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Practical Algorithm Guiding Seborrheic Dermatitis Treatment in Adults and Adolescents: New Evidence and Consensus Expert Opinion

Benjamin Ungar MD,[a] Andrew Alexis MD MPH,[b] Anneke Andriessen PhD,[c] Christopher G. Bunick MD PhD,[d] Eileen Cheever PA-C,[e] Adam Friedman MD,[f] Heather Woolery-Lloyd MD,[g] Raj Chovatiya MD PhD MSCI[h,i]

[a]Department of Dermatology, Icahn School of Medicine at Mount Sinai, New York, NY
[b]Clinical Dermatology, Diversity and Inclusion, Department of Dermatology, Weill Cornell Medicine, New York City, NY
[c]Radboud Academy; Radboud UMC, Nijmegen and Andriessen Consultants, Malden NL
[d]Yale Institute for Global Health, Yale School of Medicine, New Haven, CT
[e]Clearview Dermatology, Leominster, MA
[f]The George Washington University School of Medicine and Health Sciences, Washington, DC
[g]Skin of Color Division Dr Phillip Frost Department of Dermatology and Cutaneous Surgery University of Miami, Miller School of Medicine Miami, FL
[h]Rosalind Franklin University of Medicine and Science Chicago Medical School, North Chicago, IL
[i]Center for Medical Dermatology + Immunology Research, Chicago, IL

ABSTRACT

Background: Seborrheic dermatitis (SD) is a common, chronic inflammatory skin disease characterized by greasy, scaly plaques on the scalp or body, often with pruritus and impaired quality of life. The pathogenesis involves dysbiosis, including *Malassezia colonization*, excess sebum production, and immune dysregulation with Th17/Th22 activation. Although therapy historically targeted *Malassezia*, growing evidence supports anti-inflammatory strategies.

Methods: A systematic literature review identified current treatments and management strategies for SD. Of 399 articles screened, 77 met inclusion criteria and informed algorithm development. A draft algorithm was created from these data and refined through a virtual workshop with 6 board-certified dermatologists and 1 physician assistant.

Results: Experts recommended initial evaluation of disease extent and relevant comorbidities or lifestyle factors, along with patient education on disease etiology and scalp/hair care. Management is divided into (1) scalp and (2) face, trunk, or intertriginous involvement. First-line therapy emphasizes anti-inflammatory treatment with topical roflumilast 0.3% foam or corticosteroids. If disease persists, adjunctive antifungals are recommended. For nonscalp areas, alternatives include topicals, calcineurin inhibitors, tapinarof, or ruxolitinib. Recalcitrant or high-impact disease may warrant off-label systemic therapy such as isotretinoin, anti-IL-12/23 biologics, or oral antifungals.

Conclusions: This algorithm integrates an updated understanding of SD pathophysiology, prioritizing anti-inflammatory and immune-modulating therapies in management. It provides a practical framework for clinicians to tailor therapy based on disease location, severity, and response to prior treatment. Future studies are needed to validate outcomes and optimize long-term management strategies in diverse patient populations. Further research should clarify mechanisms and refine targeted therapeutic approaches further.

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INTRODUCTION

Seborrheic dermatitis (SD) is a common, chronic inflammatory skin disease that affects approximately 5% of the global population.[1] It is characterized by erythematous patches and plaques with white-yellow greasy scale, generally affecting sebaceous gland-rich areas such as the face, scalp, chest, and upper back.[2] Mild cases may present with isolated flaking; severe cases may involve erythematous, greasy, scaly plaques on the face and trunk. SD is associated with a significant negative impact on patients.[3] In a study evaluating patient-reported level of negative impact of SD, 76% of patients reported some degree of negative impact on their emotional well-being, and 78% reported a significant negative impact on their self-esteem.[3]

SD pathogenesis has historically been thought to be primarily driven by *Malassezia* yeast species, with first-line treatment consisting of antifungal therapy.[4] However, it has become

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increasingly understood that an altered immune response is a key step in SD pathogenesis. Rather than a direct causative role of *Malassezia*, recent research suggests that a dysregulated immune environment may allow for increased *Malassezia* growth and activity.[5,6] Recent tape-strip transcriptomic studies of SD skin reveal significant upregulation of T-helper (Th)17 and Th22 pathways, reflecting some similarities in its immune dysregulation with psoriasis.[7] In addition, significant downregulation of key epidermal barrier markers was observed, including CLDN1, CLDN8, FA2H, and ELOVL3, which suggests dysregulation in both barrier integrity and lipid metabolism in the skin.[7] These findings support a hypothesis of immune dysregulation and skin barrier dysfunction underlying inflammation-driven pathogenesis for SD that is distinct from psoriasis and atopic dermatitis. Thus, novel advances in understanding and treating SD are driving a shift in the traditional treatment paradigm, highlighting the need for practical guidance that incorporates emerging innovations.

Historically, the management of SD was based on symptom control and flare reduction, with treatment selection guided by severity and anatomic area. Frequently used topical therapies include topical corticosteroids (TCS), topical antifungals, topical calcineurin inhibitors (TCI), and both prescription and over-the-counter (OTC) and prescription antifungal shampoos.4 A topical phosphodiesterase-4 inhibitor, roflumilast 0.3% foam, was recently approved for the treatment of SD.[4] Systemic agents, such as isotretinoin, oral antifungals, and biologics (anti-IL-12/23 monoclonal antibodies), are also used for severe, refractory cases of SD.[2,4,8,9] In general, most topical and systemic agents used in SD have varying levels of evidence supporting their use. Topical roflumilast is the only agent that has high-quality, proven, and reliable clinical data supporting its efficacy and safety in the treatment of SD.[10,11]

Here, we present an expert-developed practical algorithm for the management of SD in adults and adolescents, informed by recent clinical and translational research to optimize treatment outcomes for patients with SD.

MATERIALS AND METHODS

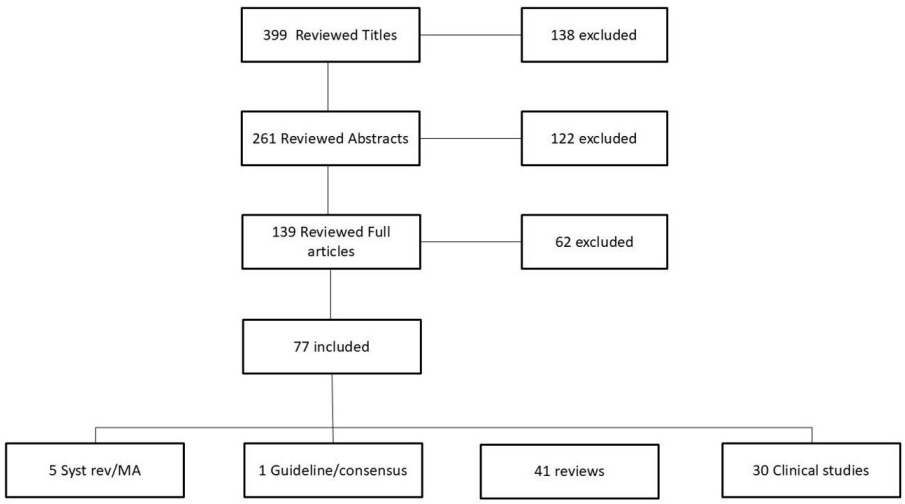
The algorithm was developed with input from a group of experts (BU, AA, CGB, EC, AF, HWL, RC) with extensive experience in SD treatment. Initial development was based on the results of a systematic literature review. A draft algorithm was then discussed among the experts and finalized for publication. This manuscript presents the algorithm and considerations that went into its development.

Systematic Literature Review

Articles addressing SD treatment for adults and adolescents (ages 9 and older) were included from 1 January 2015 to 1 November 2025, including guidelines, meta-analyses, systematic literature reviews, and clinical studies. The literature search was restricted to the past 10 years to inform the treatment algorithm. Review articles were included to represent older seminal literature that would otherwise be outside the 10-year window. The search was conducted on PubMed with Google Scholar as a secondary source. Search terms were as follows: seborrheic dermatitis AND (topical treatment OR antifungals OR corticosteroids OR calcineurin inhibitors OR PDE4 inhibitors) AND (systemic treatment OR JAK inhibitors OR biological therapies OR antifungals OR isotretinoin).

Two reviewers (A Andriessen and H Andriessen) first reviewed titles and abstracts, followed by the full articles. Disagreements were resolved through consensus. Exclusion criteria were pediatric interventions, nonEnglish language, and case reports/series (<20 patients). This search yielded 399 articles, which

FIGURE 1. Literature search results.



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TABLE 1.

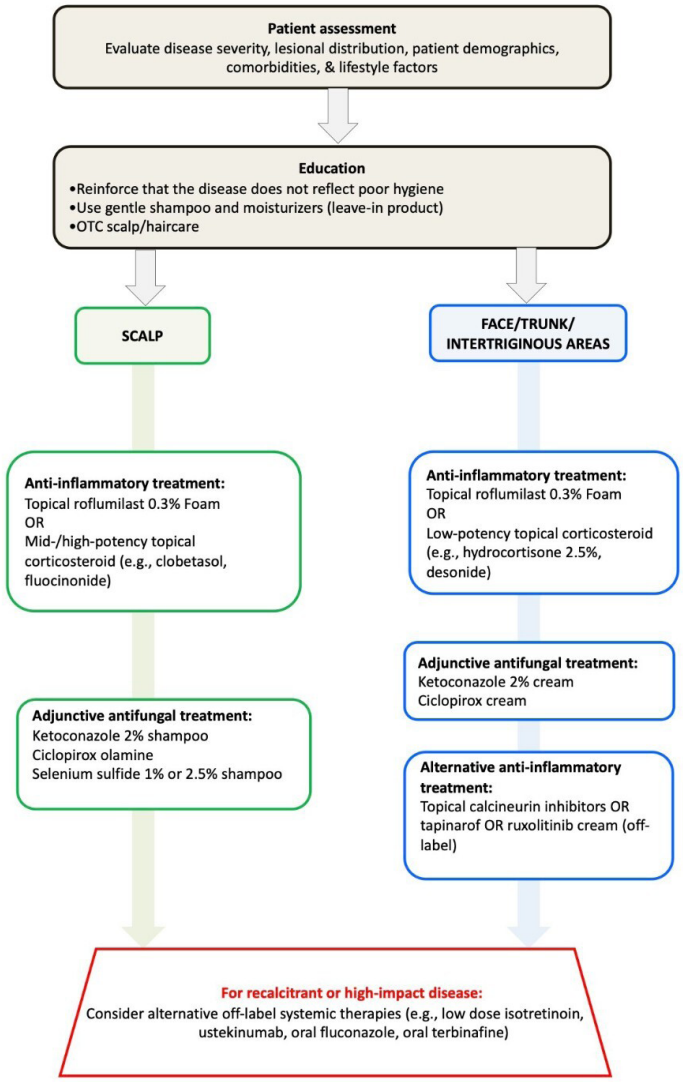
Grading of Clinical Studies on Skincare[12]	
A	Randomized, double-blind clinical trial of high quality
B	Randomized clinical trial of lesser quality
C	Comparative trial with severe methodologic limitations
1	Further research is unlikely to change confidence in the effect
2	Further research is likely to change confidence in the effect
3	Further research is very likely to change the effect
4	Any estimate of effect is very uncertain

were screened; after title evaluation, 138 were excluded, and after abstract evaluation, another 122 were excluded. Of the 139 articles evaluated in full, 62 were excluded. A total of 77 articles were extracted for content analysis, including 5 systematic reviews, 1 clinical guideline, 30 clinical studies, and 41 reviews. (Figure 1). Studies were graded in correspondence with the American Academy of Dermatology evidence-based guideline development process (Table 1).[12]

Role of Experts

A panel of 6 board-certified dermatologists and one physician assistant convened on December 11, 2025. Following the literature review and the creation of a draft algorithm supported by the literature, the experts participated in a virtual workshop to discuss and adapt the algorithm. The algorithm is based on expert opinions and discussions regarding the challenges of treating adult and adolescent patients with SD, and the need for clear, actionable guidance for clinicians in practice (Figure 2). The algorithm seeks to shift from traditional severity-based disease assessment to recommend therapies based on anatomic location, patient symptoms, and history of treatment resistance. Treatment recommendations are similar for adults and adolescents; however, pediatric disease requires a separate approach and is therefore not covered in this manuscript.

FIGURE 2. Algorithm for the treatment of seborrheic dermatitis in adults and adolescents.



Topical treatments
FDA approved: Roflumilast 0.3%, Ketoconazole 2%, Ciclopirox 1%, Selenium disulfide. Not FDA approved (off-label): Miconazole 2%, Betamethasone valerate 0.12%, Clobetasol propionate 0.05%, Hydrocortisone 1%/2.5%, Pimecrolimus 1%, Tacrolimus 0.1%, Ruxolitinib 1.5% Cream, Tapinarof 1% Cream.

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Seborrheic Dermatitis Algorithm for Adults and Adolescents

The algorithm reflects current evidence and expert consensus on the treatment of SD and begins with patient assessment and education, followed by the provision of treatment options based on anatomic location (Figure 2).

Patient Assessment

SD is a clinical diagnosis with clinician-determined severity. Clinicians often classify SD as "mild", "moderate", or "severe", or in clinical studies use an investigator global assessment (IGA) tool; however, these classifications do not consider the anatomic location of affected sites. Instead, the algorithm here proposes patient assessment by affected site, separating scalp SD from face/trunk/intertriginous areas. Patient demographics, such as skin type, must also be considered, as patients with skin of color may also present with hypopigmentation or hyperpigmentation, or as annular, scaly, erythematous patches or polycyclic pink or hypopigmented coalescing rings.[13] Severe, refractory cases of SD may raise clinical suspicion for immunodeficiency or neurologic disease.

SD may also be influenced by environmental conditions and lifestyle/cultural factors.[1] Thus, clinicians should take a thorough patient history as part of their evaluation and assessment. Understanding a patient's life will help inform their understanding of disease. For example, SD has been associated with urban environments, heavy traffic, temperature, ultraviolet (UV) index, stress, and humidity.[2] In addition, the use of added hair extensions and infrequent hair oil application (every 2 weeks) were strongly associated with increased SD incidence in African American females.[14] Further studies have also identified chemical relaxers, pomades, oils, and other scalp treatments were associated with SD flares. Thus, reviewing certain lifestyle factors and comorbidities is important in appropriately guiding patients.

Patient Education and Supportive Care

Effective SD management begins with patient education and support. Many patients are unaware of their SD diagnosis and report having difficulty finding information online about the disease. A healthcare provider (HCP) survey study reported that 86% of HCPs felt that "most patients had not heard of SD before their diagnosis".[3] Thus, educating patients about the etiology and course of SD is paramount for SD management.

Part of the discussion should center around hair care, including OTC products and washing habits. There has been a longstanding belief among patients that SD is associated with poor hygiene or poor skin/hair care habits (eg, insufficient hair washing or lack of moisturization on the face). However, evidence has been inconsistent in its support for this. In one study, hair washing every 1 to 2 weeks was not associated with a higher prevalence of SD in girls aged 1 to 15 years.[14] However, a 2-week study of an Asian population reported that a lower frequency of shampooing was associated with increased flakiness, itchiness, and *Malassezia* species burden.[15] More recently, a 3-week study in 60 women of African descent (aged 20–40 years) suggested that SD symptoms may begin as early as 1 week after the last hair wash, leading to recommendations of weekly to biweekly hair washing to prevent SD flares.[16] Thus, patient education should encourage appropriate hygiene regarding the patient's hair type and style without leading to over- or under-washing. The use of gentle shampoos and conditioners without irritating or allergenic ingredients should also be encouraged to further prevent exacerbation. OTC shampoos containing selenium sulfide or zinc pyrithione may help support dandruff treatment through antifungal and anti-inflammatory effects.[17] In a seminal study, Danby et al found that selenium sulfide 2.5% shampoo and ketoconazole 2% shampoo were both equally effective in the treatment of moderate to severe dandruff, making both ingredients appropriate for SD treatment regimens.[17] Additionally, multiple reports have linked decreased lipid levels in the stratum corneum with impaired barrier function and SD.[18,19] Few data exist to make clear recommendations regarding shampoos and body washes for SD. Thus, OTC products containing lipid-replenishing ingredients may prove helpful in SD treatment.

Topical Treatment

Topical treatments are a central pillar in SD treatment (Table 2). Topical antifungals have generally been regarded as first-line treatments, alongside TCS and TCI. However, there is a lack of overwhelming/strong data for topical antifungals and TCS.

Topical roflumilast

We propose monotherapy topical roflumilast 0.3% foam as a first-line anti-inflammatory, nonsteroidal treatment for SD on the scalp and face/trunk/intertriginous areas, reflecting our shift in understanding of underlying immunological drivers of disease (Figure 2). Roflumilast is a phosphodiesterase 4 inhibitor that decreases the production of proinflammatory cytokines by inhibiting cyclic adenosine monophosphate degradation and is an FDA-approved SD treatment for patients 9 years and older.[20] In the multicentre phase 3 vehicle-controlled trial, roflumilast 0.3% foam was applied once daily as monotherapy in patients aged 9 years or older with SD covering up to 20% of their body surface area (scalp, face, trunk, and/or intertriginous areas). By week 8, roflumilast-treated patients showed significant improvement in their SD, with 79.5% achieving IGA scores of 0 (clear) or 1 (almost clear).[20] Roflumilast also demonstrated a significant reduction in itch as early as week 2 compared to vehicle, with improvement in mean itch scores within 48 hours of the first application, making it an appropriate first-line option for SD management.[20] In addition, long-term efficacy and safety demonstrated a durable response at week 52, with no significant treatment-related safety signals.[10]

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TABLE 2.

Topical Treatment for Limited, Localized Seborrheic Dermatitis: Evidence and Side Effects

	Treatment	Class	FDA-Approved	Level of Evidence	Conventional Dosing Regimen	Common Potential Side Effects
Topicals	Ketoconazole 2%	Antifungal	Yes	A [1]	1–2×/day for 2–4 weeks, then 1×/week	Skin dryness, pruritus, stinging, burning, tingling
	Ciclopirox 1%	Antifungal	Yes	A [1]	2×/day for 4 weeks, then 1×/week	Skin dryness, pruritus, stinging, burning, tingling
	Miconazole 2%	Antifungal	No	A [1]	1–2×/day	Skin dryness, pruritus, stinging, burning, tingling
	Betamethasone valerate 0.12%	Corticosteroid	No	A [1,2]	For the body; 1–2×/day for up to 3 weeks, then 2×/week	Skin atrophy, telangiectasias, hypertrichosis, perioral dermatitis (all with long-term use)
	Clobetasol propionate 0.05%	Corticosteroid	No, off-label	A [1,2]	For the body; 1–2×/day for up to 3 weeks, then 2×/week	Skin atrophy, telangiectasias, hypertrichosis, perioral dermatitis (all with long-term use)
	Hydrocortisone 1%/2.5%	Corticosteroid	No	A [1,2]	For the face; 1–2×/day for 7–10 days, then 2×/week	Skin atrophy, telangiectasias, hypertrichosis, perioral dermatitis (all with long-term use)
	Pimecrolimus 1%	Calcineurin inhibitor	No, off-label	A [2,3]	1–2×/day for up to 4 weeks	Application-site burning, erythema, irritation, or itching
	Tacrolimus 0.1%	Calcineurin inhibitor	No, off-label	A [2,3]	1×/day for 4 weeks, then 2×/week	Application-site burning, erythema, irritation, or itching
	Roflumilast	Phosphodiesterase 4 inhibitor	Yes	A [4,5]	1×/day	Nausea, diarrhea, application-site pain
	Zinc pyrithione	Antifungal	Yes (combined with ketoconazole)	B [2]	2–3×/week for 2–4 weeks, then 1×/week or every other week	N/A
	Selenium disulfide	Keratolytic, antifungal	Yes	C [1]	2–3×/week for 2–4 weeks, then 1×/week or every other week	Itching, irritation
	Ruxolitinib 1.5% Cream	JAK inhibitor	No, off-label	C [2]	Twice daily for 4-6 weeks	Skin redness or irritation
	Tapinarof 1% Cream	Aryl hydrocarbon receptor agonist	No, off-label	C[2]	Once daily	Folliculitis

**For level of evidence explanation, see Table 1

Roflumilast is formulated as an emollient, fragrance-free, water-based foam without propylene glycol, polyethylene glycol, isopropyl alcohol, or ethanol, making it well tolerated by most patients and associated with a favorable safety profile.[20] Excipients were selected for their low irritant and allergenic potential. This also makes it an appropriate option for diverse skin and hair types, with minimal side effects. Minimal side effects from clinical trial data included nausea (1.6%) and application-site burning and stinging (0.3%).[20]

Topical Corticosteroids

If patients experience persistent pruritus or flaking, mid- to high-potency TCS may be used on short-term duration, such as clobetasol propionate 0.05% or fluocinonide 0.05% solution or shampoo on the scalp, to reduce inflammation.[21] For the face/trunk/intertriginous areas, low-potency topical corticosteroids such as hydrocortisone 2.5% or desonide 0.05% may also be considered as adjunctive treatment for recalcitrant areas. Use of TCS is limited by its adverse event profile, including skin atrophy, acne, striae, hypopigmentation, or rarely systemic absorption leading to suppression of the hypothalamic-pituitary-adrenal axis. These side effects may lead many patients to be "steroid-phobic," leading to poor treatment compliance and under-treatment.[22]

Topical calcineurin inhibitors

TCI, such as tacrolimus or pimecrolimus, may also be considered as a nonsteroidal, anti-inflammatory alternative. In a systematic review of 4 randomized controlled trials, topical

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TABLE 3.

Systemic Treatment for Limited, Localized Seborrheic Dermatitis, Evidence and Side Effects

	Treatment	Class	FDA-Approved	Level of Evidence	Conventional Dosing Regimen	Common Potential Side Effects
Systemics	Itraconazole	Antifungal	No, off-label	A [1]	200 mg/day for7days, then 200 mg weekly for 2–11 months	Nausea, headache, drug-drug interactions
	Fluconazole	Antifungal	No, off-label	A [1]	200 mg/weekly for 4 weeks, OR 300 mg/ week for 2 weeks	Elevated liver function tests, nausea
	Terbinafine	Antifungal	No, off-label	A [3]	250 mg/day for 4–6 weeks	Taste disorders, tachycardia, insomnia, gastrointestinal symptoms, migraines, rash; rarely liver injury
	Isotretinoin	Retinoid	No, off-label	A [4]	10–20 mg/day or every other day for 2–6 months	Cheilitis, nose bleeds, skin fragility, skin dryness, myalgias
	Ustekinumab	Monoclonal Antibody	No, off-label	C [4]	45 mg or 90 mg subcutaneously at week 0, 4, ~every 12 weeks thereafter	URIs

**For level of evidence explanation, see Table 2

pimecrolimus was found to be comparable in efficacy to topical corticosteroids and antifungals in reducing erythema, pruritus, and scaling in patients with SD.[23] These nonsteroidal options are appropriate for use on the face/trunk/intertriginous areas if needed for refractory SD treatment or in long-term therapy. Other nonsteroidal topical treatments have been shown to be effective for SD. An open-label trial of the topical JAK inhibitor ruxolitinib 1.5% cream demonstrated efficacy in treating SD by targeting JAK1 and JAK2, thereby reducing pro-inflammatory cytokine signaling and improving inflammatory skin disease.[24, 25] Similarly, topical tapinarof 1% cream, an aryl-hydrocarbon receptor agonist, has demonstrated efficacy in a 4-patient case series of patients with moderate-to-severe SD, with clinical improvement observed within 1 week of treatment (Table 2).[26]

Azoles

Antifungals may help reduce *Malassezia spp.* burden, potentially further exacerbating inputs into the proinflammatory state in SD.[18]

Topical azoles such as ketoconazole 2% shampoo, ciclopirox olamine, and selenium sulfide 1% or 2.5% shampoo may be used as adjunctive treatment for those with resistant forms of SD across different body areas (Figure 2). However, azole resistance has become an increasingly significant global concern.[27] Evidence shows that dermatophyte resistance to azoles is increasing, with rates reported as high as 19%.[28] This is largely driven by repeated exposure to topical azole therapies and inappropriate use of over-the-counter antifungals and steroid combinations.[27-29] Ketoconazole 2% shampoo used once weekly has been shown to be effective in preventing SD flares, and when combined with clobetasol propionate 0.05% shampoo, leads to greater efficacy in SD clearance.[30] Ketoconazole 2% shampoo (twice weekly application) has been shown to be superior to zinc pyrithione and 2.5% selenium sulfide in scalp SD.[31,32] However, more recent studies demonstrate earlier symptom relief with selenium sulfide than ketoconazole.[33] For the facial and trunk SD, multiple studies have demonstrated that 2% ketoconazole cream was superior to vehicle or equivalent in efficacy to 1% hydrocortisone cream.[34] Antifungal treatment may be helpful in skin of color populations, where SD was found to be amongst the top 5 most common dermatological conditions in African Americans compared to White, Hispanic, and Asian patients.[6]

Given emerging evidence supporting SD as an immune-driven disease, the use of nonsteroidal agents such as roflumilast may be a more appropriate option. Overall, treatment of SD with effective anti-inflammatory agents may reduce the necessity and frequency of antifungal application. Lessening antifungal use, such as ketoconazole, will improve antifungal stewardship and may reduce the development of azole resistance in the future.[27]

Systemic Treatment/Advanced Treatment

Severe, treatment-refractory SD with high BSA involvement may be treated with systemic agents (Table 3). Oral, low-dose (<0.5 mg/kg/day) isotretinoin has demonstrated efficacy in the treatment of SD, with patients reporting a significant reduction in erythema, scaling, and sebum production by 3 months of treatment.[35,36] Further studies compared 10 mg to 20 mg isotretinoin and reported no significant difference in reducing SD symptoms.[37] Isotretinoin reduces sebum production, thereby indirectly targeting *Malassezia* colonization by reducing the

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availability of its lipid substrate, while also conferring anti-inflammatory effects.

IL-12/23 targeting with ustekinumab has some evidence supporting its effectiveness in treating SD, with clearance after only a few doses and lasting remission.[9]

Oral antifungals have also been recommended for severe SD.[2] Terbinafine (250 mg/day for 4 to 6 weeks) has demonstrated efficacy in some cases, while oral fluconazole has shown more variable results.[2,38] Although these medications may benefit certain patients, current evidence suggests that SD management should move away from routine use of oral antifungals. Both terbinafine and fluconazole carry the risk of liver toxicity with long-term use and significant drug-drug interactions. In addition, their overuse may contribute to rising antifungal resistance while failing to address the primary inflammatory driver of SD.

CONCLUSION

SD is a common, chronic inflammatory skin disease characterized by immune dysregulation and skin barrier dysfunction leading to increased sebum production and commensal *Malassezia* growth. Clinical presentation may range from mild scalp dandruff to severe erythematous, greasy, scaly plaques on the scalp, face, and trunk, representing a wide SD spectrum. Here, we present a practical algorithm that integrates novel understanding of SD pathogenesis as an inflammatory condition with Th17/Th22 skewing, thereby requiring immune-modulatory treatments. The practical algorithm reflects a shift from antifungal to anti-inflammatory and immune-modulatory treatments. The length of treatment, adjunctive treatment, and maintenance regimens will be important to consider for each patient. Future studies and discussions will be necessary to evaluate these long-term regimens and to delineate SD treatment for pediatric patients under 9 years of age.

DISCLOSURES

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AUTHOR CORRESPONDENCE

Anneke Andriessen PhD

E-mail:..... anneke.a@tiscali.nl

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