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Review Article

Review Article on Seborrheic Dermatitis

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ABSTRACT

Seborrheic dermatitis is a common, long-lasting inflammatory skin condition that mostly affects parts of the body with a lot of sebaceous (oil-producing) glands, such the scalp, face, chest, and upper back. It causes the skin to become red, itchy, and covered in greasy, yellowish scales or flakes. Seborrheic dermatitis's specific etiology isn't known, although a number of things are thought to have a role in its development. These include too much sebum production, having yeast from the genus Malassezia on the skin, being genetically prone to it, hormonal fluctuations, and environmental variables including stress and changes in the weather. Seborrheic dermatitis is most frequent in two age groups: babies and adults. It is sometimes called cradle cap in babies. It looks like thick, oily scales on the scalp, although it normally goes away on its own. In adults, the illness may last for a long time and typically comes and goes in cycles of flare-ups and remission. Some things, such a poor immune system, neurological diseases, and stress, might make symptoms worse. A clinical evaluation of the afflicted skin regions is generally used to make a diagnosis. The primary goal of treatment is to reduce symptoms and lower inflammation and fungus development. Some common treatments include medicated shampoos with antifungal ingredients, topical corticosteroids, and lotions that reduce inflammation. Seborrheic dermatitis is neither infectious or life-threatening, but it may make life much harder since it is uncomfortable and can make people look bad. In general, good skin care, stress management, and the right medical care may help keep the symptoms under control and stop them from coming back often. Researchers are still working to learn more about the underlying causes of this widespread skin ailment and how to properly treat it.

INTRODUCTION

It is a common ailment that is chronic and relapsing, and it is known as seborrheic dermatitis. This skin condition is characterized by inflammation. The scalp, the face, and the upper trunk as well as other areas of the body that have a high concentration of sebaceous glands are often affected by this condition (1). The clinical signs of this illness include erythematous plaques that are

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accompanied by scales that are oily and yellowish in color. Additionally, it is usually linked to pruritus and cosmetic discomfort because of its inflammatory properties. It is a sickness that affects individuals of all ages, with the maximum frequency happening throughout childhood and maturity, particularly between the third and sixth decades of life. The illness is a disorder that affects people of their whole lives. It is theorized that seborrheic dermatitis is brought on by a complicated interaction between the colonization of Malassezia yeast, the activity of sebaceous glands, the breakdown of the epidermal barrier, and the immune responses of the host (2). On the other hand, the particular etiology of seborrheic dermatitis is not yet established with complete certainty. It has been shown that people who suffer from neurological disorders, immunosuppression, and infections caused by the human immunodeficiency virus have a greater incidence and severity of the condition. This finding underscores the role that immunological dysregulation plays in the genesis of disease (3). In contrast to the clinical approach, which is the primary way of diagnosis, the treatment of the illness focuses on antifungal medications, anti-inflammatory treatments, and long-term maintenance approaches to reduce the frequency of flare-ups . It is important to establish treatment techniques that are effective, safe, and well tolerated . Although seborrheic dermatitis is a benign condition, it may have a significant impact on quality of life. This underscores the need of creating treatment methods that are of this kind (4,5).

History

When it comes to dermatology, seborrheic dermatitis has a rich history that is still in the process of evolving. In the earliest medical texts, for instance, there may be references to scaly and inflammatory diseases that affect the face and scalp. Conversely, seborrheic dermatitis was not recognized as a distinct clinical entity until the later part of the nineteenth century (6). This was the case as well. It was in the year 1887 when Paul Gerson Unna was the first person to put out the idea of seborrheic eczema. A important contributor to the course of the sickness, according to his hypothesis was the abnormal activity of the sebaceous glands (7). This hypothesis had an impact on the development of early categorization systems, and it was also responsible for establishing a relationship between the condition and eczema and seborrhea. However, further studies indicated that increased sebum production alone was not adequate to completely explain the development of the health issue. This was shown beyond a reasonable doubt (8).

Raymond Sabouraud made a significant contribution to the expansion of knowledge at the beginning of the twentieth century by determining that affected skin was contaminated with lipophilic yeasts. These yeasts were initially referred to as Pityrosporum, but they were later reclassified as Malassezia species (9). This discovery was a significant step forward in the field of dermatology. As a consequence of Sabouraud's findings, the focus turned toward a microbial etiology, which helped to the creation of antifungal drugs. In the middle of the twentieth century, advancements in dermatopathology made it feasible to identify seborrheic dermatitis from psoriasis, contact dermatitis, and other papulosquamous illnesses in a more exact way . This was made possible by the fact that seborrheic dermatitis developed during this time period. The results of histological studies showed a number of features, such as parakeratosis, spongiosis, and inflammatory infiltrates. These findings add credibility to the idea that the illness ought to be

categorized as an inflammatory dermatosis rather than a merely sebaceous ailment (10,11).

Beginning in the second part of the twentieth century and going ahead, the involvement of host variables became an increasingly major focus of study. This trend is expected to continue. The clinical observations revealed that patients with neurological conditions, in particular Parkinson's disease, as well as immunocompromised individuals, including those with human immunodeficiency virus infection, had a higher prevalence of seborrheic dermatitis and a more severe form of the condition (12). This was the case for both patients and immunocompromised individuals. These discoveries provide insight on the relevance of immune control as well as the interactions that occur between the neurocutaneous system. Research in the current period continues to research molecular causes, dysfunctions of the epidermal barrier, genetic vulnerability, and immunological responses, which finally leads to the creation of targeted and combination therapy techniques (13).

Epidemiology

Seborrheic dermatitis is a common skin disease that affects individuals of all races and socioeconomic levels across the world. The rate of occurrence in the general adult population is thought to be between 1 and 5 percent, however this number may change depending on where the study was done and how it was done. The age distribution of the condition shows that there is an initial peak in infancy, which is usually called infantile seborrheic dermatitis or cradle cap, and a second, more persistent surge in maturity, especially between the ages of 30 and 60. The sickness affects people of two different ages a lot (14).

It has been shown over and over again that there are more men than women with the illness. Androgens are thought to be responsible for this discrepancy because they stimulate sebaceous glands, which leads to more sebum production, which in turn helps Malassezia multiply. People with weak immune systems are far more likely to have seborrheic dermatitis and have worse cases of it. There is a link between the severity of the sickness and a drop in CD4 cell counts. The frequencies of human immunodeficiency virus infection may range from 30% to 80%. People with neurological diseases including Parkinson's disease, epilepsy, and stroke are also more likely to develop the disorder. This shows that there is a link between brain dysfunction and changes in skin physiology (15).

Environmental and lifestyle factors may also affect how a disease shows up. Stress, tiredness, and certain medicines, on the other hand, may serve as triggers or make things worse. Exacerbations are more likely to happen in the winter and in places with little humidity. There have been a number of elements found, such as genetic vulnerability and familial clustering, that support the idea of a complex etiology. Seborrheic dermatitis is a long-term ailment that doesn't kill you, but since it's not deadly, it's frequently misunderstood or not taken seriously. This shows how important it is to raise awareness and come up with good ways to do long-term rehabilitation (16).

Causes and how they work .Seborrheic dermatitis is a common chronic inflammatory skin illness that affects the scalp, face, chest, and upper back the most. The exact reason is a complicated process that involves interactions between the activity of sebaceous glands, the colonization of microbes, immune responses, and both hereditary and environmental factors (17).

Etiopathogenesis

1. An increase in the activity of the sebaceous glands :

Sebaceous glands produce sebum, an oily substance that keeps skin and hair soft and smooth. Seborrheic dermatitis makes the skin produce more sebum, which makes it easier for lipophilic bacteria to grow. Because of this, the disease usually shows up in areas with a lot of sebaceous glands, such the scalp, the nasolabial folds, the eyebrows, and the chest (18). Sebum doesn't directly cause inflammation, but it does provide bacteria the resources they need to start the inflammatory response (19).

2. Role of Malassezia Yeast

Malassezia yeasts are very crucial for the overgrowth of lipophilic yeasts from the genus Malassezia, especially Malassezia globosa and Malassezia restricta, is a major cause of seborrheic dermatitis. In seborrheic areas, these fungi may proliferate excessively, even though they are often part of the skin's microbiota (20). Malassezia species may break down the triglycerides in sebum using lipase enzymes. This produces free fatty acids like oleic acid. These fatty acids may get through the stratum corneum and cause irritation, damage the epidermal barrier, and start inflammatory processes. This causes redness, scaling, and itching to break out (21).

3. Immune and inflammatory Response

Responses of the immune and inflammatory systems the host's immune system has a big role in how the disease becomes worse. People with seborrheic dermatitis show an unusual immunological response to Malassezia metabolites. Keratinocytes and immune cells are responsible for making inflammatory mediators such cytokines and interleukins. These mediators cause the usual redness and flaky scales, which in turn cause the formation of the epidermis (22). Another piece of evidence that supports the idea that immunological dysfunction plays a role is that people with weaker immune systems, including those with HIV/AIDS, are more likely to have seborrheic dermatitis (23).

4. Epidermal barrier dysfunction

Problems with the structure of the epidermal barrier the skin barrier must be intact to prevent bacteria from getting in and causing irritation. Seborrheic dermatitis causes the stratum corneum to become disturbed because of the breakdown of lipids and the release of inflammatory mediators (24). This not only makes the skin more likely to become irritated and inflamed, but it also makes the transepidermal layer lose more water (25).

5. Genetic and hormonal factors

things that have to do with genetics and hormones some research suggests that having a genetic predisposition may make it more likely that you may have seborrheic dermatitis. People who have had the sickness in their family are more likely to have it again. Androgens control the activity of the sebaceous glands, which indicates that hormones also play a role. Seborrheic dermatitis usually shows up when the sebaceous glands are working the most, such when a person is a youngster (cradle cap) or an adult. That's why it's so frequent during these times (26).

6. Neurological and Systemic Factors

Things that have to do with the nervous system and the system seborrheic dermatitis is more likely to happen to those who have neurological disorders like Parkinson's disease. For other individuals, the disease may be caused by a mix of things, such as less movement in the face muscles, too much

sebum production, and immune system problems (27).

7. Environmental and Lifestyle Factors

Things that are caused by the environment and way of life there are a variety of outside factors that might make seborrheic dermatitis worse or even cause it to happen. These include:

- Dry and cool temperature
- Anxiety for mental health reasons Worry and tiredness
- The usage of oil-based beauty products these factors might make the skin produce more sebum or modify the microbiome of the skin, which could cause discomfort (27-29).

Role of sebaceous glands and sebum

Sebaceous glands and sebum are very significant. Seborrheic dermatitis generally affects areas of the body with a lot of sebaceous glands, which shows how important sebum is in the evolution of sickness (30). Sebum creates an environment rich in lipids that helps lipophilic Malassezia species grow. Seborrheic dermatitis is not the only disease that causes too much sebum to be produced, however sebaceous activity may be common in many people who have it. Some people think that changes in the amount of sebum, specifically changes in the profiles of free fatty acids, are what cause the skin to become irritated and inflamed (31).

Role of Malassezia Species

Different kinds of Malassezia and what they do several species of Malassezia, especially M. globosa and M. restricta, are very important in the development of seborrheic dermatitis. These yeasts may break down sebum triglycerides by lipase activity, which frees up liberate fatty acids. People who are susceptible to these free fatty acids may experience irritation and inflammation because they may get through the stratum corneum (32). Even while Malassezia organisms are a natural part of the skin flora, people with seborrheic dermatitis have an unusual inflammatory response to the metabolic byproducts of these organisms instead of just overcolonizing the disease. The fact that antifungal drugs work well in clinical settings is another proof that Malassezia is a pathogen (33).

Immune And inflammatory Mechanism

A shift in the host's immune response is a big part of what makes them sick. People with seborrheic dermatitis have higher amounts of inflammatory cytokines, such interleukin-6, interleukin-8, and tumor necrosis factor-α, in the skin that is affected by the disease (34). The fact that seborrheic dermatitis is so common and severe among people with weakened immune systems, including those who have HIV or have had an organ transplant, shows that their cell-mediated immunity is not working properly. The growing number of neurological diseases is another sign that neuro-immunological pathways may be involved in their development (35).

Epidermal Barrier Dysfunction

Seborrheic dermatitis is caused in large part by damage to the epidermal barrier. Affected skin has shown increased transepidermal water loss and altered corneocyte cohesiveness, which makes it easier for irritants and microbial antigens to get through (36). This barrier problem makes skin irritation worse and keeps the illness from going away (37).

Environmental, and Hormonal Factors

There have been reports of family clustering, which suggests that genetic predisposition may play a role in susceptibility (38). Stress, weariness, and a chilly temperature with low humidity are all environmental variables that are known to make the illness worse (39). Hormones, especially androgens, make sebaceous glands work harder. This would explain why adult seborrheic dermatitis is more common in men and starts after puberty (40).

Integrated Pathogenic Model

An integrated model that shows how Malassezia metabolites interact with sebum and a weakened epidermal barrier is the best way to describe seborrheic dermatitis right now. This model shows how these interactions cause an increased inflammatory response in those who are genetically and immunologically susceptible (41). This interplay of several factors explains why the illness lasts a long time, has different levels of severity, and comes back often.

Histopathology

The histological signs of seborrheic dermatitis change depending on how bad the illness is and what stage it is at. No one result is pathognomonic on its own, but a group of alterations in the epidermis and dermis supports the diagnosis when compared to clinical characteristics (42).

Changes in the epidermis

Seborrheic dermatitis lesions in their early stages display modest spongiosis and localized parakeratosis, usually surrounding follicular ostia (43). The stratum corneum usually shows parakeratotic scaling with orthokeratosis in between, which gives the greasy scales that are seen in people (44). As the condition becomes worse, psoriasiform epidermal hyperplasia may show up. However, it is generally less noticeable and more uneven than what is observed in psoriasis (45). There may be neutrophils in the parakeratotic stratum corneum, but Munro microabscesses, which are a sign of psoriasis, are usually not there or are very small.

Follicular involvement

Follicular lipping is a unique histological hallmark of seborrheic dermatitis. It is marked by parakeratosis around the entrances of the follicles (46). This study shows that the illness likes to attack places with a lot of sebaceous glands. It also helps tell seborrheic dermatitis apart from other types of eczema (47).

Dermal changes

There is a superficial inflammatory infiltration in the dermis around the blood vessels and hair follicles. Lymphocytes make up most of this infiltration, while histiocytes and neutrophils show up from time to time (48). There may be mild papillary dermal edema and vascular dilatation, both of which make the lesions seem red. The little skin fibrosis seen in chronic lesions indicates that the condition does not result in scarring (49).

Specific Changes in Color and Microbes

You may employ special stains like periodic acid–Schiff to establish that Malassezia species are present in the stratum corneum. However, these organisms are not specific since they are part of the normal skin flora (50). In fact, their increased density in injured skin shows that they are involved in the disease's pathogenesis rather than being a clear diagnostic criterion.

Differential Histopathology Analysis

Histological investigation must differentiate seborrheic dermatitis from psoriasis, contact

dermatitis, and tinea infections. Seborrheic dermatitis has decreased neutrophil numbers in the stratum corneum, elevated spongiosis, and reduced regular acanthosis when contrasted with psoriasis (51).

Genetics of immunity

There are several things that affect how an individual's immune system reacts to Malassezia species, such as their genetic susceptibility, their innate and adaptive immune responses, and the immunogenetics of seborrheic dermatitis. These things all work together in a complicated way. Even though Malassezia yeasts are a normal part of the skin's microbiota, seborrheic dermatitis is uncommon in those who are genetically prone to it. This suggests that they play a big role in controlling the immune system of the host (52,53).

Innate Immune Response

The innate immune system is the first to notice Malassezia, and it is a very important aspect of the process. Keratinocytes and antigen-presenting cells are the cells that have pattern recognition receptors on their surfaces. Toll-like receptors (TLR2 and TLR4) are some of these receptors. They help the body recognize parts of fungal cell walls (54). In seborrheic dermatitis, these receptors are turned on more, which produces more pro-inflammatory cytokines such interleukin-1β, interleukin-6, interleukin-8, and tumor necrosis factor-α. This causes the epidermis to become inflamed and immune cells to come to the area (55).

Adaptive Immune Response

Changes in adaptive immunity are another thing that might cause sickness. Researchers have shown that the skin that has been hurt has higher amounts of cytokines, such as interferon-γ and interleukin-17 (56). This means that T helper pathways, including Th1 and Th17, are more active. It is also plausible that regulatory T-cell failure might make the immune system less tolerant to Malassezia, which could lead to chronic inflammation (57). The fact that those who are well have the same amount of fungus yet become ill again and again may be due to a stronger inflammatory response rather than the fungus spreading (58).

Genetic susceptibility

Genetics may affect how the immune system responds to seborrheic dermatitis. Some people think that polymorphisms in genes that affect innate immunity, the skin barrier, and inflammatory signaling pathways are linked to the illness (59). Changes to the genes that code for pattern recognition receptors, antimicrobial peptides, and cytokines may modify how microorganisms interact with their hosts and how much inflammation they can handle (60). Seborrheic dermatitis tends to run in families, which suggests that there is a genetic component, even if specific susceptibility loci have not been fully identified (61).

Weak immune system and weak immunological control

The strong link between seborrheic dermatitis and illnesses that weaken the immune system shows how important it is to manage the immune system in the development of sickness. People who have the human immunodeficiency virus (HIV) frequently get the illness more often and more severely, which is linked to a drop in the number of CD4+ T-cells (62,63). People with neurological diseases also have problems with their immune system, which might be because of neuroimmune interactions that affect how sebaceous glands work and how the skin's immune system works (64).

Clinical and Diagnosis

Seborrheic dermatitis is usually diagnosed by a clinical examination, which looks for the disease's unique features and how it spreads, rather than by lab testing. The illness commonly shows up as red patches covered with greasy, yellowish scales in places with a lot of sebaceous glands, such the scalp, face (particularly the nasolabial folds, eyebrows, and behind the ears), chest, and intertriginous regions. Patients typically say they have itching, burning, or cosmetic pain, even though the symptoms may not always be the same. The condition is chronic and comes and goes. Flare-ups are common and are generally caused by stress, weariness, or changes in the seasons, especially in the winter (65). The main things that help make the diagnosis are the shape and spread of the lesions and the complete removal of any skin conditions that look like them. Seborrheic dermatitis and psoriasis have similar symptoms, but psoriasis has thicker, silver scales and well-defined plaques that are often seen on the nails, elbows, and knees. Most of the time, atopic dermatitis is characterized by more itching, involvement of the flexures, and a long history of atopy. People may confuse tinea infections with seborrheic dermatitis, although tinea infections usually show up with ring-shaped lesions with center clearing (66). A bacteria culture or a potassium hydroxide test can confirm this. Seborrheic dermatitis in kids looks like "cradle cap," which is when the scalp scales are dense and stick to each other. In adults, however, the disorder is more likely to cause persistent problems with the face and scalp.

There is no specific test in the lab that can confirm seborrheic dermatitis. A skin biopsy might sometimes show nonspecific indications, such spongiotic dermatitis with parakeratosis, even though it is usually never needed. The treatment response supports the diagnosis even more. The fact that antifungal or anti-inflammatory medicines worked shows that seborrheic dermatitis is likely the cause of the problem. It is important to have a greater degree of clinical suspicion since people with weakened immune systems, particularly those with HIV, may have more severe and widespread symptoms of the disease (67).

In short, a doctor can tell whether someone has seborrheic dermatitis by doing a physical exam, ruling out other possible diagnoses, and keeping an eye on how well the treatment works. It is important for doctors to know how to correctly diagnose this ailment since it depends on a combination of the shape, distribution, chronicity, and typical relapsing history of the lesions (68).

Clinical feature

1. General Feature

It is a long-term skin disease that comes and goes and usually affects areas of the body with a lot of sebaceous glands. Seborrheic dermatitis is a skin disease that makes the skin inflamed. The most common signs of this illness are redness, greasy or yellowish scales, itching, and mild irritation. The severity of the ailment may vary from a little patch of dandruff on the scalp to more extensive inflammatory lesions that affect numerous parts of the body. Most of the time, the sickness will become worse and then better in ways that are hard to foresee (69).

2. Distribution of Lesions

Seborrheic dermatitis lesions usually show up in parts of the body that have a lot of sebaceous glands. This skin ailment commonly affects the scalp, hairline, eyebrows, eyelids, nasolabial folds, ears (the outer auditory canal), beard region, chest,

and upper back. There are a lot of lipids in these places, which makes it easier for lipophilic yeasts like Malassezia to grow. These yeasts are considered to have a role in the disease's pathogenesis (70).

3. Scalp involvement

The scalp is the region that is most affected first and foremost. Patients frequently have scaling that looks like dandruff. The scaling might be anything from tiny white flakes to yellow scales that are bigger and oilier (71). When the illness is moderate to severe, you might see red spots that are covered with scales. The problem may also go beyond the hairline and onto the forehead or the region behind the ears. People frequently talk about how their scalp keeps itching (72).

4. Facial Expressions

A lot of patients with seborrheic dermatitis also have problems with their faces. Lesions are often seen on the eyebrows, nasolabial folds, glabella, eyelids, and the area surrounding the beard. These areas could have crimson patches, greasy scales, and mild irritation. When the eyelids are affected by seborrheic blepharitis, it might lead to a disease. The margins of the eyelids are red and scaly when you have this disease (73,74).

5. Lesions on the Vernal and Intertriginous Tracts

Seborrheic dermatitis may extend to other places of the body in some persons. These include the upper back, the armpits, and the groin. It also has an effect on the middle of the chest. Lesions in these areas show up as well-defined erythematous plaques with greasy scales on top. The plaques may seem like rings or patches, and they may also cause some discomfort or itching (75).

6. Seborrheic dermatitis in babies

Seborrheic dermatitis, which is more well known as cradle cap, may also show up in newborns. It usually shows up in the first few weeks of a person's life and is marked by thick yellow or brown scales on the scalp. The area surrounding the diaper, the wrinkles of the neck, and the face may also be affected by the condition. Infantile seborrheic dermatitis usually goes away on its own in a few months, and there are usually no significant problems that come with it (76).

7. Patient complaints and their symptoms

Patients most commonly say they have itching (also called pruritus), a burning sensation, and worries about how their skin looks because of visible scaling. The level of itching might be different for each individual, although it is usually mild to severe. In worst cases, scratching might lead to greater pain or an infection later on (77).

8. Associated Conditions

People with certain systemic diseases are more likely to be diagnosed with seborrheic dermatitis. For example, those who have HIV/AIDS have a significantly higher frequency, and the disease may show up in a more severe or widespread way. It is also commonly connected to neurological diseases like Parkinson's disease, which may be caused by more sebum production and less movement of the face (78).

9. Course and Recurrence

Most of the time, seborrheic dermatitis has a long-lasting and recurrent clinical history. When you are stressed, tired, or in cold weather, your symptoms may become worse. Changes in hormone levels might also make them worse. The medicine may help with the symptoms, but a full cure is quite unusual, and it's typical for people to experience relapses from time to time (79).

Seborrheic Dermatitis and HIV

Seborrheic dermatitis is one of the most common skin problems that people with HIV have. It often acts as a first clinical sign of immunological dysfunction (80). Seborrheic dermatitis is a skin disorder. People with HIV are more likely to have seborrheic dermatitis, and it is usually worse, more extensive, and harder to cure (81).

Epidemiology and Clinical Significance

The prevalence of seborrheic dermatitis in HIV-infected patients ranges from thirty percent to eighty percent, significantly above the one to five percent seen in the normal adult population (82,83). The sickness may occur at any stage of HIV infection; however, it is more common when CD4+ T-cell levels drop, and the severity of the disease frequently has a detrimental effect on immunological function (84). Some individuals may become worse with seborrheic dermatitis, which might mean that the disease is becoming worse or that the treatment isn't working (85).

Clinical Features of HIV

Individuals infected with HIV often have seborrheic dermatitis characterized by extensive erythema, thick greasy scales, and pronounced inflammation. This problem affects not only conventional sebaceous areas but also unusual sites, including the extremities and intertriginous regions (86). Facial involvement is often evident, characterized by notable erythema of the nasolabial folds, eyebrows, and forehead. Pruritus may be rather intense, increasing the likelihood of future infections (87).

Pathogenesis of HIV:

A dysregulated immune system, rather than an increase in fungal colonization, is the fundamental cause of the increased prevalence and severity of seborrheic dermatitis in HIV patients (88). A diminished cell-mediated immune response leads to an intensified inflammatory reaction to Malassezia antigens. Another thing that makes inflammation last longer is changes in cytokine profiles, such as greater levels of pro-inflammatory cytokines and lower levels of regulatory immune responses (89). Changes in the skin barrier function and sebaceous gland activity associated with HIV may exacerbate the presentation of disease (90).

Impact of Antiretroviral Therapy

For people with HIV, the introduction of highly active antiretroviral therapy (HAART) has led to a significant decrease in the occurrence and severity of seborrheic dermatitis (91). Even while immune reconstitution following antiretroviral therapy usually leads to clinical improvement, it may sometimes lead to a paradoxical worsening of the condition as part of immune reconstitution inflammatory syndrome (IRIS) (92).

Management Considerations

The concepts of care are similar to those used to immunocompetent individuals; however, the treatment often requires greater intensity and duration. Topical antifungal medicines remain the primary treatment, often used with low-potency topical corticosteroids or calcineurin inhibitors (93). Due to the frequent relapses, maintenance therapy is often necessary. To manage the illness over the long term, it is very important to make the most of antiretroviral therapy (94).

Treatment for seborrheic dermatitis

The aim of the treatment for seborrheic dermatitis is to control the symptoms, lower inflammation, lower the number of Malassezia colonies, and stop relapses. Seborrheic dermatitis is a long-term

disorder that might come back, therefore therapy usually lasts a long time and is tailored to each person based on how bad the disease is, where it is, the patient's age, and their immune system (95,96).

1. General Steps and Steps

You may get rid of scales and reduce the quantity of sebum that builds up by washing your skin and scalp regularly using mild soaps or shampoos (97). Avoiding things that might make the condition worse, such stress, cold weather, and harsh chemicals or cosmetics (98), is very important.

• Moisturizers: Non-comedogenic emollients may help reduce irritation and improve barrier function, especially when the air is dry (99)

Stress, cold weather, and harsh chemicals or cosmetics are all things that might make the condition worse, so it's crucial to stay away from them (100).

• Emollients that don't clog pores may help reduce irritation and improve barrier function, especially in dry places (101). This is particularly true with lotions.

Topical therapy

a) Antifungal Agents

These are first-line therapy, targeting *Malassezia* species (102)

- Ketoconazole 1–2% cream or shampoo: Applied twice weekly; effective in reducing fungal load and inflammation (103).
- Ciclopirox 1–2% cream or shampoo: Alternative antifungal with broad activity against *Malassezia* (104).
- Selenium sulfide 1–2.5% shampoo: Reduces fungal colonization and excessive scaling, particularly on the scalp (105).
- Zinc pyrithione 1–2% shampoo: Useful for mild to moderate scalp involvement; decreases microbial proliferation and inflammation (106).

b) Anti-inflammatory Agents

Used for acute flares or severe lesions.

- **Low-** to mid-potency topical corticosteroids (e.g., hydrocortisone 1%, desonide 0.05%): Applied for short courses (1–2 weeks) to reduce erythema and pruritus (107)
- Topical calcineurin inhibitors (e.g., tacrolimus 0.03–0.1%, pimecrolimus 1%): Safe for long-term use, particularly on the face and intertriginous areas; avoid steroid-induced atrophy (108).

3. Systemic Therapy

Reserved for severe, refractory, or widespread disease, or in immunocompromised patients (109).

- Oral antifungals:
  - Itraconazole 100–200 mg/day for 1–2 weeks
  - Fluconazole 150 mg once weekly for 2–4 weeks.

These reduce *Malassezia* overgrowth and are particularly effective in scalp, face, and trunk involvement (110).

- Systemic corticosteroids**:** Rarely used due to side effects; considered only for acute severe flares (111).

4. Special Considerations

- Infants (Cradle Cap): Gentle scalp cleansing and mild emollients; topical antifungals may be used if persistent (112)

- HIV-infected patients: Often require higher-potency or longer-duration antifungal therapy, sometimes combined with topical corticosteroids. Optimization of antiretroviral therapy improves treatment response (113).

- Chronic or relapsing cases: Maintenance therapy with antifungal shampoos 1–2 times per week is recommended to prevent recurrence (114).

5. Emerging and Adjunctive Therapies

- Phototherapy (UVB**):** Can improve resistant cases but limited by practicality and risk of skin damage (115).

- Novel antifungal agents and anti-inflammatory compounds**:** Ongoing research on topical agents with dual antifungal and anti-inflammatory action (116).

Topical Medications for Seborrheic Dermatitis

Topical therapy is the mainstay of treatment for seborrheic dermatitis and is often sufficient for mild to moderate disease. The goals of topical therapy are to reduce inflammation, control *Malassezia* overgrowth**,** relieve pruritus, and prevent relapses (117).

Topical medications can be divided into antifungal agents and anti-inflammatory agents, with combination therapy used in more severe cases

1. Antifungal Agents

Class / Example drugs	Route (main)	Mechanism of action	Main clinical uses	Important adverse effects
Polyenes (Amphotericin B, Nystatin)	Amphotericin B: IV; Nystatin: topical, oral (not absorbed) pharmascholars +1	Bind ergosterol in fungal cell membrane, form pores → leakage and cell death. pharmascholars+2	Severe systemic mycoses (Amphotericin B), mucocutaneous candidiasis (Nystatin). pharmascholars+1	Nephrotoxicity, infusion reactions (fever, chills), electrolyte disturbances (hypokalemia, hypomagnesemia).
Azoles – Imidazoles (Clotrimazole, Miconazole, Ketoconazole)	Mostly topical; ketoconazole oral in selected cases. Sciencedirect +2	Inhibit fungal lanosterol 14-α-demethylase (CYP450) → impaired ergosterol synthesis, defective membrane. jptcp+2	Superficial infections: dermatophytosis, vulvovaginal and oropharyngeal candidiasis. sciencedirect+1	Local irritation (topical), hepatotoxicity and endocrine effects with systemic ketoconazole, drug interactions via CYP inhibition. ncbi.nlm.nih+1
Azoles – Triazoles (Fluconazole, Itraconazole, Voriconazole, Posaconazole, Isavuconazole)	Oral and IV (drug-dependent). ncbi.nlm.nih +2	Same as imidazoles: inhibit lanosterol 14-α-demethylase → ↓ergosterol. jptcp+2	Systemic and mucosal candidiasis, cryptococcosis (fluconazole), aspergillosis (voriconazole, isavuconazole), endemic mycoses	Hepatotoxicity, QT prolongation (some), visual changes and photosensitivity (voriconazole), significant CYP-mediated

			(itraconazole). ncbi.nlm.nih+2	drug interactions. ncbi.nlm.nih+1
Azoles – Triazoles (Fluconazole, Itraconazole, Voriconazole, Posaconazole, Isavuconazole)	Oral and IV (drug-dependent). ncbi.nlm.nih +2	Same as imidazoles: inhibit lanosterol 14-α-demethylase → ↓ergosterol. jptcp+2	Systemic and mucosal candidiasis, cryptococcosis (fluconazole), aspergillosis (voriconazole, isavuconazole), endemic mycoses (itraconazole). ncbi.nlm.nih+2	Hepatotoxicity, QT prolongation (some), visual changes and photosensitivity (voriconazole), significant CYP-mediated drug interactions. ncbi.nlm.nih+1
Echinocandins (Caspofungin, Micafungin, Anidulafungin)	IV only. ncbi.nlm.nih +1	Inhibit β-(1,3)-D-glucan synthase → impaired cell wall synthesis, osmotic lysis. ncbi.nlm.nih+2	Invasive candidiasis, salvage or combination therapy for invasive aspergillosis. ncbi.nlm.nih+1	Generally well tolerated; histamine-related infusion reactions, mild hepatotoxicity.
Allylamines (Terbinafine, Naftifine, Butenafine)	Terbinafine: oral and topical; others: topical. Sciencedirect + 2	Inhibit squalene epoxidase → ↓ergosterol, toxic squalene accumulation. pubmed.ncbi.nlm.nih+2	Dermatophyte infections of skin and nails (tinea corporis, pedis, unguium). sciencedirect+1	GI upset, hepatotoxicity (oral terbinafine), taste and smell disturbances, rash. ncbi.nlm.nih+1
Pyrimidine analogue (Flucytosine)	Oral, often combined with Amphotericin B. ncbi.nlm.nih + 1	Converted to 5-FU in fungal cells → inhibits DNA and RNA synthesis. ncbi.nlm.nih+1	Serious systemic infections: cryptococcal meningitis and some Candida infections (always in combination to prevent resistance). ncbi.nlm.nih+1	Bone-marrow suppression, hepatotoxicity, GI upset.
Miscellaneous (Griseofulvin)	Oral. pubmed. ncbi.nlm.nih +2	Binds fungal microtubules → disrupts mitotic spindle and cell division; deposits in keratin precursor cells. pubmed.ncbi.nlm.nih+1	Dermatophyte infections of skin, hair, nails (e.g., tinea capitis). pubmed.ncbi.nlm.nih+1	Hepatotoxicity, GI upset, photosensitivity, CYP450 induction (drug interactions).

Combination Therapy

A combination of corticosteroid and antifungal drugs may help with sudden, painful flare-ups. Use an antifungal shampoo once or twice a week as part of your maintenance treatment to keep the problem from coming back (118).

CONCLUSION

Researchers and doctors find it hard to treat seborrheic dermatitis, an inflammatory skin disorder that comes back a lot. A weak skin barrier, an immune system that isn't working well, sebaceous gland activity, and Malassezia species

invading the skin all contribute to this skin problem. The present treatments, which are largely topical antifungals, corticosteroids, and keratolytics, help with symptoms like itching, scaling, and redness, but they don't always lead to long-term remission. Because traditional methods only treat symptoms and not the root causes of the problem, it is apparent that they do not work to stop relapses.

It has recently become obvious that the skin microbiota and immune regulation have a big role in how long an infection lasts. A recent research on the microbiome suggests that restoring microbial balance might provide new treatment options. In addition, the creation of biologics and non-steroidal anti-inflammatory drugs gives promise for treating instances that don't respond to standard treatment. Seborrheic dermatitis has a big effect on mental and social health when lesions grow on visible regions like the scalp and face, in addition to the apparent physical symptoms. This is why holistic treatment, which includes care for both physical and mental health, is so important.

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