given dermatologists' familiarity with its risk profile and monitoring requirements, isotretinoin has become a commonly used off-label treatment across a broad range of dermatologic diseases.

This article reviews the evidence for off-label use of isotretinoin, focusing on dosing, treatment duration, and efficacy data in conditions where it is most prescribed, beyond severe acne vulgaris.

Common Off-Label Uses of Isotretinoin

"Off-label" refers to prescribing a medication for a condition not formally approved by regulatory authorities such as Health Canada. In dermatology, off-label isotretinoin is frequently used for conditions that are acneiform, follicular, inflammatory, infectious, granulomatous, or neoplastic in nature. Documented off-label uses, also summarized in **Table 1**, include:

- **Acneiform and follicular conditions:** rosacea, perioral dermatitis, hidradenitis suppurativa, gram-negative folliculitis, keratosis pilaris, sebaceous hyperplasia, pseudofolliculitis barbae, keratosis follicularis spinulosa decalvans, nevus comedonicus, and chronic folliculitis.
- **Alopecias:** dissecting cellulitis of the scalp (DSC), frontal fibrosing alopecia (FFA), folliculitis decalvans, and lichen planopilaris.
- **Granulomatous diseases:** lupus miliaris disseminatus faciei and granuloma annulare.
- **Infectious dermatoses:** flat warts and pityriasis versicolour.
- **Inflammatory skin conditions:** lichen planus, seborrheic dermatitis, Morbihan's disease, pityriasis rubra pilaris, and erosive pustular dermatosis of the scalp.
- **Keratinization disorders:** Darier's disease, ichthyosis vulgaris, lamellar ichthyosis, and harlequin ichthyosis.
- **Neoplastic or premalignant conditions:** basal cell carcinoma, squamous cell carcinoma, mycosis fungoides, and field cancerization/actinic keratoses in high-risk patients.

Summarized below are selected conditions where higher-level evidence supports the use of isotretinoin off-label with details on dosing, duration, and treatment outcomes.

Low-Dose Isotretinoin for Acne Vulgaris

A 2021 systematic review by Sadeghzadeh-Bazargan et al. evaluated the available evidence on the efficacy of low-dose isotretinoin for acne treatment.[4] The authors concluded that low-dose regimens, defined as 0.1–1 mg/kg/day, particularly within the 0.1–0.3 mg/kg/day range, achieved comparable clinical outcomes to standard dosing while significantly reducing mucocutaneous and systemic side effects. Notably, recurrence rates after treatment discontinuation were not significantly different between standard- and low-dose groups (34.6% vs. 21.5%, respectively). Low-dose isotretinoin also demonstrated improved patient adherence and cost-effectiveness, making it an attractive option for long-term acne management.

Rosacea

In a 2024 systematic review, Desai and Friedman evaluated the use of isotretinoin in treating rosacea, particularly in patients with recalcitrant papulopustular disease.[5] Dosing regimens ranged from 0.1–0.5 mg/kg/day or fixed doses of 10–20 mg/day, typically for 16 weeks. Reported clearance rates ranged from 71–100%, with improved outcomes observed at higher doses. Isotretinoin was generally well tolerated and may be especially useful for patients with persistent inflammatory rosacea who do not respond to antibiotics or topical agents.

Dissecting Cellulitis of the Scalp

Guo et al. conducted a meta-analysis on isotretinoin use in DSC, which showed improvement or complete remission in 75–100% of patients across five studies, with a pooled efficacy rate of 90% (95% confidence interval: 0.81–0.97).[6] The average dosing ranged from 0.5–0.75 mg/kg/day for a minimum of three months of treatment. Recurrence, where reported, occurred in 24% of patients following discontinuation. While data are limited, isotretinoin appears to be a reliable systemic option for this difficult-to-treat scaring alopecia.

Frontal Fibrosing Alopecia

Shahpar et al. reviewed the available evidence supporting isotretinoin as an adjunctive therapy for FFA.[7] Doses ranged from 10–40 mg/day, with nearly 90% of patients reporting significant symptomatic improvement, including reduced facial papules and stabilization of hair loss. Although current evidence is limited to case series and small observational studies, isotretinoin may be considered for patients who do not respond to topical or intralesional steroids, antimalarials or antiandrogens.

Flat Warts

In a randomized placebo-controlled trial, Olguin-García et al. investigated isotretinoin at 30 mg/day over 12 weeks in patients with recalcitrant flat facial warts.[8] All patients in the treatment arm (N=16) achieved complete clearance, compared to none in the placebo group (N=15). Adverse effects were minimal and consistent with the known safety profile of isotretinoin. These findings suggest that isotretinoin may be a highly effective alternative for managing flat warts, particularly when topical therapies or cryotherapy have failed.

Seborrheic Dermatitis

Luque-Luna et al. conducted a systematic review on the use of isotretinoin in moderate-to-severe seborrheic dermatitis.[9] Low-dose regimens (≤0.5 mg/kg/day) were used in most studies. Clinical improvement was observed in 96% of patients, with complete remission achieved in 45%. The recurrence rate at 3 months post-treatment was 11%. These data suggest that isotretinoin may be a safe and effective alternative in refractory cases where conventional topical therapies are insufficient.

Conclusion

The off-label use of isotretinoin has become an important tool in dermatologic practice, supported by a growing body of evidence across a wide range of conditions. Its therapeutic potential lies in its well-understood mechanisms of action and a familiar safety profile.

This review summarizes the current evidence to provide a practical reference for Canadian dermatologists who regularly manage these conditions. With continued research, and appropriate patient counselling and monitoring, isotretinoin may find even broader applications in clinical dermatology.

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Financial Disclosures

F.E.L: Consultant/Speaker/Advisory Board Honorarium: Abbvie, Amgen, Arcutis, Bausch, Celgene, Celltrion, Galderma, JAMP, Janssen, LEO, Lilly, Novartis, Pfizer, Sanofi-Genzyme, UCB; **Grants, Research or Clinical Trials:** Abbvie, Amgen, Bausch, COREvitas, GSK, Incyte, Janssen, LEO, Lilly, Sanofi-Genzyme, UCB

Disease	Treatment Details	Highest Level of Evidence Available
Acneiform/follicular		
Acne (low-dose)	**Dose:** 0.1–0.3 mg/kg **Duration:** >6 months **Outcome:** fewer side effects than standard dosing.	Systematic Review[4]
Gram-negative folliculitis	**Dose:** 0.5–1.5 mg/kg **Duration:** 14–36 weeks	Case series[10]
Hidradenitis suppurativa (HS)	**Outcome:** 16 patients (23.5%), the condition completely cleared during initial therapy, and 11 patients (16.2%) maintained their improvement during the follow-up period.	Case Series[11]
Keratosis follicularis spinulosa decalvans (KFSD)	**Dose:** 20 mg (0.25 mg/kg) daily **Duration:** 12 months **Outcome:** patient responded to treatment.	Case Report[12]
Perioral Dermatitis (POD)	**Dose:** "Low dose" **Duration:** 6 months	Case report[13]
Rosacea	**Dose:** 0.1–0.5 mg/kg/day or fixed 10–20 mg/day **Duration:** average of 16 weeks **Outcome:** 71–100% resolution.	Systematic Review[5]
Sebaceous Hyperplasia	**Dose:** 1 mg/kg/day **Duration:** 2 months **Outcome:** average lesion count had reduced from 24 to 2 at study end.	Prospective Case Series (N=20)[14]
Alopecias		
Dissecting Scalp Cellulitis (DSC)	**Outcome:** overall efficacy rate of 0.9 with a 95% confidence interval (0.81–0.97). Sensitivity analysis suggested consistently high efficacy, ranging from 0.83–0.94. Recurrence was reported in 24% (6/25) of patients.	Meta-analysis[6]
Folliculitis DeCalvans (FDC)	**Dose:** 0.1–1.02 mg/kg/day (10–90 mg/day) **Duration:** median of 2.5 months (range: 1–8 months) **Outcome:** 82.0% of patients achieved healing following treatment. Those who received oral isotretinoin ≥0.4 mg/kg/day for ≥3 months responded better, with 66% remaining relapse-free.	Retrospective Case Series[15]
Frontal Fibrosing Alopecia (FFA)	**Dose:** 10–40 mg/day **Outcome:** Nearly 90% of patients experienced a significant reduction of symptoms.	Systematic Review[7]
Granulomatous		
Lupus Miliarius Disseminatus Faceii (LMDF)	**Dose:** 20 mg/day **Duration:** 6 months **Outcome:** Complete resolution.	Case Report[16]
Infectious		
Flat Warts	**Dose:** 30 mg/day or placebo was administered to 16 and 15 patients. **Duration:** 12 weeks **Outcome:** all participants in the isotretinoin group showed complete clearance.	Randomized, Placebo-Controlled Trial[8]

Disease	Treatment Details	Highest Level of Evidence Available
Pityriasis (Tinea) versicolour	**Dose:** 20 mg/day **Duration:** 2 months **Outcome:** Complete resolution by 6 weeks; sustained at 1 year.	Case Report[17]
Inflammatory		
Morbihan's disease	**Dose:** mean sustained daily dose of 60 mg/day (range, 40–80 mg/day). The mean cumulative dose was approximately 285 mg/kg (range, 170–491 mg/kg) **Duration:** 10 to 24 months **Outcome:** mean disease-free follow-up period of 9 months (range, 1–24 months). A substantial clinical improvement was not noted until 6 months of treatment.	Case series (N=5)[18]
Pityriasis Rubra Pilaris (PRP)	**Outcome:** 61.1 % (102/167) of patients treated with isotretinoin achieved an excellent response.	Systematic Review[19]
Seborrheic Dermatitis (SD)	**Dose:** Low dose (≤0.5 mg/kg/day) **Outcome:** 96% of patients showed an improvement in SD, with 45% achieving a complete response. The recurrence rate at 3 months after discontinuing the drug was 11%.	Systematic Review[9]
Miscellaneous		
Darier's disease	**Dose:** 0.5 mg/kg/day **Duration:** 16 weeks **Outcome:** approximately 94% improvement.	Open-label multicenter study (N=104)[20]
Harlequin Ichthyosis	**Dose:** 1 mg/kg/day **Duration:** starting on day 7 of life.	Case Report[21]
Neoplastic		
Basal cell carcinoma (BCC)	**Dose:** 10 mg/day **Duration:** 3 years **Outcome:** No significant difference in new BCC occurrence or annual tumour rate between isotretinoin and placebo arms.	Randomized, Double-blind, Controlled Trial[22]
Mycosis fungoides	**Outcome:** objective clinical response was observed in 44% (11 patients), including three clinical complete responses.	Prospective Case Series (N=25)[23]
Squamous cell carcinoma (SCC)	**Dose:** 1 mg/kg **Duration:** >4 weeks **Outcome:** "striking responses" in all patients.	Prospective Case Series (N=4)[24]

Table 1. Summary of dermatologic conditions treated with off-label isotretinoin, including treatment details and the highest level of supporting evidence referenced; *courtesy of Fiona E. Lovegrove, MD, PhD, FRCPC, DERM.*

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Atopic Dermatitis in Asians: A Review on Genetics, Clinical Presentation, and Therapeutic Implications

Harry Liu, MD, FRCPC, FAAD

Introduction

Atopic dermatitis (AD) is a common, multifactorial, and pruritic inflammatory skin condition with a chronic relapsing course. It typically manifests in infancy or early childhood and is often associated with other atopic conditions, such as asthma and allergic rhinoconjunctivitis. As the most prevalent chronic inflammatory skin disease worldwide, AD affects individuals across a wide range of racial and ethnic backgrounds. Recent studies suggest that Central Asia has the highest pediatric prevalence of AD in 2021, with a rate of 10.5%, surpassing the 5.4% prevalence observed in high-income North America.[1] According to the 2021 Census, individuals of Asian descent constitute the third-largest population group in Canada, following those of European and North American descent, with nearly half originating from East or Southeast Asia. Furthermore, Asians represent Canada's fastest-growing demographic. This review highlights the genetic factors, clinical presentations, and therapeutic

considerations for AD in Asian populations, aiming to improve understanding and inform tailored treatment approaches.

Epidemiology

Ethnic diversity across Asia is vast, encompassing East Asians (EAs), South Asians (SAs), and Southeast Asians (SEAs). A recent population study in Singapore estimated AD prevalence rates of 9.2% in EAs, 8.5% in SAs, and 8.4% in SEAs.[2] A 2025 study on Asian American and Pacific Islander children found an overall AD prevalence of 6.0%, with significantly higher rates among Filipino (12.8%), Chinese (12.0%), Vietnamese (11.7%), Native Hawaiian/Pacific Islander (9.5%), South Asian (8.4%), and Black (8.2%) children compared to non-Hispanic White and Hispanic children.[3] These disparities highlight the need for further research into the genetic and immune mechanisms contributing to increased AD risk in these populations. Notably, Canadian studies on this topic remain scarce.

Multi-ethnicity is also emerging as an important factor influencing the global prevalence of allergic diseases. A Korean study found that AD prevalence was significantly higher in non-multi-ethnic individuals compared to those from multi-ethnic backgrounds. Furthermore, within multi-ethnic groups, a parent's region of birth had a significant impact on the prevalence of allergic diseases.[4] These findings underscore the need for further investigation into the role of genetic and environmental factors in AD susceptibility across diverse populations, especially within Canada's growing multi-ethnic population.

Genetics

AD is a complex genetic disorder influenced by environmental factors. Among various ethnic groups, loss-of-function variants in the filaggrin (*FLG*) gene represent the strongest genetic risk factor for AD and play a crucial role in its pathogenesis.[5] Studies in Europe report that up to 50% of AD patients carry one or more *FLG* null mutations. In contrast, Asian populations exhibit lower *FLG* mutation frequencies but higher diversity. Reported prevalence rates of *FLG* mutations include 31.4% in Han Chinese, 27.0% in Japanese, 26.0% in Singaporean Chinese, and 15.7% in Koreans.[5,6] Besides the differences in prevalence, *FLG* mutations also show significant population specificity. For example, mutations common in European populations are rarely detected in Asian groups.[5,7] Moreover, *FLG* mutations vary considerably among different Asian subpopulations, including Chinese, Korean, Japanese, and Singaporean individuals.[6,8,9] Genetic diversity analyses further suggest that the geographic distribution of *FLG* mutations reflects prehistoric human migration patterns in East Asia, underscoring their potential as ancestryinformative markers.[10]

Genetic studies of *FLG* mutations have revealed unique associations across different Asian subpopulations. Unlike findings in European populations, *FLG* mutations do not appear to increase the risk of asthma in EAs with AD.[10] In the Han Chinese population, these mutations have been linked to a higher predisposition for food sensitization.[5] Among Korean patients, *FLG* mutations are significantly associated with elevated immunoglobulin E (IgE) levels, palmar hyperlinearity, and a family history of allergic diseases.[8] Among Indian populations, *FLG* mutations have been correlated with more severe hand eczema.[11] However, given the rising prevalence of allergic diseases in India over the past decade, further research on *FLG* mutations in this population is needed.[12]

Beyond *FLG* mutations, AD in East Asian populations has also been linked to loss-of-function mutations in the *SPINK5* gene, which encodes a serine protease inhibitor crucial for maintaining epidermal homeostasis.[13] Moreover, genomic and proteomic analyses suggest that East Asian AD exhibits fundamental differences from its European counterpart.[2] Additionally, studies have identified associations between AD and polymorphisms in immune-related genes such as interleukin *(IL)-4* and *IL-13/IL-13Ra1* in Chinese, Japanese, and Korean populations.[13] These findings highlight the need for a more nuanced understanding of genetic factors contributing to AD across diverse Asian subpopulations.

Immune Polarization

The AD phenotype observed in Asian populations exhibits a unique immunological profile, resembling a blend of both European AD and European psoriasis at the cellular and molecular levels.[13,14] While strong Th2 activation is a universal feature of AD, Asian AD patients also show significant upregulation of Th17 and Th22 pathways.[2,15] Increased Th17 activity is accompanied by a downregulation of the Th1 axis, with higher counts of IL-17–producing cells in Asian AD patients.[2,16] Notably, Th17-related mediators are elevated in the skin but not in peripheral blood, whereas IL-22 levels are increased in both compartments.[15] Histological analysis of lesional skin from Asian AD patients shows greater acanthosis, increased Ki67 expression, and more frequent parakeratosis compared to European AD.[16] These features may be attributed to elevated IL-22 levels, which promote keratinocyte proliferation, migration, and impaired differentiation by inhibiting proteins involved in terminal keratinocyte maturation.[2] Given these distinct immunological and histological differences, further research is needed to determine whether targeting Th2 alone is sufficient for managing all AD phenotypes, particularly those with a stronger Th17 component.

Clinical Presentation

In the United States, individuals identifying as Asian/Pacific Islander and Black are significantly more likely to seek medical care for AD despite generally lower overall healthcare utilization rates. Asian/Pacific Islanders, in particular, are nearly seven times more likely than White individuals to have an office visit resulting in an AD diagnosis.[17] One contributing factor may be the lack of familiarity with AD among Asian populations, compounded by the heterogeneity of clinical manifestations across diverse ethnic backgrounds.

Across all ethnic groups, the most universally prevalent AD features include pruritus, lichenification, and xerosis. However, in EA populations, AD lesions tend to be more sharply-demarcated due to the psoriasiform Th17/Th22 endotype (**Figure 1**).[15,18] Compared to White patients, Asian individuals with AD more frequently exhibit increased scaling and lichenification (**Figure 2**).[13] Perifollicular accentuation is a commonly observed feature, while papular eczema—characterized by small, flat-topped papules—can mimic lichen planus in appearance (**Figure 3**). In individuals with darker skin tones, such as those of SA populations, erythema may present as a violaceous hue or be subtle enough to go unnoticed altogether.[13] Detecting erythema in darker skin requires careful assessment of secondary signs, such as edema, increased skin warmth, and scaling.[13] Clinicians should compare affected areas to the patient's baseline skin tone, while pruritus, excoriations, and skin induration—best visualized with tangential lighting or palpation—serve as additional diagnostic clues. Moreover, patients with darker skin tones are at greater risk for post-inflammatory hyperpigmentation, which can be more distressing than the primary skin manifestations of AD itself.[19]

Beyond these general characteristics, AD phenotypes vary among Asian subpopulations. Studies from SEA populations report higher rates of exudative eczema, truncal involvement, lichenification, and prurigo nodularis.[2] In contrast, EA populations exhibit a higher prevalence of erythroderma, truncal, extensor (**Figure 4**), scalp, and auricular (**Figure 1**) involvement.[2] SA populations, particularly Indian patients, more commonly present with flexural involvement.[2] Environmental and cultural factors further shape the AD phenotype, both within Asia and among Asian immigrant communities worldwide. As a result, additional research is needed to determine whether these reported Asian AD phenotypes remain consistent among Asian Canadians, despite differences in environmental exposures, microbiome composition, and cultural practices. Understanding these variations will be essential for developing more tailored and effective treatment strategies.

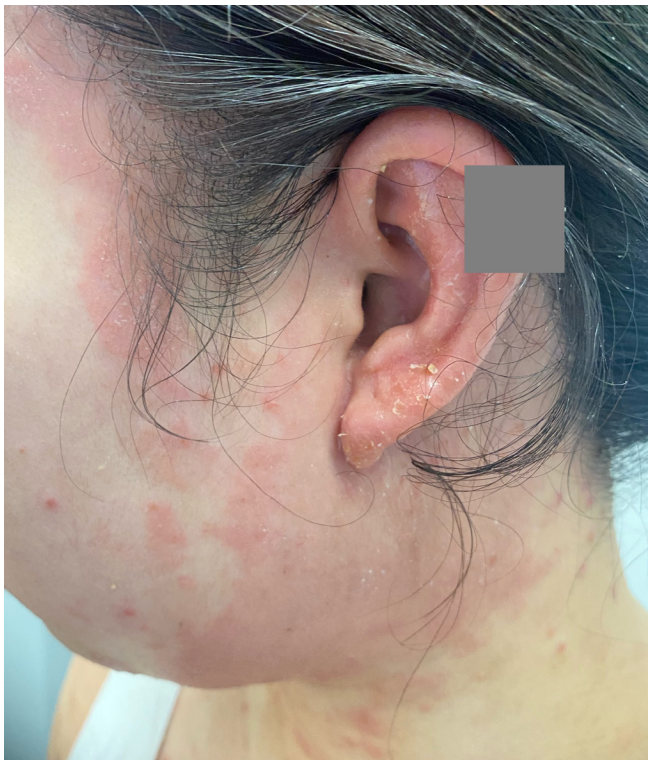

Figure 1. Biopsy-confirmed atopic dermatitis in a young East Asian female, presenting with well-demarcated psoriasiform plaques ranging from pink to erythematous during an acute flare; *courtesy of Harry Liu, MD, FRCPC, FAAD.*

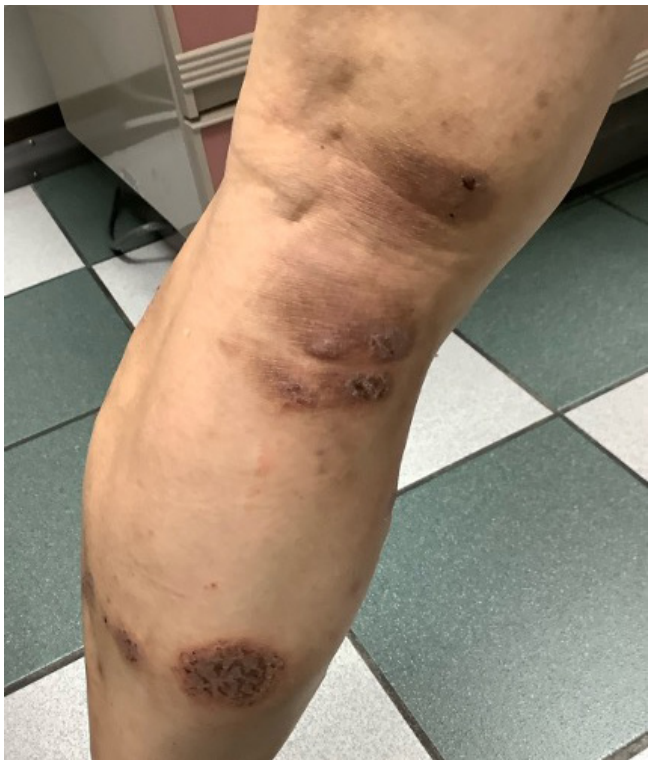

Figure 2. Marked lichenification with well-defined borders in a middle-aged Chinese male with atopic dermatitis; *courtesy of Harry Liu, MD, FRCPC, FAAD.*

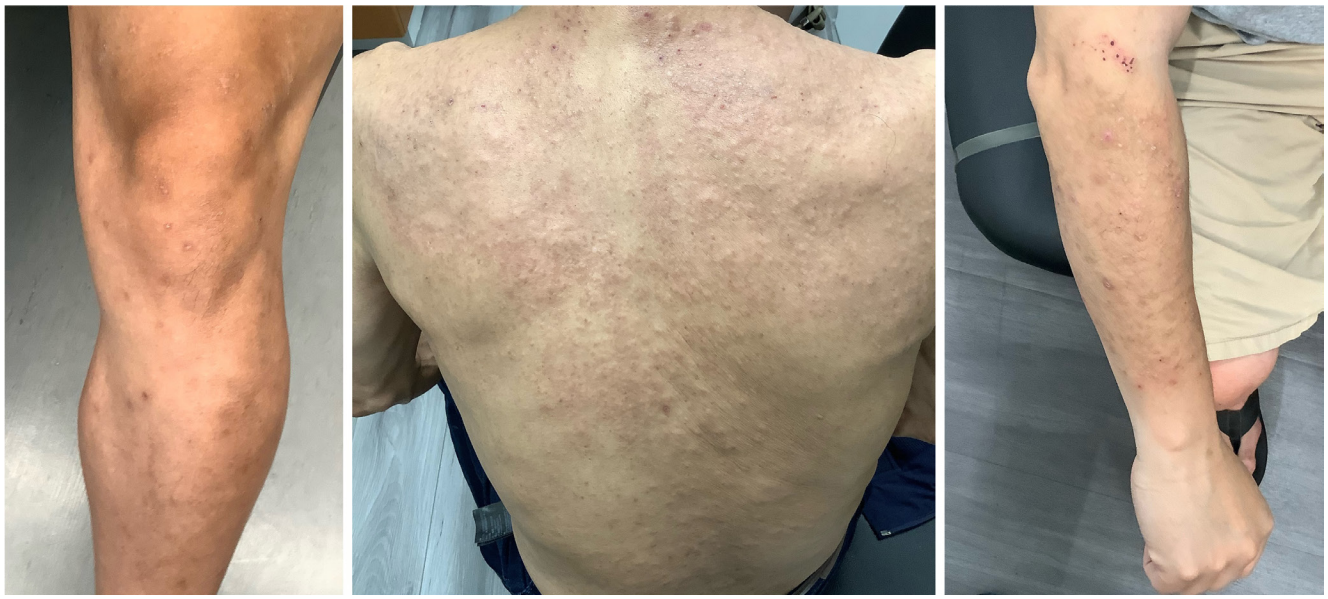

Figure 3. Distinct papular lesions in three different patients with atopic dermatitis (Indian patient on the left, and East Asian patients in the middle and on the right); *courtesy of Harry Liu, MD, FRCPC, FAAD.*

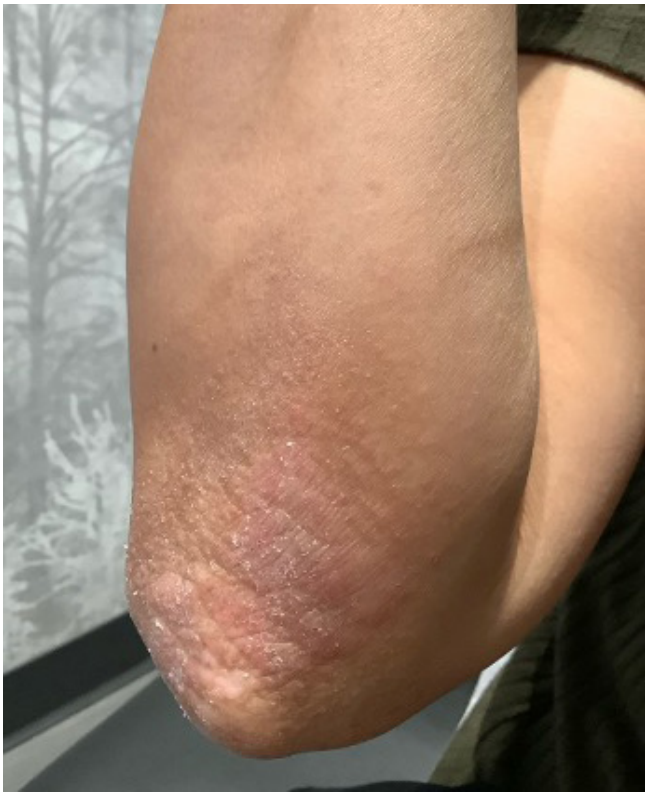

Figure 4. Notable extensor involvement in a young male with atopic dermatitis in a Chinese patient; *courtesy of Harry Liu, MD, FRCPC, FAAD*.

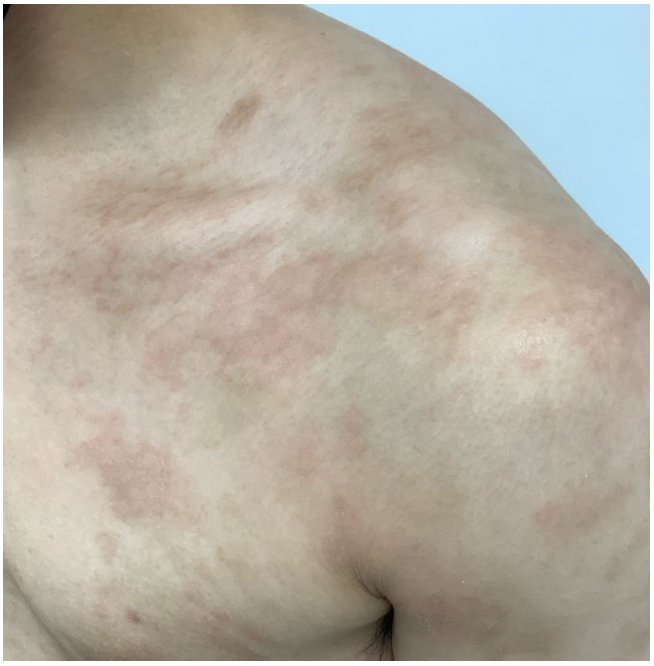

Figure 5. Active atopic dermatitis with significant post-inflammatory hyperpigmentation in a Filipino patient; *courtesy of Harry Liu, MD, FRCPC, FAAD*.

Therapeutic Implications

With the increasing burden of AD in Asia, the development of effective therapeutics is critical.[20] A major challenge contributing to this burden is the variable and often unpredictable response to treatment. While individuals from the same racial groups may share phenotypic and genetic traits, treatment efficacy can vary widely among different ethnic backgrounds. This highlights the complex and heterogeneous nature of AD, underscoring the necessity for personalized and region-specific therapeutic approaches.

In many Asian cultures, topical treatments, bath therapies, and oral herbal preparations are commonly used. These treatments can significantly impact the outcome of dermatological care and may have been used by patients prior to seeking professional treatment. Therefore, it is crucial for clinicians to inquire about these therapies before discussing the treatment plan. For prescribed topical therapies, prolonged use of high-potency topical corticosteroids can lead to hypopigmentation, a concern particularly relevant for patients with darker skin tones. Clinical studies have demonstrated the efficacy of pimecrolimus cream and tacrolimus ointment in treating AD in Asian patients.[13] Phototherapy, including narrow-band ultraviolet B (NB-UVB) and ultraviolet A (UVA), has shown effectiveness in managing moderate to severe AD in Asian populations.[13] Given the increased melanin content in more pigmented skin types, higher doses of NB-UVB may be required to achieve optimal results. Regarding systemic treatments, phase III trials of dupilumab that included 20–27% Asian participants demonstrated efficacy across diverse ethnic groups. A subsequent pooled sub-analysis further confirmed that treatment responses in Asian patients were comparable to those observed in other populations.[13]

Overall, studies have consistently highlighted the persistent underrepresentation of patients with skin of colour in global clinical trials for AD.[21] Despite the high prevalence of AD among non-White populations, data on the efficacy of common therapies in these groups remain limited. A significant barrier is the incomplete reporting of race and ethnicity in clinical trials. For instance,

among AD clinical trials published between 2000 and 2009, only 59.5% included race and ethnicity as part of baseline demographic data.[13] Furthermore, Asian participants accounted for just 6.9% of trial enrolment, underscoring the need for greater diversity in AD research.

Future studies focused on identifying specific and potentially unique molecular targets in Asian populations could pave the way for developing therapies that address the unmet needs of Asian patients with AD. However, the broader spectrum of *FLG* mutations observed in Asian populations may present challenges for developing targeted treatments. Additionally, the increased Th17 axis observed in Asian AD patients suggests they may be promising candidates for therapies targeting IL-17/IL-23.[22] Furthermore, the persistent elevation of IL-22 levels in both skin and peripheral blood among Asian populations presents another potential treatment target.[15,22]

Conclusion

AD presents a significant and growing challenge, particularly within Asian populations, due to its multifactorial nature, genetic diversity, and variable clinical manifestations. As AD prevalence continues to rise, it is essential to adopt more personalized and culturally competent approaches to diagnosis and treatment. Advancing our understanding of the genetic underpinnings, immune polarization, and environmental influences that contribute to the distinct AD phenotypes in Asian populations will be crucial for developing more targeted and effective therapies.

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Financial Disclosures:

H.L.: Advisory Board: Sanofi, Arcutis, L'Oréal, Neutrogena, Sun Pharma; **Speaker:** Sanofi, Arcutis, Celltrion, Sun Pharma

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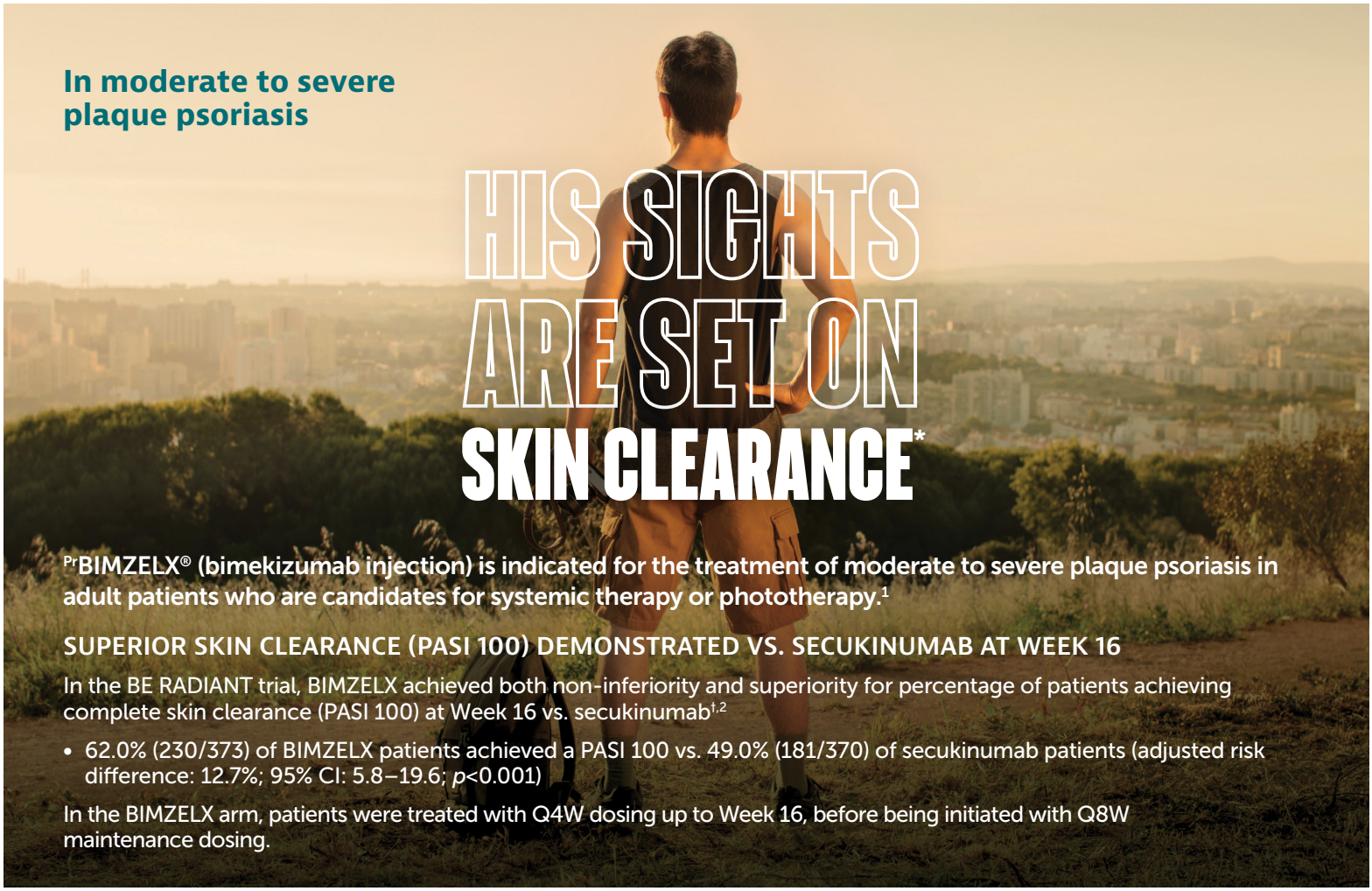

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CA-BK-2400086

doi.org/10.58931/cdt.2025.63146

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Scabies: Clinical Pearls for Community Dermatologists

Patrick Fleming, BSc (Nutrition), MSc (Community Health), MD, FRCPC

Introduction

Scabies is a common parasitic skin infestation caused by *Sarcoptes scabiei var. hominis*, with an estimated global burden of over 200 million cases annually.[1] Scabies remains underdiagnosed and can often be challenging to manage. Institutional outbreaks, diagnostic delays, and treatment failures contribute to ongoing morbidity. This practical literature review summarizes clinical pearls for community dermatologists, integrating recent diagnostic frameworks, treatment evidence, and evolving considerations related to drug resistance and public health control.

1. Identification - International Alliance for the Control of Scabies Criteria Aid in a Structured Diagnosis

The International Alliance for the Control of Scabies released a consensus diagnostic framework in 2020 that stratifies the diagnosis of scabies into three levels: confirmed, clinical, and suspected.[2]

Confirmed scabies requires direct visualization of mites, eggs, or feces, typically via dermoscopy or microscopy. Dermoscopy—revealing the characteristic "delta-wing jet" sign—is a practical diagnostic tool in outpatient settings and is widely used in clinical trials.

Treatment	Dosage/Regimen	Indications	Precautions	Brand Names (*Canada*)
Permethrin 5% cream/lotion	Apply from the neck down (include the scalp in children and older adults) Repeat after 7–14 days.	First-line treatment for most patients, including children ≥2 months and pregnant individuals.	Irritation; avoid eyes and mucous membranes.	*Nix Dermal Cream, Kwellada-P Lotion*
Oral Ivermectin	Administer 200 mcg/kg on days 1 and 8; adjust for crusted scabies or institutional cases.	Alternative first-line treatment in adults particularly when topical treatment is not feasible or if resistance is a concern.	Avoid in pregnancy; Not ovicidal.	*Stromectol*
Benzyl Benzoate 25%	Apply nightly for 2–3 days, repeat after 7 days if needed.	Resource-limited settings; not available in US/Canada.	May cause skin irritation/stinging.	N/A (not commercially available in Canada)
Sulfur 5–10% Ointment	Apply nightly for 3 consecutive nights. Rinse off each morning.	Infants <2 months, and individuals who are pregnant or breastfeeding.	Odorous, stains clothing, poorly tolerated.	Compounded product only
Crotamiton 10%	Apply daily for 5 days.	Second line if other treatments are contraindicated or unavailable.	Low efficacy; avoid in severe disease.	*Eurax*
Spinosad 0.9% Suspension	Apply once and allow 10 minutes to dry. Wash off after 6 hours. Repeat in 7 days if needed.	Approved for children ≥4 years and adults.	Well-tolerated.	*Natroba* (not yet available in Canada)

Table 1. Classic Scabies Treatment[10]; *courtesy of Patrick Fleming, BSc (Nutrition), MSc (Community Health), MD, FRCPC.*

Clinical scabies is diagnosed based on the presence of classic lesions in characteristic locations with supportive history (e.g., burrows and/or scrotal nodules).

Suspected scabies may lack classic features such as burrows, but this does not exclude a diagnosis of scabies as they can be difficult to identify. Dermatologists should maintain a low threshold for starting treatment for suspected scabies.

2. Permethrin Resistance May Be Increasing

In North America, topical permethrin remains the first-line therapy for scabies. However, a recent double-blind randomized controlled trial conducted in Austria (n = 110) demonstrated significantly lower cure rates with three consecutive applications of permethrin (n = 14/52 participants, 27%) compared to benzyl benzoate 25% (n = 47/54 participants, 87%, p<0.0001).[3] This discrepancy may be due to benzyl benzoate being more ovicidal than permethrin as there was not a repeat application of either treatment on day seven of the trial, which is a common practice with permethrin in clinical settings (refer to **Tables 1 and 2** for a summary of the treatments).

3. Consider Oral Ivermectin Earlier in Select Cases

Oral ivermectin (200 µg/kg/dose, two doses spaced 7–14 days apart) is an effective treatment option for adult and pediatric patients with extensive involvement, crusted scabies, poor adherence to topical therapies, and/or in institutional settings. A 2024 meta-analysis found lower treatment failure rates with two doses (7.1%) versus one dose (15.2%).[4] Additionally, a 2019

Component	Regimen	Notes
Oral Ivermectin	Administer 200 mcg/kg on days 1, 2, 8 (moderate); add days 9, 15 (severe); up to 7 doses total.	Usually used in conjunction with topical treatment.
Topical Permethrin 5% cream or lotion	No consensus on frequency. Typically applied on the same days as oral ivermectin.	Apply to entire body, including scalp, temples, and under nails (avoid the face).
Keratolytics (e.g., 10% urea lotion or 3% salicylic acid cream)	Apply on non-permethrin days then on a regular basis once clear.	Aids penetration of topical therapy by removing thick crusts. *Dispose of once treatment is complete to reduce cross-contamination.*
Environmental Control	Wash bedding/clothes in extra hot water; disinfect surfaces on a regular basis. Patient should be in isolation with strict contact precautions and essential visitors only until clear.	For patients in long-term care, the charge nurse and/or infection control team should be promptly notified—ideally via both urgent written and verbal communication—to coordinate outbreak prevention and contact tracing.
Empiric Treatment of Close Contacts	Permethrin and/or ivermectin, depending on feasibility.	Close contacts, including asymptomatic individuals, should be treated simultaneously. Healthcare workers (and their household contacts) with close, unprotected exposure to patients with crusted scabies should receive empiric treatment, regardless of symptoms.

Table 2. Crusted Scabies Treatment (Centers for Disease Control Protocol)[8,10]; *courtesy of Patrick Fleming, BSc (Nutrition), MSc (Community Health), MD, FRCPC.*

French study did not identify any new safety signals in children <15 kg, with mild adverse events in 4% of cases and an 85% cure rate.[5]

4. All Close Contacts Must Be Treated Simultaneously

Treatment failure is frequently due to reinfestation from untreated close contacts. All household members, sexual partners, and other close contacts must be treated concurrently to prevent recurrence.[6] Asymptomatic close contacts are common sources of reinfestation and must also be treated.[2] Because disclosure can be socially stigmatizing, it is important to provide clear education on the rationale for empiric treatment of asymptomatic close contacts—emphasizing its role in preventing reinfestation and protecting household members.

5. Environmental Decontamination is Often Inadequate

Even with optimal pharmacologic therapy, failure to implement environmental decontamination can result in reinfestation. *Sarcoptes scabiei* can survive off-host for up to 2–3 days.[7] Bedding, clothing, towels, and upholstered surfaces can act as reservoirs, especially in cases of crusted scabies. Patients should be advised to wash exposed items in hot water and dry them using high heat. Non-washable items should be sealed in plastic bags for at least 72 hours.[8] Unfortunately, many practitioners underemphasize these steps during routine care, contributing to persistent household infestation.

6. Crusted Scabies Requires Aggressive Treatment

Crusted scabies, a highly contagious and often underrecognized variant, is associated with a heavy mite burden. It occurs most often in patients who are immunocompromised, elderly, or living with advanced dementia.[9,10] Standard care involves oral ivermectin (200 µg/kg) given on multiple days (e.g., days 1, 2, 8, with optional doses on days 9, 15), combined with topical permethrin 5% (left on for 8–14 hours), which is often applied concurrently and on days when oral therapy is not given.[7,8] The use of keratolytic agents, such as 10% urea lotion, is critical to facilitate topical drug penetration through hyperkeratotic crusts.[6]

In institutional settings, patients with scabies should be placed on strict contact precautions. Given the high attack rate of crusted scabies, prophylactic treatment is recommended for asymptomatic healthcare workers—and their household contacts—if they were not fully protected. Crusted scabies constitutes a local public health emergency. Timely diagnosis and aggressive management are critical to preventing outbreaks and minimizing morbidity.

7. Persistent Pruritus and Nodules Are Not Always Treatment Failures

Post-scabetic pruritus can persist for many weeks or months after mite eradication and is not necessarily indicative of active infestation.

Treatment of post-scabetic pruritus may include fragrance-free emollients, topical 1% pramoxine hydrochloride, topical corticosteroids, and/or oral antihistamines. Nodular scabies, often affecting the axillae or scrotum, may persist and require intralesional corticosteroids or, in select cases, systemic agents such as a short course of prednisone.[9] When in doubt, there should be a very low threshold for re-treatment of suspected scabies cases and their close contacts.

8. Beware of Misdiagnosis—Especially in Eczematous Skin

Scabies is often misdiagnosed as eczema, especially in adults and children with atopic dermatitis. A high index of suspicion is warranted when the following features are present:

- Severe nocturnal pruritus.
- Eczema unresponsive to typical therapy.
- Multiple household members are affected.
- Pruritic nodules on the genitals, breasts, and/or axillae.
- Risk factors (e.g., long-term care resident or in crowded environments).

9. Spinosad: A Promising New Topical Agent

Topical spinosad 0.9% is a neurotoxin that has shown promise in the treatment of scabies. In two randomized, double-blind, vehicle-controlled trials involving over 500 participants, a single 10-minute application of spinosad resulted in a complete cure rate of 78.1% by Day 28, compared to 36.1% in the vehicle group.[11] Complete cure was defined as the resolution of all symptoms and skin lesions, absence of new lesions, negative dermoscopic findings, and no requirement for reapplication. The treatment was well-tolerated with low rates of irritation. No cases of resistance were reported.

Spinosad does not require overnight application. It is applied to dry skin, allowed to dry for 10 minutes, and rinsed off after 6 hours, an approach that may improve adherence. Although not yet available in Canada, spinosad may offer a well-tolerated, effective, and potentially more patient-centred treatment for scabies.

Conclusion

Scabies remains a significant global public health concern. Community dermatologists should be familiar with standardized diagnostic criteria, recognize atypical and high-burden presentations, and stay informed about evolving resistance patterns. Treatment should extend beyond the index patient to include all close contacts and the environment. As treatment failures and resistance become more common, agents such as oral ivermectin and topical spinosad may play a greater role in routine practice.

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Financial Disclosures

P.F.: Honorarium and/or consulting and/or advisory boards and/or speaking fees: AbbVie, Altius, Amgen, Aralez, Arcutis Biotherapeutics, Bausch Health, Beiersdorf, Bristol Myers Squibb, Catalytic Health, Celltrion, CeraVe, Cipher, Galderma, Eli Lilly, Fresenius Kabi, Incyte, Kenvue, La Roche-Posay, Janssen, Medexus Pharmaceuticals, Novartis, Pfizer, UCB, Sanofi-Genzyme, Sermo, and Sun Pharma

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The initial request is authorized for a maximum period of four months. When requesting continuation of treatment, the prescriber must provide information making it possible to establish the beneficial effects of the treatment, specifically: An improvement of at least 75% in the PASI score compared to the baseline value; or an improvement of at least 50% in the PASI score and at least 50% in the body surface area affected, compared to the baseline values; or a significant improvement in lesions on the face, palms or soles or in the genital area compared to the pretreatment assessment.

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Authorizations for tildrakizumab are given for 100 mg at weeks 0 and 4, then every 12 weeks thereafter.

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REFERENCE:
Current ILUMYA® Product Monograph, Sun Pharmaceutical Industries Limited.

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PM-CA-ILY-0077

doi.org/10.58931/cdt.2025.63147

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A Dermatologist's Beginner's Guide: Using Ambient Artificial Intelligence in Your Practice

Juthika Thakur, MD

Introduction

Automatic Speech Recognition (ASR) is the fundamental technology that enables the conversion of spoken language into written text. The strengths of ASR include interpreting voices, identifying different speakers in a conversation, and following dialogue. With the support of human trainers, ASR systems can further improve and optimize their performance based on feedback. However, their effectiveness can be limited by factors such as noisy environments, poor microphone placement, and variations in dialects and accents. See **Exhibit 1** for definitions of key AI terms.

Large Language Models (LLMs) aim to bridge these gaps by using Natural Language Processing (NLP) to fill in missing elements of a conversation. As a type of generative AI, an LLM predicts the next word in a sequence based on patterns found in its training dataset (**Figure 1**). The goal of an LLM is to mimic human language by identifying which words are likely to follow one another, given the context provided in the prompt and its prior training. LLMs can generate original content and optimize their outputs based on human input. However, because they function by predicting word sequences, they do not inherently understand whether their outputs are true or false. As a result, LLMs can produce inaccurate or fabricated responses, commonly referred to as "hallucinations" in their responses to prompts.

Real-time ambient AI scribes, which leverage machine learning to process conversations, show promising potential to reduce the documentation burden, enhance the quality of doctor–patient interactions, and support clinicians in their daily

Key AI Terms and Definitions
Artificial Intelligence: A field of computer science focused on creating systems that can perform tasks normally requiring human intelligence, such as reasoning, problem-solving, learning, and language understanding.
Machine Learning: A subset of AI where computers learn patterns from data to make decisions or predictions without being explicitly programmed for each task.
Augmented intelligence: A collaborative approach where humans and AI systems work together to enhance human decision-making and performance, rather than replacing human intelligence.
Agentic AI: AI systems designed to operate autonomously and pursue goals or perform actions proactively, often with some level of decision-making or initiative.
Large Language Models: AI models trained on vast amounts of text data to understand and generate human-like language. Examples include ChatGPT, Claude, and Gemini.
Natural Language Processing: A branch of AI focused on enabling machines to understand, interpret, and generate human language, both written and spoken.
Hallucinations: When an AI model generates information that sounds plausible but is actually false or not based on real data.
Supervised learning: A type of machine learning where the model is trained on labelled data (input-output pairs), allowing it to learn to make predictions or classifications.
Unsupervised learning: A machine learning technique where the model is trained on data without labelled outcomes, often used to find patterns or groupings (like clustering).
Bias: Systematic errors in AI outputs caused by imbalanced or flawed training data, design choices, or societal inequalities, leading to unfair or inaccurate results.
Explainability and Interpretability: The degree to which humans can understand how an AI model works, including how it makes decisions or predictions. Crucial for trust and transparency in AI systems.

Exhibit 1. Key AI Terms and Definitions; *courtesy of Juthika Thakur, MD.*

work. When combined, LLMs and ASRs can offset each other's limitations, resulting in an ambient AI scribe that is more effective and practical for use in clinical settings (**Figure 2**).

Accuracy of the Generated Electronic Medical Record by Physician Versus Ambient AI

Clinical documentation may not always fully capture the substance of the patient encounter, with omissions that do not necessarily reflect the nature or intent of the interaction. In a study that included 36 physicians at a single centre, written encounters were compared with concealed audio recordings in unannounced patient encounters. The findings showed that 90% of patient records had at least one inaccuracy, with omissions and errors of additions that did not reflect the nature of the interaction captured in the concealed audio recordings.[1] In another study, among 136,815 patients who reviewed their outpatient clinic visit notes, 20% detected an error, and 40% of those considered the error to be serious.[2] The most common errors were in diagnoses, medical histories, medications, physical examinations, and misattributed notes to the wrong patient.[2] Many users of AI complain about the diagnostic accuracy of machine learning algorithms and LLMs. Unfortunately, the patient chart often falls short as an accurate reflection of the patient-physician encounter. In a regional pilot program that scaled AI based medical scribe solutions to 10,000 physicians, both patient and physician experiences improved.[3] A random review of 35 assessed notes across multiple clinical specialties revealed that over 90% met metrics such as freedom from hallucinations, conciseness, and accuracy (**Exhibit 2**). Although this study showed nominal improvements in charting time,[3] other studies have shown a reduction in time spent charting by 20.4%.[4] Domain-specific

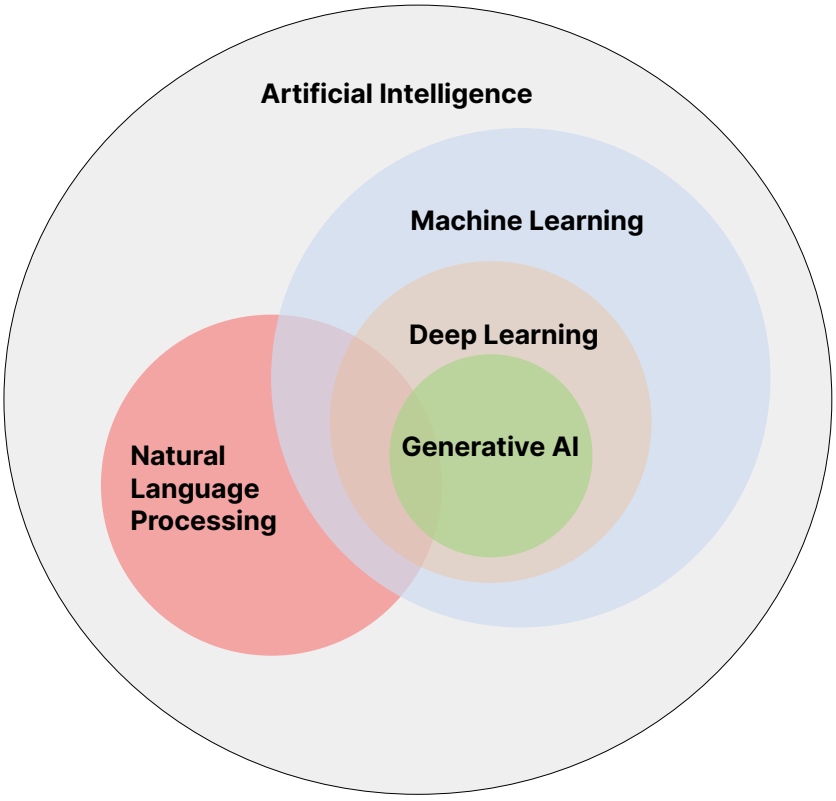

Figure 1. A Venn diagram depicting various subdomains of AI; *adapted from Rishabh Misra, 2024.*[7]

Automatic Speech Recognition

Basic Capabilities

- ✓ Capture, recognize, and interpret voices
- ✓ Identify different people in conversations
- ✓ Follow conversational patterns

Basic Limitations

- → Susceptible to environmental conditions – placement of microphones, background noise
- → May struggle with terms, accents, or dialects not part of its training data
- → May struggle with fragmented and non-linear conversations

Large Language Models

Basic Capabilities

- ✓ Identify words likely to go together given training data and context from the prompt
- ✓ Produce realistic and persuasive outputs
- ✓ Generate original content
- ✓ Learn from corrections of human users and trainers
- ✓ Improve and optimize performance over time

Basic Limitations

- → Quality of input determines the quality of output
- → Not designed to know if outputs are true or false
- → LLMs ad lib and hallucinate
- → Will struggle with concepts not part of its training data
- → Will parrot biases present in training data

Figure 2. Automatic Speech Recognition and Large Language Models Strengths and Weaknesses; *adapted from Information Services Centre of Effective Practice. (Anne Dabrowski, n.d.)*[8]

Attribute	Description of Ideal Note
Accurate	The note is true. It is free of incorrect information.
Thorough	The note is complete and free from omission and documents all of the issues of importance to the patient.
Useful	The note is extremely relevant, providing valuable information and/or analysis.
Organized	The note is well-formed and structured in a way that helps the reader understand the patient's clinical course.
Comprehensible	The note is clear, without ambiguity or sections that are difficult to understand.
Succinct	The note is brief, to the point and without redundancy.
Synthesized	The note reflects the AI scribe's understanding of the patient's status and ability to develop a plan of care.
Internally Consistent	No part of the note ignores or contradicts any other part.
Free from Hallucination	The note is free of hallucination and only contains information verifiable by the transcript.
Free from Bias	The note is free of bias and contains only information verifiable by the transcript and not derived from characteristics of the patient or visit.

Exhibit 2. Elements of an Ideal Clinical Encounter Summary; *adapted from Tierney et al., 2024.*[3]

A modified version of Physician Documentation Quality Instrument adapted for evaluating AI scribe outputs. Maximum value is 50, and each domain is based on a 5-point scale with 1 being not at all, and 5 being extremely likely.

training, with "humans in the loop" can mitigate against transcription inaccuracy. For example, by providing feedback and edits into the ambient AI module, outputs can be tailored to reflect the style of the clinician's encounters. Models that are not trained on healthcare-specific data are more likely to inaccurately capture healthcare lexicon, misinterpret drug names, inaccurately capture the complexities of medical conversations, and generally struggle with poor technology and noisy environments.

The Black Box, Explainability and Interpretability

Assessing diagnostic accuracy in LLMs remains challenging due to the opaque nature of their training datasets, which are often proprietary and not accessible to researchers. This lack of opacity limits the ability to identify, predict, or mitigate potential blind spots and biases in model outputs. Explainability refers to how clearly the processes behind an AI's output can be understood or justified. Effort has been made to put measures in place to provide some degree of explainability. For example, some systems include features such as the output note having "citations" back to specific segments of the audio transcript that the AI scribe summarized in a sentence format. This can mitigate against hallucinations but still requires intense physician effort to review and edit notes. Emerging research on physicians' use of AI indicates a genuine risk of automation bias, potentially leading to less thorough reviews and overlooking errors.[5] Ultimately, ambient AI vendors must strive to create technology that is explainable to all stakeholders, including patients and physicians on the use of AI scribes in medical encounters.

Privacy and Informed Consent with Using Ambient AI Technology

Clinicians have both ethical and legal obligations to obtain informed consent from patients. Patients may want to know information about how long recordings are stored, who can access the recording or AI summary, whether and how the physician will review them, whether the material collected is being used

to train generative AI, and the potential risk of reidentification. A key challenge is that once patients have given consent, they cannot retract it for future use of the data in model training. As a result, informed consent should also include a discussion on whether and how data will be deidentified and used to improve the algorithm. To mitigate these risks, physicians can select an ambient AI scribe that hosts the data on local servers and avoids using patient data in perpetuity.

Challenges in Record Keeping

Although the final chart entry must be retained per record retention rules, laws and regulations typically do not specify whether such audio recordings should also be included in the patient's chart. When recordings are intended to be destroyed rather than stored, it is important to have a policy that dictates the timing and process for destruction, making sure the patient chart has been accurately updated beforehand.[6] Some regulatory colleges require an additional consent form specifically for recording clinical encounters, and in some provinces, privacy laws necessitate written consent for any recordings to take place.[6] Physicians should carefully review applicable privacy laws and provincial regulations before implementing AI scribe technologies into their workflows.

Summary

Methods for thoroughly assessing the quality and safety of AI technologies—including LLMs—are still not fully established. As both the algorithms and regulatory frameworks continue to evolve, continuous benchmarking, evaluation, and monitoring will be required. Additionally, adoption and usage are likely to shift as new user groups and application areas emerge.

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Financial Disclosures

J.T.: None declared.

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Key Approaches to Pain Management in Hidradenitis Suppurativa

Helene Veillette, MD, FRCPC

Introduction

Many patients living with hidradenitis suppurativa (HS) experience a significant impairment in their quality of life. One of the most prominent symptoms in these patients is pain, which makes a major contribution to their overall distress. Despite its impact, pain control has been shown to be an unmet need for many patients.[1] Pain has physical, psychological and social impacts.[2] When poorly controlled, pain can lead to increased visits to emergency departments, a higher likelihood of self-medication, and generally poor disease control.[3,4] This review provides a clinical approach for managing pain in patients with HS.

Validating Pain

Assessing pain in patients with HS can be done efficiently during a consultation. A simple and effective tool is the Pain Numeric Rating Scale, which allows clinicians to assess the level of pain experienced on a scale from 0 to 10.[5,6] For example, you might ask, "In the last 7 days, how severe has your HS-related pain been, on a scale of 1–10, with 10 being the worst?" If you prefer, the question can be tailored to focus on the most recent HS flare. This short inquiry not only provides valuable insight into the patient's experience but also creates a bond of trust. It signals to the patient that you are a professional who understands the impact of HS on their quality of life, and that it is a priority for you.

Qualifying Pain

Pain in HS is multifaceted and can vary in its nature and duration[7] (nociceptive, neuropathic, nociplastic) or temporality (acute versus chronic). Nociceptive pain is typically acute and is caused by inflammatory tissue damage.

Neuropathic pain, experienced by 30% of HS patients,[8,9] is defined as pain initiated or

caused by a primary lesion or dysfunction in the central and/or peripheral nervous system. Patients often describe it as "shooting", "itchy", "blinding", 'stinging' or "burning". Some patients may also report pruritus, which should be carefully distinguished from pruritus induced directly by skin damage, such as irritant intertrigo, since the treatment approaches differ.

Nociplastic pain, also known as central sensitization, involves heightened sensitivity of the pain perception pathway within the central nervous system to stimuli that are normally at subthreshold.[10]

In terms of temporality, acute pain is often intense and closely associated with disease flares. Chronic pain, defined as lasting 12 weeks or more, frequently involves a combination of pain types (e.g., nociceptive and neuropathic).

Treating Pain

A variety of treatment options have been described for controlling the different types of pain experienced by patients with HS.[11] For practical purposes, this discussion focuses on the options I regularly use. However, if you are seeking more options, I encourage you to consult the article by Surapaneni et al.[11]

General Rules for Pain Management in HS

Effective pain control in HS begins with optimal disease management. Optimizing medical treatment is important for pain control. Indeed, a patient with fewer inflammatory lesions will experience less pain. However, clinical studies have shown that even after 12 to 16 weeks of therapy, many patients continue to report moderate pain, emphasizing the need for targeted pain treatment alongside disease control. When pain is caused by an abscess, the immediate priority should be incision and drainage. For patients experiencing pain from a few isolated lesions, intralesional triamcinolone acetonide, at concentrations ranging from 10 to 40 mg/ml, can be beneficial.

Managing Acute Nociceptive Pain

Pain management in HS should follow a stepwise approach based on the severity of symptoms. Step 1 describes mild pain, rated between 1 and 3 out of 10 on the pain scale, and treatment includes acetaminophen at a dose of 500 mg, taken as two tablets orally every 6 hours. Topical therapies such as diclofenac gel (e.g., Voltaren® Emulgel Extra-Strength) and lidocaine 4% or 5% cream (e.g., DeepRelief®, Dr Numb®) can provide localized relief. Non-pharmacological options such as heat or cold therapy, applied for 10 to 15 minutes, or menthol-based products (e.g., DeepRelief®), may also be beneficial.

Step 2 describes moderate pain, rated between 4 and 7 out of 10, requires additional therapeutic options. Nonsteroidal anti-inflammatory drugs (NSAIDs), such as naproxen (500 mg twice daily) or celecoxib (100 mg twice daily) can be introduced. A short course of oral corticosteroids such as prednisone (25 mg daily for 7 days) may be considered, ideally alongside a proton-pump inhibitor.

Step 3 describes severe pain, rated between 8 and 10 out of 10. Consider adding stronger analgesics. The options include tramadol (50 to 100 mg every 4 to 6 hours as needed) or oxycodone (5 mg every 4 to 6 hours as needed). These medications should be used cautiously. To minimize risk, you should always aim for the minimum effective dose and the shortest duration of treatment (**Table 1**).

Managing Chronic Pain

Chronic pain in HS often requires a multimodal approach. Pharmacologic treatments commonly include antidepressants and antiepileptics. Treatment with these drugs should be discussed with the family doctor, if possible. Psychotherapy, local wound care, and physiotherapy can also be provided.

Among antidepressants, amitriptyline, a tricyclic antidepressant (TCA), can be started at 10–25 mg daily, with a maximum dose of 150 mg daily. Venlafaxine, a serotoninnorepinephrine reuptake inhibitor (SNRI)

Pain Severity	Medication	Dose
Mild (1–3/10)	Acetaminophen	500 mg (2 tablets) every 6 hours
	Diclofenac gel (e.g., Voltaren®, Emulgel Extra-Strength®)	Topical application as directed
	Lidocaine 4% or 5% cream	Topical application as needed
	Heat or cold therapy	Apply for 10–15 minutes
	Menthol-based products (e.g., DeepRelief®)	Apply as directed
Moderate (4–7/10)	Naproxen	500 mg twice daily
	Celecoxib	100 mg twice daily
	Prednisone (short course)	25 mg daily for 7 days *with concomitant proton-pump inhibitor
Severe (8–10/10)	Tramadol	50–100 mg every 4–6 hours as needed
	Oxycodone	5 mg every 4–6 hours as needed

Table 1. Management of Acute Nociceptive Pain in Hidradenitis Suppurativa (HS); *courtesy of Helene Veillette, MD.*

Medication Type	Medication	Dose
Antidepressants	Amitriptyline	Start 10–25 mg daily; max 150 mg daily
	Venlafaxine	Start 37.5–75 mg daily; increase 75 mg/week to max 225 mg
Antiepileptics	Gabapentin	Start 100–300 mg daily; max 3600 mg/day
	Pregabalin	Start 75 mg twice daily; increase 75 mg every 2–4 weeks to max 600 mg/day

Table 2. Management of Chronic Pain in Hidradenitis Suppurativa (HS); *courtesy of Helene Veillette, MD, FRCPC.*

can be started at an initial dose of 37.5–75 mg daily, with gradual increases of 75 mg per week as tolerated, to a maximum of 225 mg daily. For antiepileptic options, gabapentin can be started at 100–300 mg daily, to a maximum of 3600 mg per day. Pregabalin is another choice, typically started at 75 mg twice daily, with dose increases of 75 mg every 2–4 weeks, to a maximum of 600 mg daily (**Table 2**).

Pregnant Women

For the pregnant patient, it is essential to assess the risk-benefit ratio and aim for using minimum effective doses. Certain drugs are recognized for their safety during pregnancy. These include acetaminophen, venlafaxine, amitriptyline, gabapentin, and pregabalin.[12] Procedural interventions may also be appropriate, such as the incision-drainage technique, intralesional injections with triamcinolone, and deroofing procedures may also be considered.[13,14]

Some medications require caution during specific stages of pregnancy. For example, naproxen should be discontinued at 30 weeks of gestation, and tramadol should not be used during the first trimester of pregnancy.[12]

Perioperative Pain

Clinical experience acquired over the years, supported by a number of studies,[15-17] has shown that combining different treatment strategies, both medical and surgical, can increase the probability of achieving therapeutic success in HS. Surgical techniques such as incision and drainage, deroofing, and both local and wide excisions can be performed to enhance treatment outcomes.

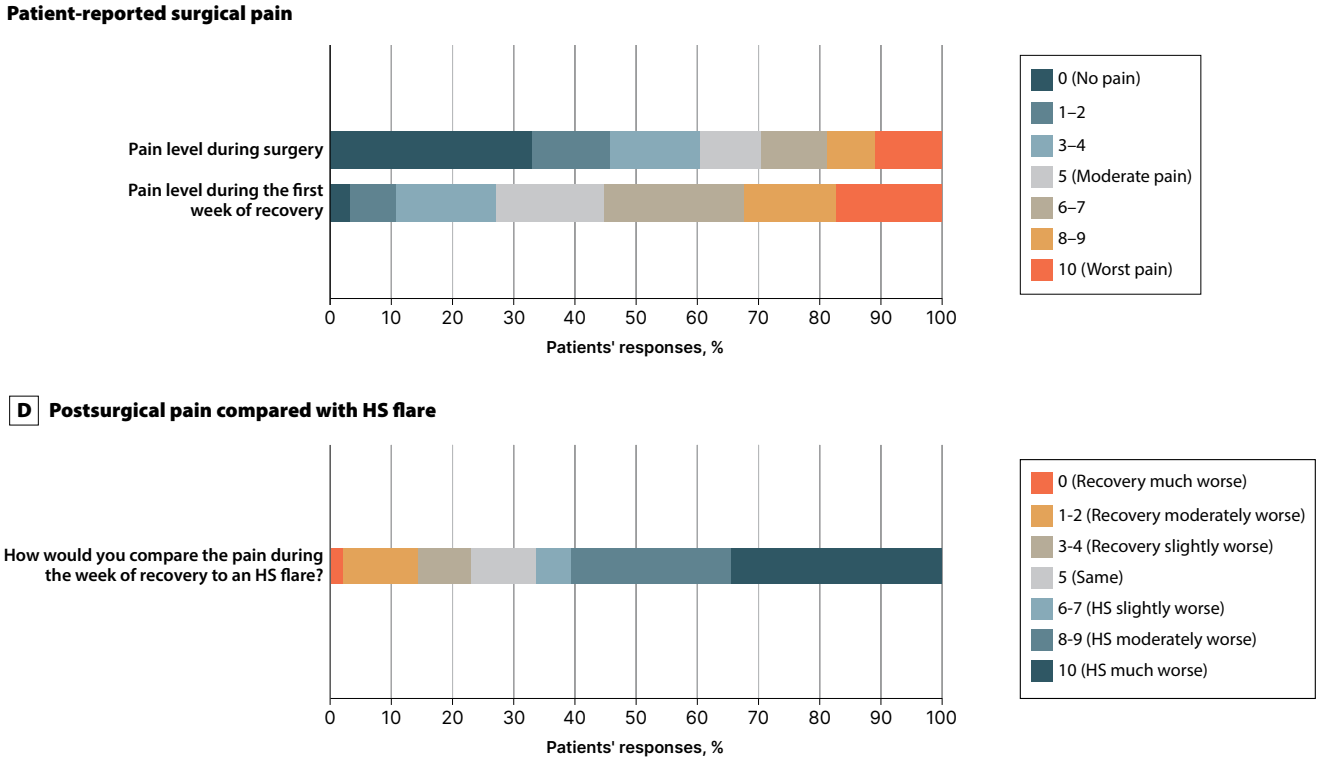

Figure 1. Patient reported surgical pain, and postsurgical pain compared with an HS flare; *adapted from Ravi, S et al, 2022.*[18]

Unfortunately for HS patients, the injection of anesthetics is particularly painful, especially in body folds, which are the typical location of HS lesions. This increased sensitivity makes it all the more important to minimize procedural pain during these surgical interventions.

Incision and drainage is a procedure designed to relieve the pain caused by a lesion, usually an abscess (see above). However, many patients with HS have had distressing experiences with this procedure in the past and remained traumatized because of the absence of anesthesia. Patients show excellent tolerance to incision and drainage if the procedure is performed under local anesthetic. Effective pain control can be achieved with a very superficial injection of anesthetic, creating a small, anesthetized zone just above the abscess cavity. Once this zone is anesthetized, a 4- or 6-mm punch can be used to incise the abscess in a manner that is much more tolerable for the patient.

Another simple surgical procedure to treat a recurrent lesion is deroofing. This procedure has demonstrated strong clinical efficacy and a high level of patient satisfaction. In a study[18] that included 78 patients and 194 roofing procedures, over 60% of patients described deroofing as painless or only slightly painful. However, during the first postoperative week, over 50% of patients experienced pain rated between 6 and 10 out of 10. Despite this, 65% of patients mentioned that the pain associated with an HS flare was more severe than the discomfort experienced after surgery (**Figure 1**).

Several strategies can help reduce the patient's pain when performing deroofing.[19] Administering a dose of analgesic, such as acetaminophen, ibuprofen, diclofenac, or celecoxib either shortly before or just after the procedure can help reduce pain during the first 24 hours postoperatively. For some patients, particularly those undergoing procedures in the inguinal and

perineal regions, the preoperative application of a topical anesthetic such as lidocaine/prilocaine cream (EMLA®) could be effective in reducing the pain. While study results are mixed, my experience is that some patients benefit from this technique.

It has been shown that preoperative anxiety is predictive of greater postoperative pain and even chronic pain. Creating a calm and reassuring environment, by ensuring patient comfort, offering distractions, or playing music can help reduce anxiety. For patients who are particularly anxious, a low dose of an anxiolytic may be considered (e.g., lorazepam 1 mg taken orally one hour before the procedure).

Inhaled methoxyflurane (Penthrox®) can also be useful for short-term relief of moderate to severe acute pain that is associated with trauma or interventional medical procedures. It is intended for use in conscious adult patients.[20] However, it is not recommended for patients under 18 years of age or for use during pregnancy.

It is important to bear in mind that in some care settings, patients are not allowed to bring their own medications. As the clinician, it is your responsibility to verify institutional policies and ensure that there are no contraindications to these treatments for the patient. In addition, for procedures involving sedation or significant discomfort, make sure that the patient has someone to accompany them home safely.

There are several practical techniques that can help reduce the pain associated with local anesthetic injections.[21] As with many routine clinical procedures you perform daily, using a small-gauge needle and injecting the anesthetic slowly will promote patient comfort. While some studies have explored whether the angle of needle insertion affects pain perception during anesthesia, findings remain inconclusive, and no clear consensus has been established.

Postoperative pain is an acute nociceptive pain. If the patient has no contraindications, they should be discharged with a prescription for acetaminophen 1 g orally every 6 hours and naproxen 500 mg orally twice daily as needed (you can refer to “Acute nociceptive pain” section above).

Conclusion

In summary, the pain experienced by patients with HS is an important issue that needs to be addressed in the context of a comprehensive management plan. Asking patients about their pain can be done quickly during an appointment and offers valuable insights into the patient’s experience. Unfortunately, few studies have focused on pain management in HS. However, the knowledge acquired from other diseases enables us to improve patients’ experience. Considering the anxiety and pain experienced during HS surgery and in the days that follow helps improve the patient’s overall experience. By identifying the medications you are comfortable using, and applying them thoughtfully, you can better support your patients and optimize their quality of life.

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Financial disclosures:

H.V.: Honorarium for presentations: AbbVie, BioJAMP, Celltrion, Janssen, Novartis, Sanofi, Bausch Health, Pfizer, Boehringer-Ingelheim, Incyte, UCB; **Advisory meetings:** Abbvie, Bausch Health, BioJAMP, Celltrion, Eli Lilly, Galderma, LEO Pharma, Janssen, Novartis, Sandoz, Pfizer, Sanofi, Sun Pharma, UCB, Boehringer-Ingelheim; **Clinical trials:** Sanofi, AnaptysBio, Boehringer-Ingelheim, AbbVie, Amgen, Bausch, Merck, Pfizer, Incyte, Novartis

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