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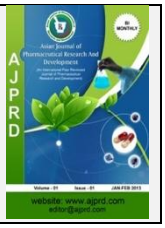

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Review Article

Review on Skin Disease Seborrheic Dermatitis Along With Its Treatment and Formulary

Rutuja Bhujbal*, Shreya Musale, Mrudula Barge, Nishigandha Naikawadi

YSPM'S, Yashoda Technical Campus, Faculty of Pharmacy, Wadhe, Satara.

A B S T R A C T

Seborrheic Dermatitis is common inflammation of the skin, occurring most often on the face, scalp and chest. It is closely related to infantile seborrheic dermatitis, or diaper rash.Seborrheic Dermatitis is particularly common in patient with Parkinson's disease or with HIV/ AIDS. Yeast of the genus Malassezia has long been regarded as a main predisposing factor, even though causal relationship has not been firmly established. Additional predisposing factors have been described, including sebaceous activity, host immunity (especially HIV infection), epidermal barrier integrity, skin microbiota, endocrine and neurologic factors, and environmental influence.

After synthesizing key evidence from the literature, we propose that intrinsic host factors, such as changes in the amount or composition of sebum and/or defective epidermal barrier, rather than Malassezia, may form the basis of SD pathobiology. The resent resurgence of interest in Malassezia yeasts has revived the old hypothesis that seborrheic dermatitis is caused by an altered relationship between these skin commensals and the host. Moreover, the success of antifungal medications in treating seborrheic dermatitis provides new evidence for this view.

Keywords: Seborrheic Dermatitis; Erythema; Greasy Scales; Cradle Cap; Diaper Rash;

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*Address for Correspondence:
Rutuja Bhujbal, YSPM'S, Yashoda Technical Campus, Faculty of Pharmacy, Wadhe, Satara.

INTRODUCTION

Seborrheic Dermatitis is chronic, recurring, cutaneous condition that causes erythema and flaking, sometimes appearing as macules or plaque with dry white or moist oily scales. (1)In adults, it commonly occurs in areas with high concentrations of sebaceous glands. The face and scalp are the most frequently affected areas, and involvement of multiple sites is common. [(2)] Dandruff is regarded as mild non inflammatory form of seborrheic dermatitis.

It usually goes away on its own by the time the child turns one year old. It starts within the first three months of life and is moderate, self-limiting, and self-resolving. [(4)]

The cause is not well understood, but appears to be related to the composition of sebaceous gland secretions, the proliferation of Malassezia yeasts, and the host immune response.[(2)]

Between 30 to 83% of immunocompromised individuals, particularly those with AIDS, have this condition on a regular basis. SD is frequently observed in association with other skin conditions, such as pityriasis versicolor, rosacea, blepharitis and/or ocular irritation, acne vulgaris, and Malassezia folliculitis. [(6)] The estimated prevalence of SD in young adults is 1-3 percent. [(19)]

Definition-

Seborrhoeic Dermatitis (SD) is chronic recurrent common skin inflammation affect sebaceous glands rich areas of skin causes scaling. [(21)]

Dermatitis, also known as eczema, is inflammation of the skin. It is characterised by itchy, erythematous, vesicular, weeping and crusting patches. The term eczema is alsocommonly used to describe atopic dermatitis, also known as atopic eczema. [(22)]

ETIOLOGY

Seborrhoeic dermatitis typically develops in locations with the largest density of these glands, excessive sebaceous gland activity and sebum overproduction may be the cause. (14)

Several factors are involved in the development of this disorder: hormone levels, individual lipid compositions, overactivity of the sebaceous glands, fungal infections, nutritional deficits, neuropsychiatric factors, and environmental conditions, in addition to host susceptibilities such as sebum production. (28) SSD begins at puberty, indicating a hormonal impact of androgens on the pilosebaceous unit. This influences the lipid composition and activity of sebum glands, as well as encouraging the establishment of Malassezia. (14) A lipophilic fungus called Malassezia spp. is a typical component of the cutaneous microbiota of humans.(19)

Malassezia Species-

A growing body of research suggests that Malassezia spp. represent a key etiologic factor in the development of SD. Malassezia species became lessprevalent with antifungaltherapy with the skin lesions going away. This is most likely the best proof yet that Malasseziaspecies are crucial to the onset of SD. Lipophilic yeasts known as Malassezia spp. are common skin dwellers.(6)The face, scalp, and trunk are lipid-rich anatomic regions that are frequently affected by Malassezia spp. SD patients also have these conditions. (27) After significant discussion in the literature regarding whether yeasts are a secondary phenomenon or of main pathogenic significance, it is now evident that the development of seborrhoeic dermatitis is caused by lipid-dependent Malassezia yeasts, particularly Malassezia restricta and globosa, which are known for their high lipase activity. People who have dandruff or seborrhoeic dermatitis typically have these two organisms on their scalps(11)

Skin sebum promotes the growth of P. ovale, or Malassezia, and hence the development of SD. Consequently, keeping reserves of leftover Sebum (from unhygienic conditions, for instance) may predispose a patient to developing the disease, as is the case with neuropathic people. The fact that SD improves when treated with antifungal medicine is proof positive that Malassezia and SD are related. (5)

Our findings, which have not been thoroughly examined before, provide credence to the theory that the hyphal form of Malassezia may be one of the pathogenic elements contributing to SD. This clinical observation opens the door to additional research on the underlying molecular processes of SD Malassezia hyphal pathogenicity. (27)

Other factors-

- Additionally, it has been noted that people with problems of the central nervous system seem to be more likely to acquire widespread seborrhoeic dermatitis, which is frequently resistant to therapy. It has been postulated that in some instances, the infection is the consequence of excessive sebum pooling brought on by immobility, which encourages yeast growth. (11)
- By initiating the initial immunological reaction against Malassezia, the innate immunity plays a crucial part. The federal government of long-term immunological inhibition SD is significantly more common among people with conditions such hepatitis C, HIV/AIDS, alcoholic pancreatitis, organ transplant recipients, and various malignancies. (12)
- Since there are no discernible variations in yeast carriage levels between healthy controls and SD patients, it has been hypothesised that a genetic susceptibility to this illness involves an inflammatory or immunological response of some kind. (6)Additionally, it has been proposed that compromised cell-mediated immunity could promote fungus survival in the skin. (1)
- Dandruff and SD are known for their greasy hair, dry scalp, and oily and dry skin, which might be signs of increased sebaceous gland activity and a compromised epidermal barrier. work(12)

SYMPTOMS

The feeling of tightness that is associated with the symptom of dry scalp comes from compromised function of the stratum corneum barrier. The most common methods for evaluating skin dryness signs are various equipment based onelectrical properties of theskin surface or trans epidermal water loss (TEWL) measurement; subjective self-evaluation ratings are usually used for tightness assessment. (16)

As it becomes worse, you'll notice an increase in skin flakes, scalp inflammation, and greasy, white or even yellow spots on your skin. You may see these skin patches on your face, nose, eyelids, chest, or armpits in addition to your scalp. (20)

Some common symptoms are (20, 21, 22 ,23, 16)

- Erythema
- Papules
- Dry, cracked skin
- Painful skin with stinging or burning
- Pruritus
- Sensation of tightness “dryness”
- Cradle cap
- Rashes that appears redder in individuals with white skin and darker or lighter in those with brown or black skin
- Oily skin patches on the scalp, face, sides of nose, eyebrows, ears, eyelids, chest, armpits, or beneath the breasts that are coated in flaky white or yellow scales or crust.

RISK FACTORS

Table: 1

Risk factor	Description
Immunodeficiency	Lymphoma Renal Transplantation HIV/ AIDS
Neurological and Psychiatric disorder	Parkinson disease Stroke Down syndrome Alzheimer disease
Life style factors	Poor nutrition Unhygienic practises
Exposure to drug treatment	Lithium Dopamine antagonists Psoralen Immunosuppressants
Lipids and hormones	Distribution of lesion on the body corresponds to distribution of sebaceous glands, with excess sebum found on the scalp, chest, eyebrows, ears

PATHOPHYSIOLOGY

Although the pathophysiology of SD's sequence of events is uncertain, the majority of publications concur that the three primary prerequisites are as follows: colonisation of Malassezia, lipid secretion by sebaceous glands and a predisposition of the immune system [8]

Mechanism for the pathogenesis of seborrheic dermatitis include [2] [8]

- Alterations in the microbiota of the skin.
- Disruption of the skin barrier leading to clinically noticeable erythema, pruritus, and scaling
- An impaired immune response to Malassezia spp. related to a decreased T-cell response and complement activation.
- cutaneous neurotransmitter disruption.
- Disturbances of the epidermal barrier associated with genetic factors.
- Malassezia spp. are also responsible for the breakdown of sebum and ingesting saturated fatty acids, that throw away the skin's lipid a state of equilibrium The division of Malassezia spp. to SD lesions and the significant improvement in SD adhering to antifungal medication provide more evidence of their involvement.
- Abnormal keratinocyte shedding

TREATMENT

The strategy will change according on the patient's age, the condition's distribution, and its severity. It is imperative to talk about general skincare best practices, such as using a soap alternative and the right amount of moisturising [2]

While there isn't a permanent cure for SD, there are a number of effective treatment methods that can manage the illness. The goal of therapy is to manage acute flare-ups.focuses on sustaining remission with prolonged treatment. [7]

Imidazole was the most often utilised topical medication, with corticosteroids coming in second at 59.9%.topical calcineurin inhibitors (27.2%),

hydrating/emollient/nutritive therapies (30.7%), and antimycotics (35.1%). (13)Intermittent use of site-appropriate potencies or steroid-sparing formulations, such as topical 1% pimecrolimus, can help reduce the side effects of topical corticosteroids. [2]

Topical treatment -

Topical corticosteroids combined with nutritional, hydration, and emollient therapies (7.5%). The most common concurrent medications given for non- cutaneous disorders were anxiolytics (2.0%) and antidepressants (2.1%) (13)

1. SD of the Scalp in Adults-
2. It is highly advised that adults utilise topical medications with antifungal (ketoconazole, ciclopirox, miconazole), anti-inflammatory (betamethasone valerate, clobetasol propionate), or keratolytic/humectant (propylene glycol) qualities. [9]

3. SD of the Scalp in Children-
4. Topical antifungals (1% ciclopirox shampoo, 1% KTZ cream/shampoo, 2.5% SS shampoo), anti-inflammatory (1% hydrocortisone cream/lotion), or keratolytic agents (3% salicylic acid combined with 1.5% ciclopirox shampoo, lactamide): efficacy and safety shampoo with monoethanolamide. [2]
5. Nonscalp SD-
6. For mild-to-moderate SD on the face and/or body, the use of topical antifungal (KTZ, ciclopirox, clotrimazole) and anti-inflammatory (desonide, hydrocortisone, lithium succinate/gluconate, topical pimecrolimus/tacrolimus) medicines is strongly advised. [9]

Oral Agents-

Before starting oral treatment for ISD, a professional team review is advised due to the lack of high-quality safety and effectiveness data. Anti retroviral therapy often improves SD in HIV-AIDS patients, while L-dopa medication may help improve SD in Parkinson's disease patients.

Prospective treatments for SD may concentrate on enhancing skin barrier performance or replenishing the lipid composition of the skin's surface. [2, 10].

FORMULARY [25, 26]

Table: 2

Drug (Reference)	Presentation	Application frequency
Corticosteroids		
Hydrocortisone	1% to 2.5% cream	Twice daily
Clotrimazole+Hydrocortisone	1% cream	Twice daily
Immunomodulator		
Tacrolimus	0.1% ointment	Twice daily
Pimecrolimus	1% cream	Twice daily
Terbinafine	1% cream 250 or 500 mg tablet	Once daily Twice daily
Antifungals		
Salicylic Acid	2% shampoo With addition of 5% coal tar shampoo With addition of 2% sulphur ointment	Once per week Once- twice per week Twice daily
Ketoconazole	2% gel 2% cream Foam Shampoo	Once daily Twice daily Twice daily Once- twice per week
Zinc pyrithione	1% shampoo	Once daily for one week, then three times per day
Ciclopirox	1% shampoo 0.77% gel	Twice in week Once daily
Selenium sulphide	1% to 5% shampoo	Twice in week

CONCLUSION

SD is a common skin condition seen frequently in clinical practice. Despite its frequency, much controversy remains regarding its pathogenesis. This controversy extends to its classification in the spectrum of cutaneous diseases, having been classified as a form of dermatitis, or a fungal disease, or a disease closely related with psoriasis. As a result treatments vary, ranging from topical corticosteroids to topical antifungals and AMPs

SD subjects should be warned about the various precipitating conditions described above, including intake of some drugs, nutritional deficiency, and concurrent

immunosuppression and/or comorbidities. Finally, the role of environmental factors (cold, low humidity, excessive sun exposure), physical/psychological stress, unhealthy lifestyle (alcohol consumption), wrong or inadequate cosmetic use, that may contribute to worsen SD, should also be considered

Some studies also reported a protective role of Cuti bacterium in maintaining healthy skin barrier function through elevating the water content of the stratum corneum. Further studies focusing on the interaction between different microbes and the host and microbes can provide better insight into the role of microbial alterations in the pathogenesis of SD/DF.

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