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Seborrheic Dermatitis: A Contemporary View of Pathogenesis, Diagnosis and Treatment – A Narrative Review of the Current Literature

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Abstract

Background. Seborrheic dermatitis (SD) is a common, chronic, relapsing inflammatory dermatosis affecting sebum-rich areas, primarily the scalp, face, and intertriginous regions. Despite its generally benign nature, the relapsing course, visible distribution of lesions, and pruritus may substantially reduce patients' quality of life.

Methods. A targeted literature review was conducted in the PubMed and Cochrane Library databases. Systematic reviews, meta-analyses, randomized clinical trials, and up-to-date narrative reviews on seborrheic dermatitis were included. The search terms comprised: "seborrheic dermatitis", "Malassezia", "topical treatment", "anti-inflammatory treatment", and "maintenance therapy".

Results. Available evidence supports a multifactorial pathogenesis of SD, involving Malassezia yeasts, epidermal barrier dysfunction, and an abnormal host immune response. Topical antifungals remain the cornerstone of treatment, while short courses of anti-inflammatory

therapy are effective during flares. Increasing emphasis is placed on maintenance strategies and steroid-sparing approaches. New nonsteroidal anti-inflammatory agents show promising efficacy and safety.

Conclusions. Long-term control of seborrheic dermatitis is best achieved through a stepwise therapeutic approach aimed at rapid management of flares and maintenance strategies that reduce relapse frequency

Keywords: seborrheic dermatitis, Malassezia, topical antifungals, calcineurin inhibitors, roflumilast, maintenance therapy

1.Introduction

Seborrheic dermatitis (SD) is a chronic, relapsing inflammatory dermatosis that primarily affects sebum-rich areas such as the scalp, face, postauricular regions, and skin folds. The clinical picture includes erythema, greasy yellowish scales, and pruritus of variable intensity, with a tendency toward periodic exacerbations and remissions [1,2]. Although SD is not life-threatening, its chronicity, visible location of lesions, and relapsing symptoms may significantly impair patients' quality of life [4,18,19,37–40].

Seborrheic dermatitis is among the most common inflammatory skin diseases. In a meta-analysis and burden-of-disease assessment including numerous observational studies, the global prevalence of SD was estimated at 4.38% (95% CI: 3.58–5.17%), with substantial variability between populations and age groups [4]. The authors also demonstrated age-related differences, with a higher prevalence in adults compared with the pediatric population [4].

Current understanding of SD pathogenesis is based on a multifactorial model in which the interaction between Malassezia yeasts, the epidermal barrier, and the host immune response plays a key role [8,25]. Increasing evidence indicates that the decisive factor may not be so much the degree of colonization as the skin's abnormal response to pro-inflammatory products of yeast lipid metabolism [8,9]. Epidermal barrier dysfunction may further facilitate penetration of irritants and antigens, sustaining local inflammation [3,8,25].

This review aims to provide a current, practical summary of knowledge on seborrheic dermatitis, with particular emphasis on immunological aspects of pathogenesis and a stepwise therapeutic approach consistent with the latest scientific evidence

2. Methods

This paper is a narrative review of the current literature on seborrheic dermatitis. A targeted literature search was conducted in the PubMed and Cochrane Library databases to identify publications addressing the epidemiology, pathogenesis, clinical presentation, diagnosis, differential diagnosis, and treatment of seborrheic dermatitis. The search included studies published up to March 2026. Search terms included “seborrheic dermatitis”, “Malassezia”, “topical treatment”, “anti-inflammatory treatment”, “roflumilast” and “maintenance therapy”, used alone and in combination.

Priority was given to English-language systematic reviews, meta-analyses, randomized controlled trials, and recent narrative reviews that were considered clinically relevant and up to date. Older landmark publications were included when necessary to provide historical or mechanistic context.

The selection of studies was guided by their relevance to dermatology practice and their contribution to current understanding of seborrheic dermatitis, particularly with regard to antifungal therapy, anti-inflammatory treatment, maintenance strategies, steroid-sparing approaches, and newly emerging therapeutic options.

As this is a narrative review, the aim was not to perform a formal systematic synthesis of all available evidence, but rather to provide a structured and clinically useful overview of the current state of knowledge.

3. Pathogenesis of seborrheic dermatitis

3.1. Role of Malassezia yeasts

Malassezia yeasts are a physiological component of the human skin microbiome, particularly in sebum-rich areas. Their role in the pathogenesis of seborrheic dermatitis (SD) is well documented; however, it is now accepted that colonization alone is not sufficient for disease development [8,25]. The key is the interaction between the yeasts and the host skin, leading to activation of inflammatory processes.

Malassezia are lipophilic microorganisms that use lipids contained in sebum as an energy source. Their metabolism generates free fatty acids and other lipid products that may irritate the skin and act as pro-inflammatory stimuli [8,28]. In predisposed individuals, these products

induce an inflammatory response, leading to the development of characteristic erythematous and scaling lesions.

Studies have shown that, in SD, filamentous forms of Malassezia are observed more frequently, which correlates with the severity of inflammation and clinical symptoms [9]. This supports the concept that certain morphological forms of the yeasts may have greater pro-inflammatory potential, although the mechanisms of this phenomenon have not yet been fully elucidated.

3.2. Epidermal barrier dysfunction

An important element of SD pathogenesis is impairment of the epidermal barrier. Damage to skin barrier integrity promotes increased epidermal permeability, facilitating penetration of irritants, allergens, and microbial metabolites [3,20,25]. This leads to intensification of the inflammatory response and perpetuation of the chronic nature of the disease. Barrier dysfunction may be primary or secondary to inflammation. Metabolic products of Malassezia may further exacerbate barrier dysfunction, creating a vicious circle involving microbial colonization, barrier damage, and chronic inflammation [8,25]. Clinically, these processes are reflected by a greater tendency to relapses and persistence of symptoms despite reduction in yeast counts.

3.3. Host immune response

Growing evidence indicates that an abnormal host cutaneous immune response plays a key role in the development of seborrheic dermatitis (SD). Keratinocytes, acting as active immune cells, respond to microbial stimuli via pattern recognition receptors (PRRs), including Toll-like receptors (TLRs), which leads to activation of intracellular pro-inflammatory pathways and production of cytokines such as IL-1β, IL-6, TNF-α, and IL-8 (CXCL8), as well as inflammasome-related mediators including IL-18 [25, 26, 27].

In the pathogenesis of SD, mechanisms of innate immunity are considered important. Activation of epidermal cells and antigen-presenting cells leads to the release of inflammatory mediators responsible for erythema, pruritus, and accelerated epidermal scaling [3,8].

Increasing evidence also indicates involvement of adaptive immune responses within seborrheic lesions. Studies have shown elevated levels of cytokines characteristic of different T-cell response axes, including IFN-γ (Th1 axis) and IL-17 and IL-23 (Th17 axis), suggesting coexistence and overlap of several inflammatory pathways in the pathophysiology of SD [25, 26, 27]. This mixed immunological profile distinguishes SD from classic autoimmune dermatoses such as psoriasis, despite partial similarity of certain inflammatory mediators.

The importance of the immune component is supported by clinical observations that SD is more frequent and more severe in patients with immune dysfunction, including individuals with HIV infection and patients with neurological diseases [21,22,25]. In addition, proteomic data obtained from seborrheic scalp lesions indicate signatures of lymphocyte activation and a predominance of Th1-type responses, among others associated with increased IL-18 activity, further supporting the concept of a significant contribution of immunological pathways to disease pathogenesis [27].

The effectiveness of anti-inflammatory and immunomodulatory therapies, including calcineurin inhibitors and newer nonsteroidal anti-inflammatory agents, confirms that modulation of the immune response is an important and rational therapeutic target in the treatment of seborrheic dermatitis [6,7,10,11,26].

4. Clinical presentation and diagnosis

4.1 Clinical presentation

Seborrheic dermatitis (SD) shows a variable clinical presentation; typical features include erythema, scaling that may be greasy or dry, and pruritus of varying intensity [1,2]. Lesions are located mainly in areas with high sebaceous gland activity, which corresponds to the role of sebum and Malassezia yeasts in the pathophysiology of the disease [8,25]. The course of SD is chronic and relapsing, with periods of exacerbations and remissions, and symptom dynamics may be modulated by environmental factors and the patient's general condition [1,2].

The most common site of involvement is the scalp, both in adults and children, where the clinical picture includes erythema and fine- or thick-plate scaling, referred to in practice as dandruff; the scales may be dry or greasy [1,2,25]. In more severe forms, erythema and scaling may extend beyond the hairline, involving the frontal and temporal areas—described in the literature as the "seborrheic crown" [1,25].

Facial involvement is also common, especially the nasolabial folds, eyebrows, glabellar area, nasal alae, and postauricular regions; clinically, erythema and fine, often yellowish scaling predominate, sometimes accompanied by burning or pruritus [1,2,25]. In some patients, SD also affects intertriginous areas, including the axillary and inguinal folds, inframammary regions, and the umbilicus; in these locations, lesions are usually erythematous, more sharply demarcated, and scaling tends to be less pronounced than on the scalp [1,25]. The moist environment of skin folds can promote persistence of inflammation and complicate differential diagnosis, especially with respect to intertriginous fungal infections and inverse psoriasis [1,25].

A distinct clinical form is infantile SD, most commonly occurring in the first months of life and manifesting as thick, yellowish scales on the scalp ("cradle cap"), sometimes with erythema in skin folds [12,24]. In contrast to the adult form, infantile SD is usually mild and tends to resolve spontaneously, which may reflect pathogenetic differences and maturation of the skin barrier in early life [24].

Although SD rarely leads to systemic complications, available data indicate that, due to the visible nature of lesions, even mild symptoms may be associated with a significant deterioration in quality of life and psychosocial functioning, underscoring the importance of accurate diagnosis and long-term, individually tailored therapeutic management [18,19,23].

4.2 Factors affecting disease course and flares

Flares of seborrheic dermatitis are usually episodic and may be modulated by a range of environmental, behavioural, and iatrogenic factors. Among these, climatic conditions—low temperature and low air humidity—as well as reduced exposure to UV radiation are consistently highlighted, which correlates with the seasonality of symptom severity observed in clinical practice, particularly during the autumn–winter months [1,25].

An important, although difficult to quantify, determinant of disease course is neuropsychological factors. Stress, chronic psychological burden, and sleep disturbances may destabilize disease control, probably through effects on the skin neuro-immunological axis and mechanisms regulating epidermal barrier homeostasis [25]. Consequently, in some patients, relapses are observed despite adherence to a stable topical treatment regimen.

The course of SD may also be influenced by iatrogenic factors, including the use of certain systemic medications. Exacerbation of symptoms has been reported in the context of therapies affecting the nervous system or immune response, which may be clinically relevant, especially in patients with neurological disorders, in whom SD often runs a more severe course [25]. Regardless of systemic factors, local exposures are also important: use of irritating or occlusive products, excessive degreasing of the skin, and discontinuation of maintenance therapy after short-term improvement may promote relapses and persistence of inflammatory activity [1,2].

4.3. Clinical diagnosis and differential diagnosis

The diagnosis of seborrheic dermatitis (SD) is based primarily on clinical pattern recognition, including the morphology of lesions, their distribution in sebum-rich areas, and the chronic-relapsing course of the disease, usually without the need for routine additional testing [1,2,24,25]. In routine dermatological practice, the diagnosis is established on the basis of

physical examination supported by medical history. Particularly helpful are data regarding the recurrent nature of symptoms, aggravating factors and the presence of comorbidities, including neurological or immune disorders [1,2,24,25].

From an analytical perspective, the diagnosis of SD should not be understood as the identification of a single pathognomonic feature, but rather as the recognition of a characteristic clinical constellation. The combination of erythema, greasy or fine scaling, typical localization, and a superficial inflammatory pattern usually allows differentiation from other inflammatory dermatoses [1,2,25].

At the same time, clinical diagnosis requires awareness of overlap syndromes and atypical presentations. Scalp and facial lesions may resemble psoriasis, atopic dermatitis, contact dermatitis, or superficial fungal infection, while flexural involvement may mimic candidiasis or inverse psoriasis. Therefore, the diagnostic process in SD is best viewed not as a purely descriptive exercise, but as a structured clinical assessment balancing typical disease features against “red flags” suggesting an alternative diagnosis [1,2,24,25].

Table 1. Differential diagnosis of seborrheic dermatitis

Condition	Typical location	Distinguishing clinical features
Psoriasis	Scalp, elbows, knees, lumbosacral areaffoci	More sharply demarcated plaques; thick, dry, silvery scales; frequent other foci and/or nail changes; possible intermediate form (sebopsoriasis) [1,25].
Atopic dermatitis (AD)	Children: face and cheeks Older children and adults: flexures (antecubital, popliteal), neck	Usually more intense pruritus; frequent atopic history; different distribution of lesions; dryness and lichenification [1,2,25].
Tinea capitis (scalp dermatophytosis)	Scalp (more common in children)	Scaling patches with hair breakage (“black dots”), possible alopecia patches or kerion; confirmation on mycological examination (direct microscopy/culture) [1,2].

Intertriginous fungal infection / candidiasis (intertrigo)	Inguinal folds, axillae, inframammary regions	Maceration and bright-red erythema; in candidiasis typical satellite lesions; frequent burning and pruritus; mycological examination helpful (direct microscopy/culture) [1,25].
Contact dermatitis	Areas in contact with cosmetics: eyelids, hairline, postauricular area, face, neck	Clear temporal association with exposure; possible acute onset with edema, vesicles, and oozing; lesion borders correspond to contact area [1,25].
Rosacea	Central face (cheeks, nose, chin, forehead)	Telangiectasia; flushing and persistent erythema; papules and pustules; absence of typical greasy scales; frequent burning and skin sensitivity [1,25].
Perioral dermatitis	Perioral, perinasal and periocular area; usually sparing the vermilion border	Small papules or papulopustular lesions on an erythematous background; minimal scaling; frequent association with topical corticosteroids [1,25].
Pityriasis versicolor	Trunk, neck, shoulders; less often face	Hypo- or hyperpigmented patches with fine “branny” scaling; less erythema and pruritus than in SD; confirmation on mycological examination (direct microscopy/culture) [25].
Inverse psoriasis	Inguinal and axillary folds, inframammary and anogenital areas	Smooth, shiny, well-demarcated erythematous plaques with minimal scaling; typical psoriasis lesions often co-exist [1,25].

4.4. Role of additional tests

Additional investigations have a limited but targeted role in the diagnosis of SD. They are not required routinely, but may be justified in atypical, treatment-resistant, unusually severe, or diagnostically uncertain cases [1,24,25]. This is an important distinction, because overuse of ancillary tests in otherwise typical SD has little clinical value, whereas selective testing may

substantially improve diagnostic accuracy in borderline cases. Routine mycological testing is not recommended in typical SD, because Malassezia yeasts are part of the normal skin microbiome and their detection does not confirm the diagnosis [8]. However, mycological examination becomes clinically relevant when the morphology or distribution raises suspicion of dermatophytosis, candidiasis, or pityriasis versicolor, particularly in scalp disease with hair involvement or in intertriginous lesions with maceration [1,2,25]. In such settings, direct microscopy and/or culture may help distinguish SD from infectious mimickers. Histopathological examination may be considered in selected cases, especially when lesions are refractory to treatment or when psoriasis, contact dermatitis, or other inflammatory dermatoses cannot be excluded clinically. Nevertheless, histopathology in SD is not pathognomonic and usually serves a supportive or exclusionary rather than definitive diagnostic role [24,25]. This limitation should be emphasized, because biopsy may clarify the differential diagnosis in difficult cases, but rarely functions as a standalone diagnostic tool for SD itself. Laboratory testing is also not routinely indicated. It should be reserved for situations in which the clinical course suggests an underlying systemic condition or immunological impairment. Unusually extensive, severe, treatment-refractory, or abrupt-onset SD may justify broader clinical evaluation, including consideration of HIV infection in an appropriate clinical context [2,24,25]. This point is relevant not only diagnostically, but also analytically, as it underscores that SD may occasionally act as a cutaneous marker of broader systemic dysregulation rather than as an isolated skin disorder.

5. Therapeutic management of seborrheic dermatitis

Treatment of seborrheic dermatitis (SD) should consider the chronic and relapsing nature of the disease and its multifactorial pathogenesis, involving Malassezia yeasts, epidermal barrier dysfunction, and an inflammatory component [1,2,25]. The aim of therapy is not only to achieve symptom remission during flares but also to prevent relapses through appropriately selected maintenance treatment.

5.1. First-line treatment – antifungal therapy

Topical antifungal agents are the mainstay of treatment for seborrheic dermatitis (SD) and are recommended as first-line therapy regardless of lesion location [1,2,5]. Their effectiveness results from antifungal activity against Malassezia yeasts and from reducing the pro-inflammatory stimulus induced by products of lipid metabolism [8]. Because SD is chronic and relapsing, they may be used both to treat flares and intermittently to maintain remission,

especially on the scalp [2,5].

In a Cochrane review including 51 studies with 9,052 patients, topical ketoconazole significantly reduced the risk of persistent symptoms after 4 weeks of treatment compared with placebo (RR 0.69; 8 studies; n = 2,520), with comparable efficacy to topical glucocorticosteroids and a significantly lower rate of adverse events (RR 0.56) [5]. In the same review, ciclopirox was 21% more effective than placebo in achieving clinical clearance of lesions, without increasing the risk of adverse events [5]. Available data do not clearly indicate the superiority of one antifungal over another; however, the best-documented efficacy is for ketoconazole and ciclopirox [5].

In scalp SD, antifungal cleansing preparations used in induction and maintenance regimens are the standard. Reviews and clinical recommendations describe the use of ketoconazole 2% or ciclopirox 1% initially daily for 2–4 weeks, and then 1–2 times per week for maintenance treatment, with the recommendation to leave the product on the scalp for at least 3–5 minutes before rinsing [2,16].

In milder forms, over-the-counter preparations are acceptable, including those containing selenium disulfide (1%) or zinc pyrithione, typically used twice weekly both during induction and for maintenance treatment [2,15]. Data from a randomized 2024 study (n = 64; 4 weeks) showed that a shampoo with 1% selenium disulfide was comparably effective for short-term reduction of disease severity to a shampoo with 2% ketoconazole in adults with moderate-to-severe scalp SD, with a reduction in disease severity (SSSD) of 71% and 69%, respectively, on day 28 ($p < 0.001$) [29].

In non-scalp locations, creams, gels, or foams are preferred. The most commonly described regimens include ketoconazole 2% applied 1–2 times daily for 2–4 weeks, followed by intermittent use in the event of relapse [1,2,17, 25]. An alternative is ciclopirox as a cream or gel, usually applied twice daily for 2–4 weeks [2]. Review literature also mentions other topical azoles (e.g., sertaconazole) as therapeutic options for facial SD, using short induction courses followed by intermittent use in relapsing disease [2]. Selection of formulation and skin tolerability are crucial, as irritants (alcohols, fragrances, occlusive bases) may intensify burning and erythema [1,2].

Topical antifungals have a favourable safety profile; the most commonly reported adverse events include local irritation, burning, and transient worsening of pruritus [5].

5.2. Anti-inflammatory treatment during flares

In cases of moderate or more severe seborrheic dermatitis (SD), short-term use of topical anti-inflammatory agents as an adjunct to antifungal therapy is justified [6]. The goal is rapid control of erythema, pruritus, and inflammation, which during flares often predominate over scaling [1,2].

Low- to medium-potency topical glucocorticosteroids show high efficacy in the short-term reduction of inflammatory symptoms of SD. In a Cochrane systematic review including 36 randomized clinical trials with more than 2,700 patients, topical glucocorticosteroids significantly increased the proportion of patients with clinical improvement or symptom resolution compared with placebo over 2–4 weeks of follow-up [6]. In comparative analyses that reported adverse events, their frequency was higher in glucocorticosteroid-treated groups than in groups using topical antifungals. In clinical practice during flares, agents with a milder profile are used primarily, especially on the face, such as hydrocortisone or desonide, whereas on the scalp formulations that facilitate application (solutions, foams, steroid shampoos) are used more often [2]. A clinical review by the AAFP described, among others, use of fluocinolone 0.01% as a solution/shampoo or betamethasone as a foam in short courses, and in the most severe forms also short-term use of clobetasol 0.05% shampoo, sometimes in an alternating regimen with an antifungal shampoo [2]. Typically, topical glucocorticosteroids are applied 1–2 times daily in short courses (usually 1–2 weeks) and then discontinued once symptom control is achieved or switched to steroid-sparing strategies [1,2,6].

Given the safety profile, use of topical glucocorticosteroids should be time-limited, especially on the face and other sensitive sites. Prolonged exposure is associated with the risk of adverse effects such as skin atrophy, telangiectasia, worsening acne or perioral dermatitis, and impairment of epidermal barrier function [1,2,6]. For this reason, glucocorticosteroids are most often used as “rescue” treatment for acute flares, followed by a switch to steroid-sparing strategies.

An alternative to glucocorticosteroids is topical calcineurin inhibitors, including tacrolimus and pimecrolimus, which have anti-inflammatory effects without the risk of skin atrophy [6,13,14]. A Cochrane review confirmed that calcineurin inhibitors are significantly more effective than placebo in reducing erythema and scaling in patients with SD, particularly on the face [6]. In direct comparisons with topical glucocorticosteroids, they showed comparable clinical efficacy, with a more favourable safety profile in the context of recurrent and long-term use [6]. In clinical practice, these agents are used especially for facial lesions. The most commonly used are tacrolimus ointment (e.g., 0.1%) and pimecrolimus cream (1%), applied 1–2 times daily

during flares (usually for 1–2 weeks), and in relapsing disease intermittently (e.g., 2–3 times per week) to reduce relapses and exposure to glucocorticosteroids [6,7].

The most observed adverse effects of calcineurin inhibitors are transient local symptoms such as burning or a sensation of warmth at the application site, which are usually mild and resolve spontaneously during continued treatment [6].

5.3. Maintenance treatment and glucocorticosteroid-sparing strategies

Due to the relapsing nature of seborrheic dermatitis, maintenance therapy is a key element of management, aiming to sustain remission and reduce the frequency and severity of relapses [1,25]. In practice, maintenance treatment is based on intermittent (proactive) use of agents with documented efficacy, with a preference for regimens that limit exposure to glucocorticosteroids, particularly on the face [1,6,7].

For the facial form, available clinical data support the use of topical calcineurin inhibitors as a glucocorticosteroid-sparing strategy. In a clinical study of tacrolimus maintenance therapy after symptom control, an intermittent regimen (e.g., 2–3 times per week) was shown to prolong time to relapse and reduce the risk of re-exacerbation, with a favourable safety profile and no risk of skin atrophy [7]. From a practical perspective, this primarily concerns preparations such as tacrolimus (e.g., 0.1% ointment) and pimecrolimus (1% cream), which are particularly useful on sensitive facial areas [6,7].

Another important pillar of maintenance therapy is periodic use of antifungal preparations. A systematic review showed that topical antifungals are effective in controlling SD symptoms and may be used not only to treat flares but also intermittently to reduce relapses [5]. This strategy is particularly important for the scalp, where antifungal cleansing products (e.g., ketoconazole 2% or ciclopirox 1% shampoo) are most often used; after improvement, they are implemented in maintenance regimens (e.g., 1–2 times per week) [2,5]. In clinical practice, emphasis is placed on planning maintenance therapy and educating the patient (e.g., identifying triggers and proper skin care), which facilitates long-term relapse control [1,25].

This approach corresponds to the clinical observation that regular, planned maintenance treatment (so-called proactive therapy) is usually more effective than reacting only when relapse occurs. In practice, this means implementing a “flare–maintenance” regimen: short, more intensive treatment during exacerbation (e.g., a few days of an anti-inflammatory agent), followed by continuation of lower-intensity therapy aimed at controlling Malassezia colonization and restoring the barrier (e.g., regular use of antifungals and emollients, with

individualized frequency). Such an approach better reflects understanding of SD as a chronic, relapsing disease, while helping to limit exposure to glucocorticosteroids and improve patient adherence.

5.4. New therapeutic options – nonsteroidal anti-inflammatory agents

In recent years, therapeutic options in seborrheic dermatitis have expanded to include nonsteroidal anti-inflammatory agents. The best-documented option in this group is the phosphodiesterase-4 (PDE-4) inhibitor roflumilast in a foam formulation [10,11]. PDE-4 inhibition increases cAMP levels and attenuates activation of pro-inflammatory pathways (including those dependent on NF-κB), which may translate into reduced erythema, scaling, and pruritus [30,32,33]. In randomized clinical trials, including a phase III study in adults with moderate-to-severe SD, after 8 weeks of treatment, the proportion of patients achieving meaningful clinical improvement was higher in the roflumilast group than in the placebo group, and the effect was also observed at earlier time points [10]. Improvement also included patient-relevant outcomes such as itch and overall disease assessment [10,33]. The safety profile was favourable: adverse event rates were low and comparable to placebo, and long-term data (up to 52 weeks) did not indicate increasing risk of adverse events during chronic use [11]. In addition, pharmacokinetic data for topical roflumilast formulations suggest high drug concentrations in the skin with low systemic exposure, although available analyses come mainly from studies conducted in other inflammatory dermatoses [31]. From a clinical perspective, roflumilast may be a useful option in patients with a relapsing course and in glucocorticosteroid-sparing strategies. However, availability of topical roflumilast formulations and the scope of indications may vary by country and current regulatory decisions [10,11].

5.5. Individualized approach and long-term management

Applying a stepwise approach that combines antifungal therapy, short-term control of inflammation, and long-term maintenance strategies helps optimize disease control and improve quality of life in patients with seborrheic dermatitis [1,5–7,10,11,25]. Increasing attention is also being paid to the role of epidermal barrier dysfunction and the skin microbiome, which is reflected in the growing importance of skin-care measures and dermocosmetics as adjuncts to pharmacological treatment (e.g., gentle cleansers, products that minimize irritation and support barrier function) [20].

Effective management of seborrheic dermatitis requires individualization of treatment according to lesion location, symptom severity, comorbidities, and response to previous

therapies [1,2]. It is recommended to adjust the duration of topical treatment to clinical response and skin tolerability, treating the time frames described in the literature as guidance rather than strict rules [1,2,6]. In moderate-to-severe cases or those resistant to topical therapy, short courses of oral antifungals (e.g., itraconazole, fluconazole, terbinafine) have also been described; however, this is not a first-line approach and requires an individual assessment of the risk of adverse effects and drug interactions [34,35,36,38].

Table 2. Therapeutic options in seborrheic dermatitis

Treatment category	Therapy	Mechanism of action	Clinical indications
Topical antifungal agents	Ketoconazole 2% Ciclopirox olamine 1% Selenium disulfide 1% Zinc pyrithione	Inhibition of ergosterol synthesis or disruption of the Malassezia cell membrane; reduction of pro-inflammatory lipid metabolites	Mild to moderate SD (scalp and non-scalp locations); first-line treatment
Topical anti-inflammatory agents	Low- to medium-potency glucocorticosteroids (e.g., hydrocortisone, desonide, fluocinolone)	Anti-inflammatory, immunosuppressive and antiproliferative effects	Short-term treatment of flares of moderate and severe SD
Immunomodulating agents	Tacrolimus 0,1% Pimecrolimus 1%	Inhibition of calcineurin and T-cell–dependent production of pro-inflammatory cytokines	Mild to moderate facial SD; glucocorticosteroid-sparing treatment and maintenance therapy

Nonsteroidal anti-inflammatory agents	Roflumilast 0,3% (foam; PDE-4 inhibitor)	Increased cAMP levels leading to inhibition of NF-κB–dependent inflammatory pathways and reduced cytokine production	Moderate to severe SD; long-term treatment and “steroid-sparing” strategies

6. Conclusion

Seborrheic dermatitis is a common, chronic, relapsing inflammatory dermatosis with a multifactorial pathogenesis involving a complex interaction between Malassezia yeasts, impairment of epidermal barrier function, and an abnormal host immune response. Diagnosis is based primarily on the clinical presentation and the characteristic distribution of lesions, while differential diagnosis remains crucial in atypical cases, those resistant to treatment, or those raising diagnostic uncertainty. Therapeutic management should reflect the chronic nature of the disease and the need for long-term symptom control. In clinical practice, strategies that reduce the risk of treatment-related adverse effects—especially on sensitive sites (e.g., the face)—as well as improved adherence through well-tolerated formulations and regimens that can be maintained in everyday life are particularly important. The emergence of new nonsteroidal anti-inflammatory agents is a meaningful addition to existing therapeutic options and underscores the importance of modulating the inflammatory response as one of the key treatment targets. At the same time, the evidence base for seborrheic dermatitis remains partly limited by study heterogeneity, diversity of assessment scales, and relatively short follow-up in many clinical trials. Further research, including work focused on immunological mechanisms and identification of markers of treatment response, may enable a more personalized therapeutic approach in the future. Effective management of seborrheic dermatitis therefore requires individualized treatment, systematic patient education, and viewing the condition as one requiring long-term control rather than solely episodic symptomatic therapy.

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