ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 11| Issue: 1| January 2025|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2024: 8.402 || ISI Value: 1.188

SEBORRHEIC DERMATITIS, SCOPING REVIEW

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Article DOI: https://doi.org/10.36713/epra20013
DOI No: 10.36713/epra20013

SUMMARY

Introduction: *Seborrheic dermatitis (SD) is a common skin disease, where its signs and symptoms may differ according to skin color, associated medical conditions, environmental factors, and vehicle preference. Seborrheic dermatitis is a common skin condition in infants, adolescents and adults, characterized by scaling, erythema and itching, especially on the scalp, face, chest, back, armpits and groin.*

Objective: *to detail the current information related to seborrheic dermatitis, etiology, epidemiology, pathophysiology, histopathology, clinical presentation, evaluation, differential diagnosis, treatment and prognosis.*

Methodology: *a total of 29 articles were analyzed in this review, including review and original articles, as well as clinical cases, of which 23 bibliographies were used because the other articles were not relevant for this study. The sources of information were PubMed, Google Scholar and Cochrane; the terms used to search for information in Spanish, Portuguese and English were: sebopsoriasis, seborrheic dermatitis, seborrheic eczema, dandruff and pityriasis capitis.*

Results: *The most relevant results indicate that seborrheic dermatitis has a significant global prevalence, affecting up to 5% of the population, with a higher incidence in individuals with HIV-AIDS. Its pathogenesis has been identified as involving a combination of factors, including Malassezia spp. colonization of the skin, alteration of the cutaneous microbiota and a deregulated immune response. The most effective treatments are topical, particularly antifungal agents such as ketoconazole and low potency corticosteroids, although treatment should be tailored to the individual characteristics of each patient. Despite being a chronic disease in many cases, early diagnosis and proper management allow control of symptoms and improve the quality of life of patients.*

Conclusions: *Seborrheic dermatitis is a common skin disease, with significant prevalence in various ages and groups, especially in people with HIV-AIDS. Its pathogenesis involves factors such as colonization by Malassezia spp, alterations in the cutaneous microbiota and immune dysregulation. Although it may be self-limiting in some cases, in others it presents a chronic course with relapses. The most effective treatment includes topical antifungal and anti-inflammatory agents, adapted to the severity and location of the lesions. Early diagnosis and appropriate treatment are key to control symptoms and improve the quality of life of patients.*

KEY WORDS: *dermatitis, seborrheic, eczema, skin.*

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INTRODUCTION

Seborrheic dermatitis (SD) is a common skin disease, where signs and symptoms may differ according to skin color, associated medical conditions, environmental factors and vehicle preference. Seborrheic dermatitis is a common skin condition in infants, adolescents and adults, characterized by scaling, erythema and itching, especially on the scalp, face, chest, back, armpits and groin. Seborrheic dermatitis is a clinical diagnosis based on the location and appearance of the lesions. The presentation may differ according to skin type and may show hyperpigmentation or hypopigmentation, with or without erythema and minimal or no desquamation. The skin changes are thought to result from an inflammatory reaction to a common skin microorganism, the yeast Malassezia. Although this pathogenesis is not certain, it is generally accepted that there are 3 crucial items:

- Lipid secretion by the sebaceous glands.
- Colonization by Malassezia spp.
- Some form of immune dysregulation that predisposes the individual to SD.

The use of different terms such as sebopsoriasis, seborrheic dermatitis, seborrheic eczema, dandruff and pityriasis capitis shows the clinical range of SD, as well as the controversy regarding its etiology, sometimes being considered a form of dermatitis, a precursor of psoriasis or a fungal disease. Management with antifungal agents such as topical ketoconazole is essential in the treatment of seborrheic dermatitis of the face and body. Topical therapies, which encompass antifungal agents and low potency corticosteroids, are the mainstay of treatment, but may be limited by efficacy and side effects(1-3).

METHODOLOGY

A total of 29 articles were analyzed in this review, including review and original articles, as well as cases and clinical trials, of which 23 bibliographies were used because the information collected was not important enough to be included in this study. The sources of information were Cochrane, PubMed and Google Scholar; the terms used to search for information in Spanish, Portuguese and English were: sebopsoriasis, seborrheic dermatitis, seborrheic eczema, dandruff and pityriasis capitis.

The choice of bibliography exposes elements related to etiology, epidemiology, pathophysiology, histopathology, clinical presentation, evaluation, differential diagnosis, treatment and prognosis of seborrheic dermatitis.

DEVELOPMENT

ETIOLOGY

There are several factors related to the development of seborrheic dermatitis, its disparate behavior has given rise to many proposals about its cause and pathogenesis. The occurrence seems to be linked to the interaction of normal microscopic skin flora (especially Malassezia spp.), the composition of lipids on the skin surface and individual susceptibility, although neither the level of sebum generated nor the amount of yeast appear to be significant factors(4-6).

EPIDEMIOLOGY

Around the globe the prevalence of DS is 5%, however, the majority of its non-inflammatory variant, dandruff, probably approaches 50%. DS involves all ethnic groups. It has a bimodal peak, with a peak in the first 3 months of life and from adrenarche to a second peak after the fourth decade. In Australian preschool children, the prevalence of this pathology was almost 72% at 3 months of age, then went to an overall incidence of 10%. There are studies showing that 14% of middle-aged and elderly adults had DS. In individuals with HIV-AIDS, 35% of those with early HIV infection have DS, and the prevalence reaches 85% in individuals with AIDS(7-10).

PATHOPHYSIOLOGY

Among the proposed mechanisms for the pathogenesis of DS are:

- Alteration of the skin microbiota.
- Impaired immune reaction to Malassezia spp. related to decreased T-cell response and complement activation.
- Increased presence of unsaturated fatty acids on the skin surface.
- Alteration of cutaneous neurotransmitters.
- Abnormal keratinocyte detachment.
- Alterations of the epidermal barrier related to genetic factors.

The role of Malassezia spp. encompasses sebum degradation and consumption of saturated fatty acids, modifying the lipid balance on the skin surface(11,12).

HISTOPATHOLOGY

The dermatopathology of DS is nonspecific, however, the superficial and infundibular epidermis usually shows a superficial perivascular infiltrate of lymphocytes, acanthosis, focal spongiosis and focal parakeratosis. Parakeratosis of the shoulder" means conglomeration of scales and crusts near the infundibular ostia. Malassezia spp. may be seen in the stratum corneum. Histologic progression from acute to chronic SD characteristically denotes a transition from spongiosis to psoriasiform hyperplasia and the development of a lymphocytic lichenoid infiltrate. Severe SD is associated with keratinocyte necrosis, destruction of the focal interface and leukocytoclasia(5,13).

CLINICAL PRESENTATION

The distribution of lesions is the most prominent clinical feature of seborrheic dermatitis (SD), with lesions occurring in areas where the skin is rich in sebaceous glands, especially on the scalp and face. Inflammatory SD is usually asymptomatic, although it frequently coexists with atopic dermatitis. In contrast, pruritus is common in atopic seborrheic dermatitis (ASD), especially on the scalp, and patients often report a burning sensation, but do not usually have a history of atopic dermatitis. SD typically shows salmon-colored papules and plaques with fine scaling and a yellowish crust, often described as a scaly greasy crust. It may appear in one or several areas, with less scaling on flexor surfaces and lesions whose borders tend to be poorly defined.

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The mildest form of SD is a non-inflammatory variant commonly known as pityriasis capitis or pityriasis capitis seca. It affects the scalp and the "beard area", and is associated with the shedding of small light-colored skin flakes, which are often visible on dark clothing as "dandruff". The sudden onset of severe SD should be an indication of the presence of HIV-AIDS, and its early identification and diagnosis greatly improves long-term outcomes. Common clinical manifestations include facial redness, scaling and dandruff. In darker skin, there may be persistent dyschromia with variable hyperpigmentation and hypopigmentation.

Other conditions associated with Malassezia spp. may occur, such as pityriasis versicolor and Pityrosporum or Malassezia folliculitis in adults, and neonatal cephalic pustulosis. Lesions on the anterior chest usually have a morphology similar to psoriasis, but frequently have a petaloid appearance; this type of annular lesions is commonly seen on the face in individuals with darker skin phenotypes. A rare variant of pityriasis (with collar-like scales mimicking pityriasis rosea) may also occur.

TEA

The face, scalp and thorax are the areas most frequently affected by ASD, with approximately 88%, 70% and 27% of cases presenting lesions in these areas, respectively. In the head and neck, ASD has a symmetrical distribution and mainly affects the central third of the face, including the malar region, the center of the forehead, the eyebrows (especially their middle parts), the retroauricular area and the external auditory canal. In addition, ASD often impacts the nasolabial and alar folds, and blepharitis is a common finding when it affects the anterior (lash) line.

Psoriasis should be differentiated from adult sebopsoriasis. It is characterized by hardened red papules and plaques with well-defined borders and loose, silvery lamellar scales. The nails may show psoriatic changes and the Auspitz sign is usually positive. The existence of sebopsoriasis as a separate clinical entity has been a matter of debate(5,14-16).

EVALUATION

There are some tests that are useful in the diagnosis of DS and related pathologies such as:
Histology and direct immunofluorescence.
Examination of skin scrapings with potassium hydroxide (KOH).
Swab for microscopy, culture and sensitivities.
Serum zinc levels.
Antinuclear antibodies (ANA); extractable nuclear antigens (ENA); erythrocyte sedimentation rate (ESR).
HIV serology; Venereal Disease Research Laboratory (VDRL).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of seborrheic keratosis includes other skin lesions such as melanoma, genital warts, actinic keratosis, lentigo maligna, pigmented basal cell carcinoma, squamous cell carcinoma, psoriasis, eczema, Darier's disease, lupus erythematosus, tinea, among others(5).

TREATMENT

The management of seborrheic dermatitis (SD) focuses on eliminating the signs of the disease, relieving related symptoms such as pruritus, and achieving long-term remission. Because the main underlying pathogenic mechanisms involve Malassezia proliferation and inflammation, the most frequently employed treatments are topical antifungal and anti-inflammatory agents. Other common therapeutic options include lithium gluconate/succinate, coal tar, salicylic acid, selenium sulfide, sodium sulfacetamide, glycerin, benzoyl peroxide, aloe vera, mud treatments, phototherapy, among others. In addition, alternative therapies such as tea tree oil, Quassia amara and Solanum chrysotrichum have been proposed. Systemic treatment is reserved exclusively for cases with generalized lesions or when they do not respond to topical treatment(17).

The approach to the treatment of seborrheic dermatitis (SD) varies according to the age of the patient, as well as the distribution and severity of the disease. It is essential to promote good general skin care practices, such as the use of a soap substitute and adequate moisturization. Treatments should focus on treating the underlying pathologic process and any secondary features, particularly hyperkeratotic scale, staphylococcal infection and associated symptoms, especially pruritus.

A group of Danish experts recommended that authorities implement topical antifungals as first-line treatment, and agreed that topical corticosteroids and calcineurin inhibitors should only be used for cases with severe symptoms or to control moderate to severe flare-ups. In ASD, cradle cap removal and parental anxiety management are key factors. Sorbolene cream or lotion and a soft-bristled toothbrush can soften and remove cradle cap scales. In addition, relieving itching and discomfort in ASD is critical(18,19).

A standard formulary should include antifungal, keratolytic, antipruritic and anti-inflammatory agents (topical corticosteroids and calcineurin inhibitors). In addition, alternation of treatment may be more effective and associated with fewer side effects than continuous monotherapy. For the treatment of dermatitis on the scalp and other areas, the evidence supports the use of topical 1% to 2% ketoconazole, 1% ciclopirox, 1% zinc pyrithione, and 1% hydrocortisone. Intermittent use of a combination of mild topical corticosteroids and imidazole antifungals is desirable and can be highly effective, but a potent corticosteroid may be necessary for short-term treatment of scalp dermatitis.

Shampoos usually contain combinations of agents such as pine or coal tar (antipruritic/keratolytic), salicylic acid (keratolytic), sulfur (antimicrobial/keratolytic) and sulfacetamide (anti-inflammatory/antibacterial). The patient can apply them on the scalp and non-scalp areas and rinse them off after 5 to 10 minutes. Since there is insufficient data on safety and efficacy to support this treatment, caution should be exercised when using topical salicylic acid, selenium or zinc to treat inflammatory seborrheic dermatitis. However, topical

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ketoconazole has been shown to be safe in infants, with minimal systemic absorption detected.

Side effects associated with topical corticosteroids should be minimized by intermittent use of potencies appropriate for the area or steroid-sparing preparations, such as topical pimecrolimus 1%. Another option is to employ the inherent anti-inflammatory effect of topical antifungals, which is estimated to be comparable to 1% hydrocortisone.

Systemic treatment should be considered in cases of widespread or refractory disease, and the standard of care uses the antifungal and anti-inflammatory properties of ketoconazole (monitoring liver function; black box warning), itraconazole (checking drug interactions with CYP450; may worsen heart failure) and fluconazole (adjusting dose according to renal function). Itraconazole has the most notable anti-inflammatory effect, while oral terbinafine may be more effective than oral fluconazole in severe seborrheic dermatitis. Low-dose isotretinoin is not inferior to standard topical treatment, but is often associated with significant mucosal and skin side effects.

Itraconazole is safe and effective in controlling Parkinson's disease episodes and preventing relapses. It has also been shown to improve quality of life in patients with moderate to severe Parkinson's disease. However, given the lack of high-quality data on safety and efficacy, a review by a team of specialists is recommended before initiating systemic treatment for HIV-induced Parkinson's disease. In the case of HIV-AIDS, antiretroviral treatment usually improves Parkinson's disease, and Parkinson's disease may improve with L-dopa therapy. Future therapies for Parkinson's disease may focus on improving skin barrier function or restoring the lipid composition of the skin surface(20-23).

PROGNOSIS

ISD usually involves the scalp, is mild and self-limited, whereas ASD has a chronic pattern of skin disease characterized by relapses and remissions. ASD is manageable, although there is no cure available at the moment, timely treatment can improve the quality of life.

CONCLUSIONS

Seborrheic dermatitis (SD) is a common skin condition that affects various ages, from infants to older adults, and whose clinical presentation can vary significantly depending on skin type and other individual factors. Although the etiology of SD is not yet fully understood, factors such as alteration of the skin microbiota, especially by the yeast Malassezia spp, immune dysregulation and excessive lipid secretion are thought to play a role in its development. The most common treatment focuses on the use of topical antifungal and anti-inflammatory agents, but therapies should be individualized according to the severity and location of the lesions. While SD may be self-limiting in some cases, in others it presents a chronic pattern, with relapses and remissions, requiring constant management to improve the quality of life of patients.

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ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
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Conflict of Interest Statement

The authors report no conflicts of interest.

Funding

The authors report no funding by any organization or company.