“Drug delivery and development for the treatment of Seborrheic dermatitis”

A PROJECT SUBMITTED TO

NIRMA UNIVERSITY

In partial fulfillment of the requirements for the degree of

Bachelor of Pharmacy

BY

SOHAM.D.PATEL (15BPH099)

Semester VIII

UNDER THE GUIDANCE OF

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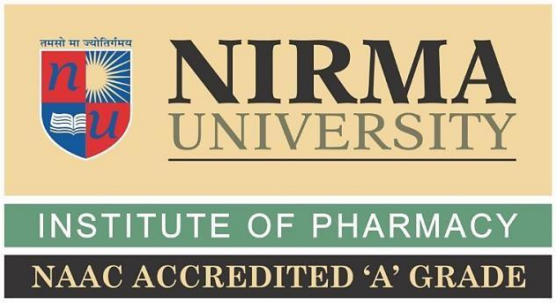

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APRIL 2020

Seborrheic Dermatitis and Dandruff

CERTIFICATE

This is to certify that "Drug delivery and development for the treatment of Seborrheic dermatitis" *is the bonafide work carried out by SOHAM PATEL(15BPH099), B.Pharm semester VIII under our guidance and supervision in the Institute of Pharmacy, Nirma University, Ahmedabad during the academic year 2019-2020. This work is up to my satisfaction.*

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Seborrheic Dermatitis and Dandruff

CERTIFICATE OF SIMILARITY OF WORK

This is to undertake that the B.Pharm. Project work entitled **"Drug delivery and development for the treatment of Seborrheic dermatitis"** *Submitted by* ***SOHAM PATEL****(15BPH099), B.Pharm. Semester VIII is a bonafide review/research work carried out by me at the Institute of Pharmacy, Nirma University under the guidance of "Name of a Guide and Co-guide". I am aware about the rules and regulations of Plagiarism policy of Nirma University, Ahmedabad. According to that, the review/research work carried out by me is not reported anywhere as per best of my Knowledge.*

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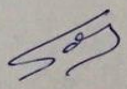
DECLARATION

*I, **SOHAM PATEL(15BPH099)**, student of VIII[th] Semester of B.Pharm at Institute of Pharmacy, Nirma University, hereby declare that my project entitled* **"Drug delivery and development for the treatment of Seborrheic dermatitis"** *is a result of culmination of my sincere efforts. I declare that the submitted project is done solely by me and to the best of my knowledge, no such work is done by any other person for the award of degree or diploma or for any other means. I also declare that all the information was collected from various primary sources (journals, patents, etc.) has been duly acknowledged in this project report.*



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ABSTRACT:

- The most frequently observed persistent inflammatory disease, which impacts people, is Seborrheic dermatitis; however, a sporadic phase of childhood exists. Seborrheic dermatitis is not necessarily induced. However, a contributing factor was identified to the proliferation of Malassezia organisms.
- Up to 5 per cent of the general population were impacted by adult type of seborrheic dermatitis. The disease mostly affects the head, face and periauricular regions and in certain instances often affects the central spine, axillae and genital area.
- Pruritus is not always present, particularly with scalp disease, and is fairly normal. Different therapies, including topical corticosteroids, topical anti-fungal drugs, topical inhibitors of calcineurin and a non-steroidal cream, are Essential. This essay discusses the clinical treatment of the adult population of seborrheic dermatitis in the USA.
- • Seborrheic dermatitis (SD) has a chronic inflammatory skin disease, with aggravation periods and recovery in adult patients with immunocompetence. In the first 3 or 4 months of childhood, a transient form of SD develops and improves, even though some cases can continue to recur for several months.
- There are instances of SD that arise and resolve within the first 3 to the last 4 months of age. Childhood SD can be limited to or more complex with scalp involvementThe SD for adults, which is far more prevalent than SD and tends to influence more males than females and in comparison to dermal lipid levels resulting from development could be present during puberty first of the androgen-driven sebaceous gland and from sebum secretion3 SD is normal after the age of 50.
- Individuals with Hiv see a marked rise, with an prevalence of SD occurring between 30 and 80 percent of people. The frequency of SD is significantly greaterResearchers have been able to reproduce SDs seen with strong association in patients with reduced CD4+T and fungal proliferation in an animal SD model.
- The rise Of this incidence is possibly linked to T cell lymphopenia and influences numbers of CD4 + cells involved in immune surveillance.

INTRODUCTION:

- The typical dermatological problems in the body's seborrheic regions, seborrheic dermastitis and dandruff, are seborrheic dermatitis. Both are seen as the same general disease, with certain features that are distinct from place and intensity and react to common treatments.
- Dandruff is scalp-restricted, itchy, skin-flaking without any noticeable inflammation. SD affects both the scalp and the neck, back-auricular and upper chest and may have identified erythema and induces attractive, swelling, inflammation and pruritus. Flaking is typically white or yellowish in the SD and dandruff and can be sticky or fresh.
- SD and dandruff together are projected to impact half the adult population. Their etiology is not well known given such a large prevalence.
- The infections are also related to a variety of intrinsic and environmental causes such as sebaceous secretions, fungal colonisation of the skin surface, human vulnerability and the relationship between these variables.
- Further knowledge on pathophysiology and methods for improved care was provided by genomic, biochemical tests and animal models study.
- Through this systematic analysis, we will outline existing experience through SD and dandruff and provide recommendations for potential study and care.

ETIOLOGY:

- While adult SD etiology is not well understood, there appear to be three major factors: sebaceous secretion, colonization alteration and skin floral metabolism (Malassezia spp), human receptivity and response.
- • Malassezia spp's work appears to be problematical in adult SD pathogenesis, however researchers concluded that malassezia spp's proliferation is a major aspect of relationship with the correlation between use of ketoconazole shampoo, decreased Malassezia spp's and clinical improvements in scalp AD.
- Since proliferation of the spp. is linked to exacerbating the SD of the malassezie spp. M. and Globe. Limta which are both commensal yeasts having an exogenous lipid source. The requirement was 1,3 M. M and balloon. M and balloon. Restricta can degrade sebum lipids, with the addition of additional saturated fatty acids and triglycerides.
- Many adjusted fatty acids, short-chain, which penetrate the skin and cause inflammation.8 A further theories suggest that host people with SD are altering their immune function and reaction. SD observations of changes in natural killer cells (NK1 +) and CD 16 + cells, an increase in inflammatory interleukins and a complementary lesion skin amplification compared with nonlesion skin and skin safety checks suggestimproving inflammatory reactions in people with SD.

EPIDEMIOLOGY:

- Throughout the United States and worldwide, SD is a popular dermatologic disorder.

- Its occurrence covers the three years-the first three months, adolescence and maturity with a median age from 40 to 60 years. Its occurrence is very high. SD covers the head (called "cradle cap"), the nose and the diaper region of children up to three months of age. Up to 42 percent can occur. In teenagers and adults, SD has an impact on the nose, upper chest, axilla, and inguinal folds of the skin and other seborrhea areas.
- The average prevalence in adolescents is 1–3%.
- The SD can be correlated with sex factors, such as androgens ,and males are more commonly influenced than In all age groups, youth (3.0% vs. 2.6%). No significant disparities were found between ethnic groups in SD.
- Patients of systemic failure Higher occurrence of SD was reported in patients such as HIV / AIDS, organ transplants [11,12], and lymphoma.
- Patients that have HIV have approximately 30% and 83% prevalence. CD4 + T lymphocytes are often identified as 200-500/mm3 and reduced numbers of CD4 + are also linked to lower SD. The cells CD4 + T were over 500/mm3 and fewer SD instances were recorded.
- Such findings suggest that SD may play a role in immunological defects.
- Neuroleptic mediated parkinsonism, lateral dyskinesia, traumatic brain damage, autism, facial nerves, spinal cord injury and attitude disturbance, as well as in people with a illness including congenital problems such as Down Symptom are also correlated with the Prevalence of developmental disabilities and mental illnesses, including Parkinson's disease.
- In fact, psoral and ultraviolet A (PUVA) psorial treatment can be used in patients undergoing seborrhoealike face dermatitis.
- Dandruff is much more prevalent in contrast to SD and impacts about 50% of the adult population worldwide.
- Consequently, it is more prevalent in men than in women.
- Dandruff starts at adolescence, at about 20 years of age hits peak occurrence and intensity and is less prevalent in people over 50 years of age.
- The frequency of a The study for African Americans in the USA and China reported 81 to 95%, for Caucasians 66 to 82, and for Caucasians 30% – 42%. Chinish. occurrence ranges across racial groups.

RISK FACTORS:

- Age
- Male sex
- Increased sebaceous gland activity
- Immunodeficiency, including[6]:
    - Lymphoma
    - Renal transplantation
    - HIV-AIDS
- Neurological and psychiatric disease, including[7]:
    - Parkinson disease
    - Stroke
    - Alzheimer dementia
    - Major depression
    - Autonomic dysfunction
- Exposure to drug treatment, including:
    - Dopamine antagonists
    - Immunosuppressants
    - Psoralen/PUVA
    - Lithium
- Low ambient humidity and/or low ambient temperature

PATHOPHYSIOLOGY:

• SD and dandruff pathogenesis were not understood , although the incidence is strong. In tests, however, many predisposing factors such as fungal colonisation, sebaceous behavior of the gland, and several factors that offer person susceptibility were identified.

- Proposed mechanisms for the pathogenesis of seborrheic dermatitis include:
    - Microbiota Disturbance of the skin Immune response compromised to Malassezia spp.
    - Related to reduced response to T-cells and complementary stimulation Increased production of unsaturated fatty acids on the skin surface Condition of skin neurotransmitter Irregular discharge of keratinocytes Epidermal barrier disorders due to genetic factors Malassezia spp.
    - It also requires sebum oxidation and saturated fatty acid intake, which disturbs the skin surface lipid balance.
    - Further evidence of Malassezia spp involvement. Requires separation from SD lesions and effective antifungal clearance of SD.
    - However, the evidence to connect ISD to ASD is inadequate.

I. Fungal colonization:

Several lines of research indicate that SD and dandruff have a pathogenic function in the yeasts of Malassezia. The lipophilic yeasts malassezia are mainly contained in seborrheic areas of the body.

Studies have shown that Malassezia is correlating with SD appearance / severity with the scalp of dandruff patientsand The Malassezia (M. globosa and M. restricta) is in greater number.

In addition, the may mechanism for action is anti-fungal behavior among the various chemical entities involved in the treatment of SD and dandruff, for example azoles, hydroxypyridone, allylamine, selenium and zinc. In fact, lipase action has been shown to Hydrolyzing human sebum triglycerides that produce acids such as oleic and arachidonic acidification that are unsaturated.

The aberrant keratinocytes are characterized by these metabolites, contributing to stratum corneal defects including parakeratosis, intracells and corneocyte envelope. Such modifications result in disturbed functionality of the epidermal membrane and Cause inflammatory reactions, with or without local inflammation apparent.

Further, Metabolites cause the activation of keratinocytes, which extend the inflammatory response, for instance IL-1α, IL6, IL 8 and TNF-α. Arachidon acid may also be the origins of pro-inflammatory prostaglandins that are able to induce inflammation by the mobilization of Vasodilatation and neutrophils. Particularly, the infection of Malasezia. was documented in the seborrhea (dry or grassy) and dermatitis of goats, dogs and monkeys.

Although such findings support the pathogenic effect There is also clear evidence for Malassezia in SD and dandruff of human predisposals and host associations with Malassezia lead to SD and dandruff pathogenesis instead of the sole involvement of Malassezia. Of regular skin, for example, most healthy people have observed malassezia, which is a commensal organism.

In fact, while the topical treatment of Oleic acid do Not cause noticeable improvements in Non-Dandruff patients, the dandruff patients' skin flaking was a consequence. Such findings Provide the SD and pathogenesis dandruff is related to the epidermal barrier deficiencies.

II. Sebaceous gland activity:

Sebaceous gland(SG)s, with the exception of palms and soils, are spread across the human skin sheet. Sebum deposition on the neck, nose and chest is highest.The development of sebum is under hormonal regulation and SGs in sebocytes are triggered by the mother's androgens at birth.

SGs are again triggered at puberty under androgens regulation, contributing to enhanced sebum secretion during adolescence that stays constant during 20-30 years, and then diminishes.

In males, the secretion rate is higher during active sebum secretion and remains elevated in 30 to 60 years; for females, the secretion rate decreases rapidly after menopause.

SD and dandruff also have a clear temporal association with SG's behavior and an elevated frequency in the adolescent from the third to sixth generations, with the cradle cap after birth.

However, natural sebum development may be in SD patients and often SD is not in the cycle of people with excessive grain output. Although SG operation is closely linked to The origin of SD and dandruff is not a reliable source for sebum production.

Besides sebum development, lipid composition abnormalities may have a function in SD formation, probably via a favorable environment for Malassezia growth.

Triglycerides and squalenes were reduced in SD patients but there have been slightly higher free fatty acids and cholesterol in patients.

Higher rates of Free Cholesterol and fatty acids can be induced by The breakdown and Triglyceride in the lipase of Malassezia and encourage malassezia development and the recruitment of inflammatory skin infiltrate.

III. Individual susceptibility:

Many conditions lead to SD pathogenesis, in addition to sebaceous development and invasion of Malassezia. It has been found to play a role in the individual's vulnerability through way of the epidermal membrane integrity, the household immune response, neurosis causes and emotional tension.

IV. Epidermal barrier integrity:

Stratum cornea (SC), a protection against microorganisms and poisonous agents from water runoff and absorption the atmosphere, serves as the anucleated external epidermis barrier.

Epidermal barrier integrity The CS is formed of Most keratinocytes, "corneocytes," are divided terminally into lamellae of lipid type, embedded in complex structures known as corneodesmosomes intercellular cell adhesionChanges in scale or form of the lamella of the corneocyte, the amount of corneodesmosoma and the thickening of SC can result in changes in the functions of the Epidermal Permeability Barrier (EPB).

Usually, the lipid organisation's sebum can affect the desquamation support.

Nevertheless, altered hydrolysis can disturb the lipid structure and disrupt the desquamation process, which results in aberrant barrier role in SD and dandruff.

In support of this definition, the electron-microscopic dandrug skin comprising intercellular malasezia yeasts, alterations in the form of corneocytes and corneodesmosomes and a distorted lamellar structure of the lipid are found to be resistant to structural abnormalities.

In line with systemic results, patients with dandruff (higher sense of scratching or flaking) were found to be more Topical therapies of histamine or scalp oleic acid are prone rather than controls.

These findings indicate that the skewed role of EPB will render the dandruff worse. Recent genetic studies have shown that disrupted activity of the barrier may also induce SD-like conditions in humans and animals.

Biochemical research has also shown that, in the absence of obvious inflammation, the skin of dandruff shows Protein compositions have improved and SC ceramides and free fatty acids have been updated. Such studies stress The significance Of the reconstruction and preservation of barriers in SD and dandruff management.

Immune response

Incidents of In fact in HIV / AIDS cases, immune suppression, both frequency and severity of SD are related. Although no significant variations were observed between people with and without SD in this population in Malassezia rates, it is possible that the predisposition could be an immune or inflammatory reaction.

In one study, high rates HLA-AW30 and HLA-AW31, and HLA-A-A32, and HLA-B18 human-leukocyte-antigene were observed in SD.

Increased concentrations of In SD trials, minimum serum IgA and IgG antibodies also were detected.

There was, however, no rise in anticorpore titres against Malassezia which indicates that higher development of immunoglobulin is more a reaction to metabolites from yeast.

Such metabolites induce intense inflammatory reactions, which includes the infiltration of normal killer cells and the macrophage, rapid local activation of additional inflammatory cytokines such as IL-1α, IL-1β, IL-6, TNF-α, and other inflammatory cytokines in contaminated skin areas[54]. The absence of progress in anti-malasses also suggests a shift in the cell's immune response rather than laughter. Controversial remains the unique function of lymphocyte operation.

Genetic factors

Before late when animal models and human experiments established hereditary and recessive, dominant types of SD and dandruff the genetic elements of SD and dandruff have been underappreciatedOF1 mouse induces seborrhea, rough fur, alopecia, spontaneous mutation in an outbred human, delays to the development of homozygous mutants in autosomal recessionary "seborrheic dermatite" (see) mouse — often irregular pigmentation.

The swollen sebaceous gland, hyperkeratotic, paraceratosis, Epidermis and dermatic acanthoses and toxic infiltrations were found during historical review There have been no presence of yeast or dermatophyte. This is the first animal SD model to display strong inheritance style, even though it is still not defined by the underlying mutation.

Transgenic mices in DBA/2 history with Transgenic cell receiver 2C T (TCR) formed a highly Seborrheic inflammatory syndrome including our face, close to the head, in the region of the moss, in keeping with the function of altered immunity in SD pathologies.

Positive pathogens by PAS were also reliably found in lesional skin but not instantly evident by diseased mouse or DBA/2 control mouse in non-lesional skin.

In fact, antifungal treatment reversed the manifestations of clinical pathology and-darkened PAS. These findings help the hypothesis SD plays a significant role in the development of fungal infections and immune deficiencies.

The raw coat (rc) mouse displays sebaceous hair hypertrophy and gray mask, and a further random mutant mouse strain reveals an SD-like phenotype. In an autosomal recessive manner, the rc is distributed. The origin of the RC-phenotype, which is expressed in the epidephrase surface layers, has since been established as a Missense mutation in the Mpzl3 gene.

Mice with white fur produced a more serious and permanent phenotype and dandruff of inflammatory skin in the seborrhoeal regions, with our Mpzl3 knockout mice re-sampling the Rc phenotype.

We have shown that immune deficiencies have not contributed to the early-start inflammatory skin phénotype .

However, MPZL3 Epidermal and mouse disturbance distortion in organotypical models MPZL3 is demonstrated by human skin is a significant epidermal differentiation regulator.

Interestingly, in patients, autosomal dominant seborrhea-like dermatitis was caused by the framework- ZNF750 shift mutation and an epidermal distinction control transcription factor and an upstream MPZL3 regulator. Such study in human and animal models

highlights the impact of SD and dandruff pathogenesis on pathological disease distinction and explores the genetic basis of some of the predisposition factors referred to above. Such Animal model should being critical instruments for discerning the mechanisms underlying such pathogens and defining new priorities in enhancing their treatment.

Emotional tension and neurogenic factors

High SD events were seen for a long time, particularly with severe seborrhea, providing favorable proliferation conditions for Malassezia in patients who suffer from Parkinson's and neuroleptic-inducing parkinsonism.

In patients with unilateral parkinsonism, bilateral seborrhea has been identified, indicating that such shifts in sebum is likely to be neuro-endocrinologically rather than strictly Neurologically controlled.

For Parkinson's patients, rates of the α-melanocyte hormone (α-MSH), possibly attributable to insufficient dopa-minergic feedback, were elevated. Furthermore, α-MSH decreased therapy with L-dopa and restored MSH-inhibiting factor synthesis, which enhanced sebum separation.

In fact, the brain impairment (e.g. fracture, spinal injury) is associated with SD[93]. There is also proof of this. Facial immobility and facial weakness in Parkinsonians (mask-like faces) will contribute to high sebum allegation and SD, but only on the impaired side.

As bad hygiene was implicated in SD, these findings indicate that the presentation of the disease could be affected by prolonged reservoirs of residual sebum correlated with immobility.

SD is even more common in psychological and mental stress conditions.

Specific considerations Food as a possible risk factor for SD has been studied in the past. Zinc deficiency can be present in seborrheicdermatitis-like rash in Patients of enteropatica acrodermatitis, riboflavin, and deficit in pyridoxine.

SD was frequently correlated with many psychiatric problems, Family polyneuropathy amyloidotic and Down syndrome, for example.

To sum up, in SD and dandruff pathogenesis several predisposing factors were established .

In addition to many other variables and relationships, Host conditions of outbreak and sebaceous secretion are present and plentiful and decide a person's vulnerability to SD and dandruff. In a plausible case, an aberrant epidermal barrier feature may occur due to the predisposition of chromosomes, and unnecessary or changed composition of the sebum will intensify EPB destruction and establish a desirable climate for colonization of Malassezia. The compromised EPB function allows Malassezia and its metabolites easy to reach and provokes Host immune reaction and epidermis.

The inflammatory host reaction also disturbs the separation of the epidermis and the development of membrane, which thus damages pruritus which resulting itching, contribute to immune activation loops, pathological epidermal distortion and membrane dysfunction.

Histopathology:

Superficial perivascular infiltrate of lymphocyts, acanthosis, focal spongiosis and focal parasites are typically seen in the dermatopathology of seborrheic dermatitis.[11][12] Shoulder parakeratosis refers to a scale-crust aggregation of infundibular oscillations. The stratum corneum may have malassezia.

The past of the progression from acute to persistent SD usually reveals the production of a lymphocytic lymphocytic infilltrate via spongiosis to psoriasiform hyperplasia.

Severe SDs also include keratinocyte necrosis, degradation of the focal system, and leukocytoclasis.

History and Physical:

The spread of seborrheic dermatitis is the most important clinical function. In places where the skin is rich in sebuminous gland, particularly on your head and on your neck, lesions occur. ISD is typically asymptomatic, although it also coexists with atopic dermatitis. Pruritus, on the other hand, is normal in ASD, especially in scalp and patients experience frequent burning, while atopic dermatitis is typically not documented in history.

Seborrheic dermatitis is distinguished by folliculocentric, brown, white, and sometimes represented as a dark, salmon-colored papules and plaques with a fine scale. It that occur in one or more places where bent surfaces are less scaled and the margins of lesions are poorly described.

The mildest type of SD is a non-inflammatory variety generally named pityriasis capitis or sicca.

In this situation, it has an effect on the scalp and "barbal area" and is related to the shedding of little light-colored skin flasks often referred to as "dandruff" in the past.

Rodening the nose, scaling and dandruff are growing explanations for appearance. Persistent dyschromia with variable hyper / hypo-pigmentation can occur on darker skin. Additional Malassezia spp circumstances. The lesions on the anterior chest are typical for psoriasiform Morphology, but also have a petaloid look, with these ring lesions usually found on the face of darker skin phenotypes.

In adolescents, folliculitis can be present, like pityriasis versicolor and (Pityrosporum / malassezia). There is a unusual improvement in the pityriasis (which is imitated by a collarette scale of pityriasis rose).

a. Adult SD:

Seborrheic dermatitis in head and neck is characteristically symmetrical and covers the central third of the face (including the region of the malar), central area of the forehead and eyebrows (especially their medicines); it occurs in a number of different areas: the nose, skin and arms. SD affects nasolabial and alar folds, and with involvement of the anterior (lash) side, blepharitis is widely mentioned.

Difference from adult seborrheic dermatitis in psoriasis is important. The red papule and plaque is indurated and has a given margin and a loose, silversome lamella scale. Psoriatic adjustments will occur to the clocks and the Auspitz indicator is optimistic. Sebopsoriasis has been contested as a separate scientific group.

b. Infantile SD

ISD usually occurs for 4 to 6 months in the second week of life. The face distribution of ASD, the diaper region, the skin of the back, and the axillae may be present. In fact, rash is not jerky or unpleasant, so children seem delighted, but parents may be upset. This is gentle and independent in general.

A typical appearance, known as a cradle cap, refers to an adherent, yellowish scale layer, forming a firm mass that may grow on a crown and on the front of the scalp and growing from a bran-like surface, significant ooze and grassy layer.

The amiantaceous pityriasis in the ISD can occur. This reflects a variety of clinical findings, but is not unique to seborrhoic dermatitis, which may develop in older babies or small children. There is typically a dense, red, or yellow layer, which covers and links scalp hairs with tufts and can also occur with Psoriasis of the head, dermatitis atopic and capitis of the tinea.

the clinical treatment Of ISD includes atopic Dermatitis. Usually, it develops on facial and limbs (elbows and knees) and it is pruritistic and vulnerable to crying and spares the head. The infant is generally disturbed by the rash and scratch symptoms are a normal finding.

Evaluation:

Routine examination of seborrheic dermatitis is not mandatory, but HIV serology in serious SD cases, particularly when sudden onset, should be speededed up. In older adults, clinical features of Parkinson's disease need identification.

Medications for the condition are to be investigated.

The following tests may be helpful in the differential diagnosis:

- KOH examination of skin scrapings
- Swab for microscopy, culture, and sensitivities
- Histology and direct immunofluorescence
- HIV serology; VDRL
- Serum zinc levels
- ANA; ENA; ESR

Burden of Disease:

An approximate 50 million American pelvis suffering, which expend an total of $300 million a year on scalp scratching and flatulences, has been reported .

In addition to physical pain, dandruff becomes socially uncomfortable and adversely influences self-esteem in patients.

While SD is much less common, ambulatory visitors in the United States alone cost $58 million in 2004, and prescription drugs $109 million.

The total direct loss of SD was calculated at 179 million dollars, along with over-the-counter goods and healthcare care, and another 51 million indirect losses of missed business days.

Moreover, since SD frequently takes the shape of psychological depression or poor self-esteem on the face and on other noticeable regions, it dramatically adversely affects patients' standard of living (QOL) at $1,2 billion.

In comparison, while in SD patients, QOL influence was smaller than in dermatitis atopic or contact patients, QOL results have been more severe damage to skin and sunshine and more highly affected are children, younger patients and higher educational topics.

Management of Adult Seborrheic Dermatitis:

SD therapy primarily aims to explain and improve the obvious symptoms of the condition, Pruritus in particular. Since SD is associated over several years to intermittent reciprocal periods, several preventive regimes were recorded.

A thorough review of literature indicates that there have been no clear interventions to assess the severity of SD, with symptom progress and intermittent (short-term) success steps, improvements in Malassezia spp colony count, patient satisfaction and cosmetic acceptability of care.

Given several clinical trials testing several SD therapies, a longitudinal analysis of existing results shows that only randomized, regulated experiments "comply with the clinical proof requirements.

However, there are several studies showing efficient SD therapies for scalp and glabrous skin areas.

Multiple topical therapies have been used for the diagnosis of SD of the scalp and/or glabra, mainly shampoo and cream formulations.

There were many trials including multiple agent (number of tests, N = Number of successful treatments) including the topical drug choices for SD: Selenium sulfide (2.50 sulphide) in shampoo (1, N=95) propylene (13%), N=37%), hydrocortezone (1%), ketoconazole (3%), propylene glycol (1%) (1%). (3%), propylene glycol (1%).in the shampoo

Many topical compounds Terbinafine 1% cream / solution, metronidazole 1% gel and azelaic acid 15% gel are used for treating SD, although there is evidence of this. limited 3, 8 These same compounds have been tested with additional trials as well as agents such as topical calcinerurin inhibitors. 3, 8 Newly released, "proof" evaluation of SD diagnosis has included.

The practical application for the clinician is discussed below, in particular with the main agents for SD which have been studied and examined in detail. Written proof, detailed research reviews and professional knowledge are practically accessible.

Therapy:

Several methods of treating seborrheic dermatitis may be successful. The most popular therapies are strategies to prevent skin yeast invasion, Remove salt, scale and reduce bacteria, alleviate pruritus and erythema. Such treatments contain antifungal, corticosteroid, immunomodulator and keratolytic agents (see Table 3). Nonetheless, there are many forms of anti-inflammatory properties in several modes like the keratolytic properties of Selenium, Zinc, and Tear formulations, which are found in various antifungal agents.

TABLE 1 : Categories of Medications Used

Drug Types used in seborrheic dermatitis Classification
AntifungalS: • **Azole** • **allamines** • **benzylamines** • **hydroxypyridones** • **selenium** • **zinc** • **t tree oil** • **imunomodulators corticosteroids** • **mitronidazole** • **TAR** • **phototherapy**

Prescription AntifUngal Medications:

a. Azoles.

The center Of the Antiseborrheic medication is antifungal drugs, often in the form of azoles. Such agents act by interaction with the P-450 fungal cytochrome (CYP 450) mechanism by inducing ergosterol as an essential part of the fungal cell wall.13 It

contributes to an rise in sterol precursor development, a fungal cycle that does not allow the fungus to develop or replicate. Azolol is often widely known to have anti-inflammatory properties; they impede the development of 5-lipoxygenase, that inhibits skin synthesis of leukotriene-B4.

At least ten randomized controlled trials have been conducted of ketoconazol (Nizoral, PriCará) that have shown their effect on scalp dermatitis and other body sections. Through numerous common, Ketoconazole is effective in the manufacture of over-the-counter products such as foams, gels and creams. In case of daily stimulus reversal, occasional usage of ketoconazole is also successful 16, and in conjunction with other medications such as zinc and selenium, it may also be used as a 4-week 200 mg / day scheme.

TABLE 2 : Antifungal Formulations

Antifungal formulations
azoles • **Ketoconazole (nizoral) : 3% Shampoo, Foam, Cream, Gel, 300-Mg Tablets; Itraconazole (sporanox) : 100-Mg Tablet,Bifonazole (canesten)** **allylamines** • **Terbinafine (lamisil): 1% Gel, Cream, Solution, 250-Mg Tablets** **benzylamines** • **Butenafine (mentax) : 1% Cream** **hydroxypyridones** • **ciclopirox (loprox): 0.77% Gel, Cream , Shampoo , Solution, Suspension**

Itraconazole (Sporanox, Janssen) is another valuable azole. Oral itraconazol is an association for the skin, the hair, and the nails for strongly keratinised regions of the body. The topical pool, which is beneficial for a shorter period, tends to improve the compliance of skin for around two to vier weeks.17 The recommended The dose is 200 mg a day for 7 days for the itracónazole capsules. 17 Ointment of bifonazole in Canada, but not in United States (e.g. Canesten, Bayer) is safe. Few antifungals have also been used successfully. Drug Allylamines, benzylamines (butenafine), and hydroxyridones are supplementary anti-fungal drugs used in the diagnosis of seborrheic dermatitis.

Allylamines and benzylamines. Related action mechanisms have been producedSqualene epoxidase is an enzyme which is essential to the development of the fungal membrane in both terbinafine (Lamisil, Noviartis), and allylamine and butenophine (Mentax,

Penederm). Terbinafine also extends directly through sebum. It's orally available. It can be accessed. Residual amounts persist in the skin for up to 72 hours following the topical application of butenafin. Butenafine has anti-inflammatory properties, which suppress the erythema of ultraviolet B (UVB).

b. Hydroxypyridones.

The hydroxypyridone family is comprised of Ciclopirox (Loprox, Medicis). This medicinal drug may be used in the form of paste, spray or solution as a residual material (topical suspension). Ciclopirox's mechanism of action differs from other antifungals.19,20 Moreover, ciclopirox has anti-inflammatory propagations that prevent prostaglandin and leucotriene production. The Ciclopirox also has anti-inflammative properties. Cyclopirox has been recommended to be handled with 1 to 1,5% shampoo used twice or third times a week before removal, and then one week before each other for prophylaxis22.24. The procedure for the production of fungal cell membrane prohibits essential substances from being released in the membrane cells.

Harmful events. Harmful incidents. In approximately two to three percent of patient, the adverse effects associated with topical antifungal products are irritating contact dermatitis, and burning , itching and dryness. As CYP 450 system is interfered with by oral antifongal agents in the fungus, the CYP 450 system may also interfere and limit their practical application in seborrheic dermatitis treatments. Itraconazole and fluconazole (Diflucan, Pfizer) of the antifungals that function through the fungal CYP 450 mechanism have the lowest binding and thus least adverse effects to the CYP 450. Ciclopirox is more recognized and tolerated than ketoconazole among the antifungal agents.

TABLE 3: **Adverse Effects of Various Therapies**

Similar medication risk consequences:
Topical anti-fungal agents (for starters, azoles and allegamine): dermatitis touch, scratching and pain, dryness
Metronidazole: touch sensitization
selenium: hyper-pigmentation (rare) tea tree oils, irritant direct contact dermatitis
Topical correctional steroids: skin atrophy, telangiectasis, follicular tissue, hypopigmentation

c. ***ANTIBIOTICS:***

Metronidazole (Flagyl, Pfizer) was successful in gel-formulation in a controlled, double-blind Parsad et al. trial for 8 weeks, although used twice daily.25 While not typically correlated with topical metronidazole, adverse effects may consist primarily of a unusual sensitisation of touch during frequent use.26

d. ***Nonprescription Antifungal Agents:***

Selenium. The over-the-counter shampoo formulas (for starters, Selsun, Ross) include selenium sulfide. Seborrheic dermatitis was affected as a twice-every-week diet,27 but still significantly less than ketoconazole was observed in the same sample. A unusual correlation with hyperpigmentation has been recorded with the topical usage of selenium.

Pyrithione zinc. The active ingredient is Pyrithione Zinc, but its mode of action is unclear in certain dandruf safe shampoos over-the-counter (such as Head & Shoulders and Procter & Gamble). This drug has been shown to be less than ketoconazole during a head-to-head study31, but can also be successful either alone or in conjunction with ketoconazole or cyclopirox.27 This substance is known to be in shampoo concentrations of 1 % and 2% and the 1% in the mixture between cream.

Oil from the tea tree. Description: Tea tree oil is alternifolia melaleuca. a safe solution to treating seborrhea skin dermatitis extracted from an Australian vine. For one analysis a certain advantage was observed, with a concentration of 5%;32 the estrogenic and antiandrogenic effects of the drug therefore hinder its functional use33. Occasional irritant dermatitis was primarily the product of unusual harmful incidents.

e. ***Topical Corticosteroids.***

Short term topical corticosteroid treatment is not linked to antimicrobial action, often administered to reduce the disease's inflammatory portion. Several different-potence corticosteroids, primarily hydrocortisone and beclomethasine dipropionate are used for the diagnosis of seborrheic dermatitis. The subsequent occurrence of skin atrophy, telangiectasias, folliculitis and hypertrichosis have also been linked with topical corticosteroids. Such incidents lead to better-tolerated anti-fungal medications replacing

topical corticosteroids.35 Topical dermatitis, as consistent with HIV infection, was not linked with an elevated amount or rise in colonies of malassezia, i.e. Pityrosporum, and corticosteroid treatment could also be more effective under this circumstance.

f. IMMUNOMODULATORS :

Tacrolimus and tacrolimus and tacrolimus. The Hollywood. In the treatment of seborrheic dermatitis, calcineurin-inhibited tacrolimus (Protopic, Astellas) and pimecrolimus (Elidel, Galderma) are effective. In in-vitro action against Malassezia Tacrolimus has, however, also potent fungicide.37 Tacrolimus and pimecrolimus have been successful in randomised trials, and are not consistent with the impact profile of the harmful effect of corticosteroids. They are primarily anti-inflammative hindered by cytokine production.

The side-effect history of such medications is, though, problematic in itself. The label change states that although there was no defined a causal link, uncommon cases of malignance (for instance, Patients treated with topical hemispheric calcineurine is confirmed to skin and lymphoma; hence a longer-term treatment of these agents and a Tacrolimus38 and PimeCrolimus box warning problem in 2005 and 2006 should be prevented. Therefore, tacrolimus and pimecrolimus should mainly be used in the short term and should be noted as off-label in sebrheic patients dermatitis. The long-term usage profile appears to be problematic owing to potential harmful reactions.

Scalp Seborrheic Dermatitis Shampoos:

(Nizoral, McNeil Public Health, McNeil-PPC Party, Inc.) SHANPOL 2 percent. 2 percent. Ketoconazole is a malassezia spp imidazole antifungal agent and may have a slight direct anti-inflammatory benefit.

A total of seven double-blind, randomized, vehicle-controlled trials using a "evidence-oriented" review of ketoconazole shampoo (N=575) showed good clinical results with 88% of subjects8. Importantly, ketoconazole was found to be 2% more effective than ketoconazole available in the United States by prescription.

The normal prescribed dosage of ketoconazole 2% shampoos has been shown to be efficient, for instance regularly, occasionally over an ordinary duration of four weeks3,8,12,13 moderate usage of ketoconazole 2% shampoo, for example once a week.

Studies demonstrating minimal percutaneous absorption and a low risk for irritance or touch susceptibility help the desirable health profile of the ketoconazole 2 percent shampoo.

1% shampoo with Ciclopirox: (Medicis, Arizona). (Loprox, Scandinavia.). Five controlled double-blind clinical studies of ciclopirox are conducted, including an assessment of dose response8. Twice weekly usage is usually acceptable over a duration of at least four weeks.

Like in the case of ketoconazole, 2 percent shampoo is health- and tolerable. Ciclopirox has demonstrated to have anti-fungal efficacy against Malassezia and other superficial fungi.

Other shampoos:

Scalp SD effectiveness has been seen in several trials of Shampon Selenium Sulfide 2,5% and zinc pyrithium 1,5% and 2,0% (Head & Shoulders, Procter & Gamble), (Selsun, Ross Products Group, Abbott Laboratories, Columbus, Ohio)of tests finding both agents to be stronger than the firs.

The Tar shampoo may be darkened in the colour.

Tar shampoos, Also not well researched for adult Skin SD, either greenish or Light dye coloring black, brown, or grey eyes.

Practical application:

The monotherapy of Ketoconazole 2 percent or shampoos 1 percent can be successful when used twice weekly for a span of at least four weekly for patients with mild to moderate scalp SD.

More regular usage in most patients is unlikely to have added benefit. More extreme situations require the application of Typically a paste, spray or mist for 1-2 weeks, the topical corticosteroid (TCS) at bedtime.

The therapy of the antifungal shampoo has been ongoing for at least four weeks, thereby helping to speed up signs and effects. When recurrence is a concern, all anti-fungal shampoos may be used once or twice a week over the long term.

Scalp seborrheic dermatitis nonsteroidal "put on" formulations:

(Extino SmithKline, boot / glaxo) Ketoconazole 2 percent foam. Ketoconazole 2 percent (Extine) is also eligible for scalp SD as an alcohol-based "leave on" foam formulation. For more refractory situations, though, the usage of a mid-to-high power TCS for 1 to 2 weeks is likely to result for quicker and greater gain.

Although a variety of therapies have been studied for non-scalp SD, ketoconazole 2% cream and cyclopirox 1% cream are the primary topical non-steroidal agents in the United States.

Topical calcineurine inhibitor therapy (e.g. pimecrolimus), particularly for patients who recover sometimes, was also investigated and often used.

AntifUngal Agents.

2% Ketoconazole (Nizoral, Kuric) cream (Xolegel, Aqua Pharmaceuticals, LLC, 2% Ketoconazole) mist. Ketoconazole use was shown to improve SD twice daily for four weeks, even for glabrous Skins of the face and of the chest; but the response is sometimes lighter than the TCS3,

Other Ketoconazole 2 percent formulations, such as gel or foam may provide a gain for Any patients prefer hair-present body places (E.g. Central malignant breast). Application of Ketoconazole 2 percent

1% cream (Loprox) from Ciclopirox. Ciclopirox 1 percent of cream twice daily was substantially better than vehicle cream in the 28-day, SD-controlled dual-blind vehicle tests (N=129) and successfully sustained over the next 28 days with a single-day treatment other antifungal products.

Other antifungal products. Data are also provided with topical 2% miconazole cream (Micatin, WellSpring Pharma, Sarasota, New JerseyTerbinafine 1% (Lamisil, Novartis PHC, East Hannover, New Jersey) and Terbinafine 1% (Lamezil). 1% cream (Terbinafine). Terbinafine 1% cream is also given for this drug. Other agents are restricted in their data.

Topical calcineurin inhibitors.

Topical calcineurin (TCI), 1 percent pimecrolimus (Elidel, Novartis Pharmaceuticals), 0.03 and 0.1 percent tacrolimus (Protopic, AstellasPharma, Inc., Deerfield, Illinois) has been recorded for two years in the United States in patients to manage inflammatory lesions caused by the atopic dermatitis.

Due to their anti-inflammatory effects and the lack of adverse reactions related to the long use of TCS agents , particularly face-to - face, TCIs have been examined for face SD treatment20. these agents are decreased by inhibiting development of T lymphocyte cytokine. These effects have been studied for TCIs.

1% cream of pimecrolimus. The test of pimecrolimus 1 percent (pimacrolam) cream was Conducted with mild to extreme facial SD in a 4-week dual-blind, randomized, car-controlled, four-week, sample of male mostly adult (mean 59,6 years of age) and was compare with vehicle cream (n=47).

Several more studies have shown that 1 per cent cream of pimecrolimus is successful in SD, especially in the facial region (22–20). Posite therapeutic outcomes test results have included even patients who are not completely receptive TCS, facial SD (N=20) in Korean patients, and African patients who had a good therapeutic outcome. (N=20). Moreover, SD is a non-label program for TCIs. also necessary To note THAT BOTH TCI Have a "blau box warning" mark, licensed in their US Food and Drugs Administration (FDA), based on possible issues of malignancy interaction based on animal evidence. Moreover, rosacea-like dermatitis was reported for use with rosacea, including facial SD. It is evident that the lack of clinical evidence for the use of TCI and increased risk of malignancy is focused on existing knowledge.

Many subjects that do not include drugs. Theme cream program, anti steroidal (promiseb, Pharma, LLC, Bridgewater, New Jersey). Topical NS cream program. A water dependent, fragrance-free, non-steroidally (NSTD) cream licensed by the FDA in the United States as 'natural aid' is 'to relieve and reduce signs and symptoms such as itching, erythema, scaling and discomfort of seborrhea or SD.' The following is indicated: As a current "treatment product," no specific active ingredient(s) can be reported. Neither the piroctone olamine, nor any multifunctional antioxidant Many emollients, skin disorders (e.g. ethylhexyl palmitate, bisabololol, shea butter, Vitis vinificera), (e.g. telmesteins, tocopheryl acetate, tetraphalmitate ascorbyl), or the allantoins and glycerins containing allantain and glass are some ingredients that that lead to SD improvement following application of this NSTD cream. Some studies indicate that NSTD creams can at least partially be successful for SD because of Malassezia spp reduction and the presence of piroctone olamine is probably associated with this antifungal effect.

A randomized, paralegal, multicenterIn order to evaluate NSTD cream safety and effectiveness (N=38) twice daily, desonid cream twice daily was conducted with 0.05 percent (N=39) for mild facial SD in adults for a period of time. Of 14 days or more and up to a maximum of 28 days. The number of subjects who achieved "clear" or "almost clear" on the basis of the IGA was 92% in the arm of the Desonide test and 85% in the arm of the NSTD cream. Whereas IGA's ranking of Day 14 patients were higher for the desonid-treated community (39%) than for the NSTD category (20%), 71.4% for the clearly identified patients on day 14 were noticeable in the NSTD arm, relative to 14.3% for the desonid arm.33 The amount of patients surveyed in that pilot was small, but Howev was smaller than 14.3% for the desonide arm.

Many forms that are not steroidal. For some patients with SD it was shown to be successful for Topical and 15 percent azelae gel, and both papulopustular rosacea (0.75 percent gel and 1 percent gel) and mild to moderate SD face monotherapy were shown to be effective in patients.8,34–36 Eight percent succes of succinate lithium / zinc sulfide was also shown in SS,3,8 percent positive for SS,3,8 percent achieved for SS.3 succinate /zinc sulphate 0.05% ointments

Many non-steroidal treatments have been shown to work in some patients with face SD, including topical metronidazole (0.75 percent gel and 1 percent gel) and azelaic 15 percent gel, and both papulopustular rosacea and SD are monotherapies with the facial sensitivity with patients.

Practical application:

Most patients react favorably with a variety of non-steroidal treatment approaches, In specific, for treating moderate to extreme teenage and non-scalp SD, pimecrolimus 1 percent cream, or NSTD cream for moderate to serious skin, like ketoconazole 2 percent crème, ciclopirox 1 percent crème. Numerous doctors may have a stronger effects and indications of SD within 1 to 4 weeks if the effects involved are moderate. Such agents will help to avoid re-occurrence without fear of adverse sequelae except pimecrolimus 1% oil. It could be necessary for an antifungal agent to be used every everyday to avoid reoccurrence of face SD in certain cases.

Such The tracking of milder flares by four agents may be performed acceptable pace at periodic intervals. Nonetheless, in some situations that are quicker and/or linked to mild to extreme symptoms of mild-to-serious intervention, a short-term course TCS used once or twice a day over the period of one to two weeks (subject to potential) and used along with a non-steroid agent is a reasonable option. When the SD is "cooled off" into a milder situation or cleaned up and is normally within a couple of days, the TCS should be suddenly halted or tapered periodically for at least a few more weeks, to avoid

reoccurrence of the nonsteroidal substance. No way to get close to SD is feasible. The clinician will modulate the manner in which the particular patient treats facial SD on the grounds of disease frequency, therapeutic reaction and mutual propensity.

Scalp and Nonscalp Seborrheic Dermatitis Topical Corticosteroid Treatment:

The adult care analysis for SD revealed a fairly weak publication of inquiry into the use of TCS for SD, particularly compared to the large number of studies on TCS for psoriasis.

Moreover, The first or second line SD agents depending on the nature of a disease are known to be TCS.

TCS is also successful at rapidly removing noticeable indicators and related effects from a poor to a higher-mid-performance. For scalp SD, often patients are diagnosed with more pruritus than face SD, which supports initial care with a higher TCS intensity for the first week or two and daily tape over the subsequent 1 to 2 weeks.

This was reported that the relapse of SD happens earlier and more commonly by utilizing TCS relative to The clinician is able to choose the power and vehicle of TCS according to the strength of TCS and its action locations. Antifungal agents and other topical non-steroids.3,8,33

Because TCS is in a variety of formulations for vehicles. For most situations, The usage of TCS is generally restricted to 1 to 4 weeks, as reaction is typically fast. In comparison to sudden discontinuation, tapering of frequency can theoretically lower the probability of re-occurrence found with TCS usage on SD, but not officialally tested.

Practical application:

TCS is used to monitor signs and effects as rapidly as possible independent of the location or extent of adult SD. A TCS may be used 1 to 2 weeks and sometimes for less, typically at small to medium power. Higher potency agents are also particularly required for mild and moderate to extreme symptoms for adults with scalp SD including pruritus, petting and/or fire. This physician tends to use a small to medium-power product on the face and other glabrous skin sites for 1 to 2 weeks in adults for a total of Each day for 1 to 2 weeks. It helps to minimize duplication, Which anecdotally Which is just a recovery as it occurs soon after cessation of TCS. This is also beneficial to use a product that is not irritant and ideally moists, such as hydrogel, lipocream or emollient cream. The short-term usage of TCS with concomitant topical non-steroidal treatment, which persisted for a more extended time in order to maintain clinical effect and avoid replication is of interest, In fact in adult patients at risk for repeated facial SD recurrence. The medium to high-potency TCS, foam or spray solution sometimes manages the condition in a span of 2 to 3 Weeks , And often longer, for Adult Scalp SDs especially moderately to serious. It can be controlled in a way close to the previously mentioned facial SD therapy. Usage of 2% shampoo or 1% shampoo twice weeks with cetoconazole should Using a TCS for adult scalp SD respectively, which starts After the tcs stops. It Is necessary to be cautious because extended usage of TCS can lead to adverse reactions including atrophy, striae, and telangiectasis

Clinical presentations:

Adult SD is more commonly present on the face and/or skirt as misunderstood erythematous scaling dots, with one more preferred location being involved. Scalps, front hair (Figure 1), lips, forehead glabel region, nasal slats, melolabic fold (Figure 3), Ears (Including outer Canals, front Auricular Region, RetroAuricular field), middle of the chest (the sternum portion) and genital area are common affected. Such locations are regular affections. Pruritus is not an unavoidable adult SD trait, but sometimes happens with the presence of scalp.

The problem of Whether AND no Dandruff , Which Is characterized AS thin Scalp scaling is part of adult scalp without any apparent inflammatory intervention, has been discussed for a long time. More recent research has demonstrated that in most instances dandruff is a gentle type of SD. For other cases, SD can produce more entangled regions, often Oval plaques (medallion lesions) of detached plaques.

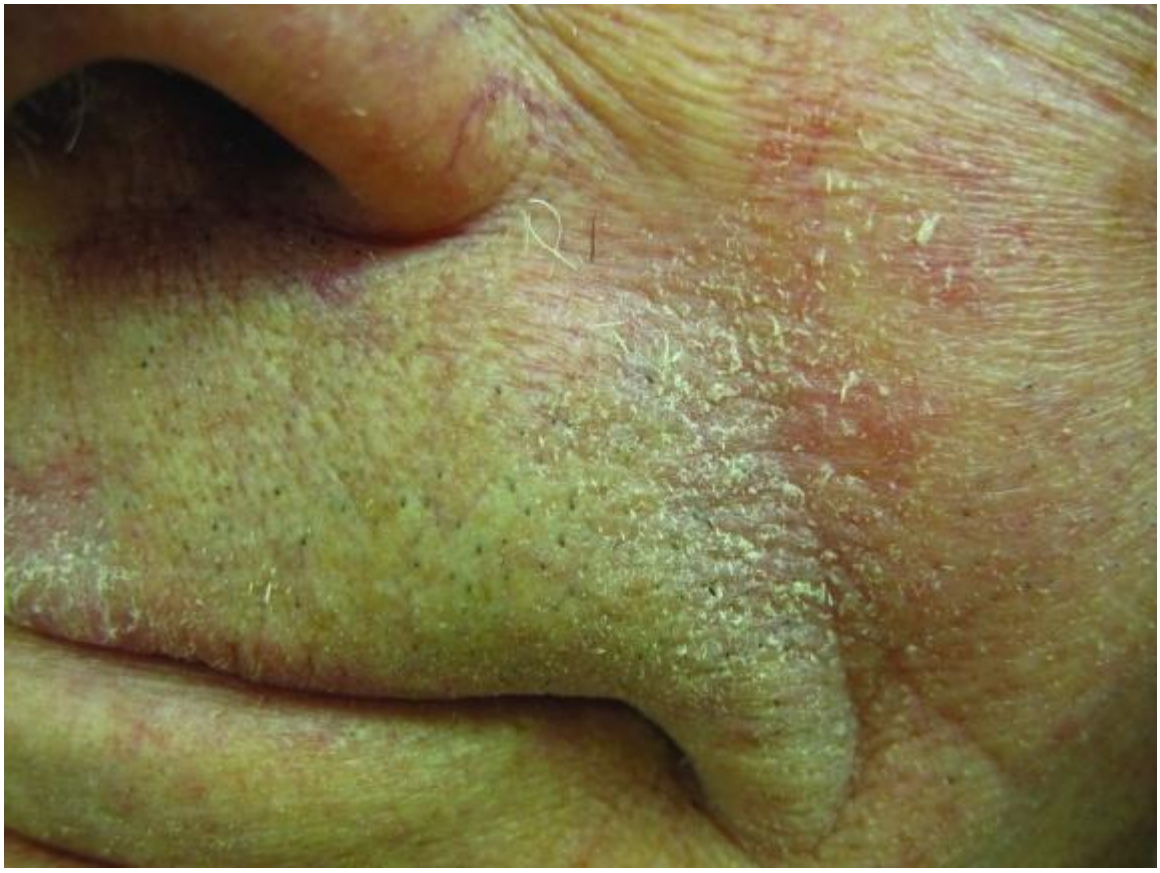

Figure 1 : Face seborrheic dermatitis

Melolabial fold seborrheic dermatitis, distinguished in a 68 year old Caucasian man by rose erythema and fine scaling.

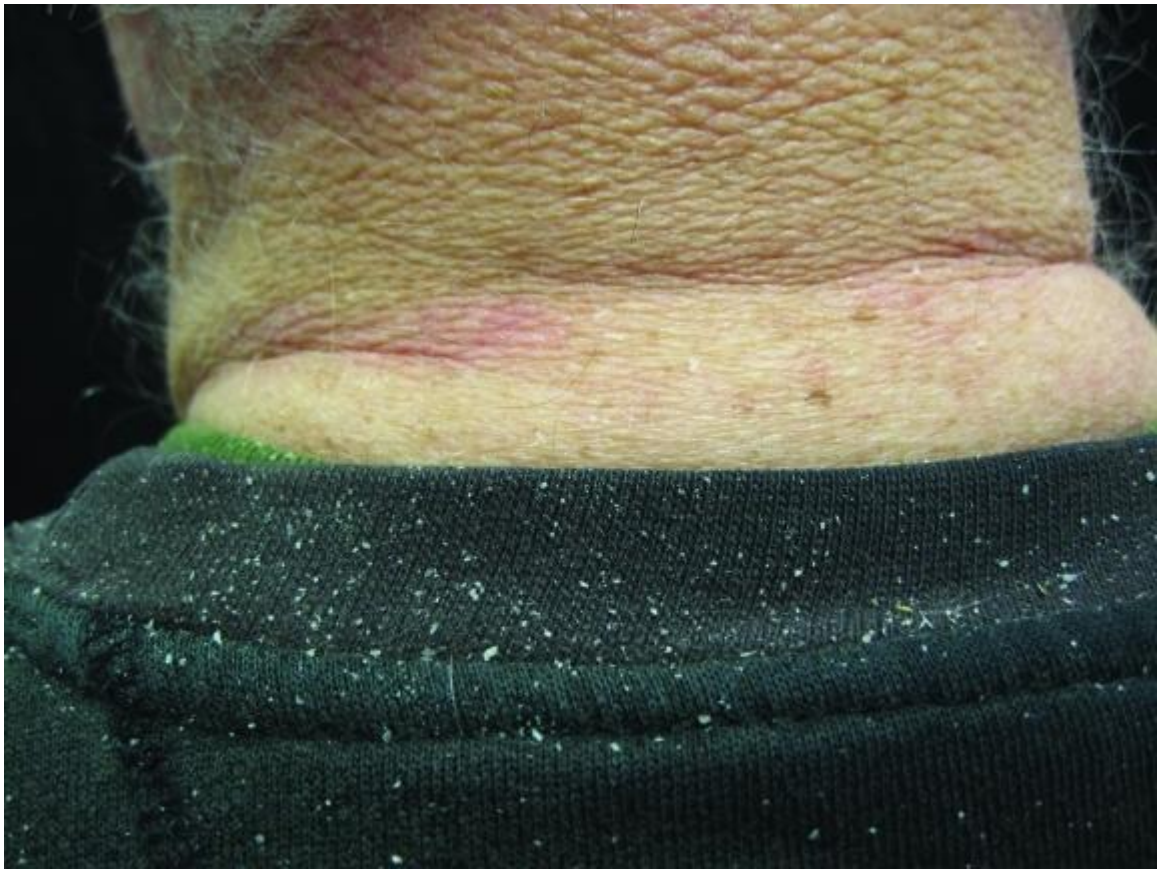

FIGURE 2 : seborrheic dermatitis at neck

Extreme skin scaling (dandruff) with continuous elimination of scalp particles, which are readily noticeable and dropped on dark clothes.

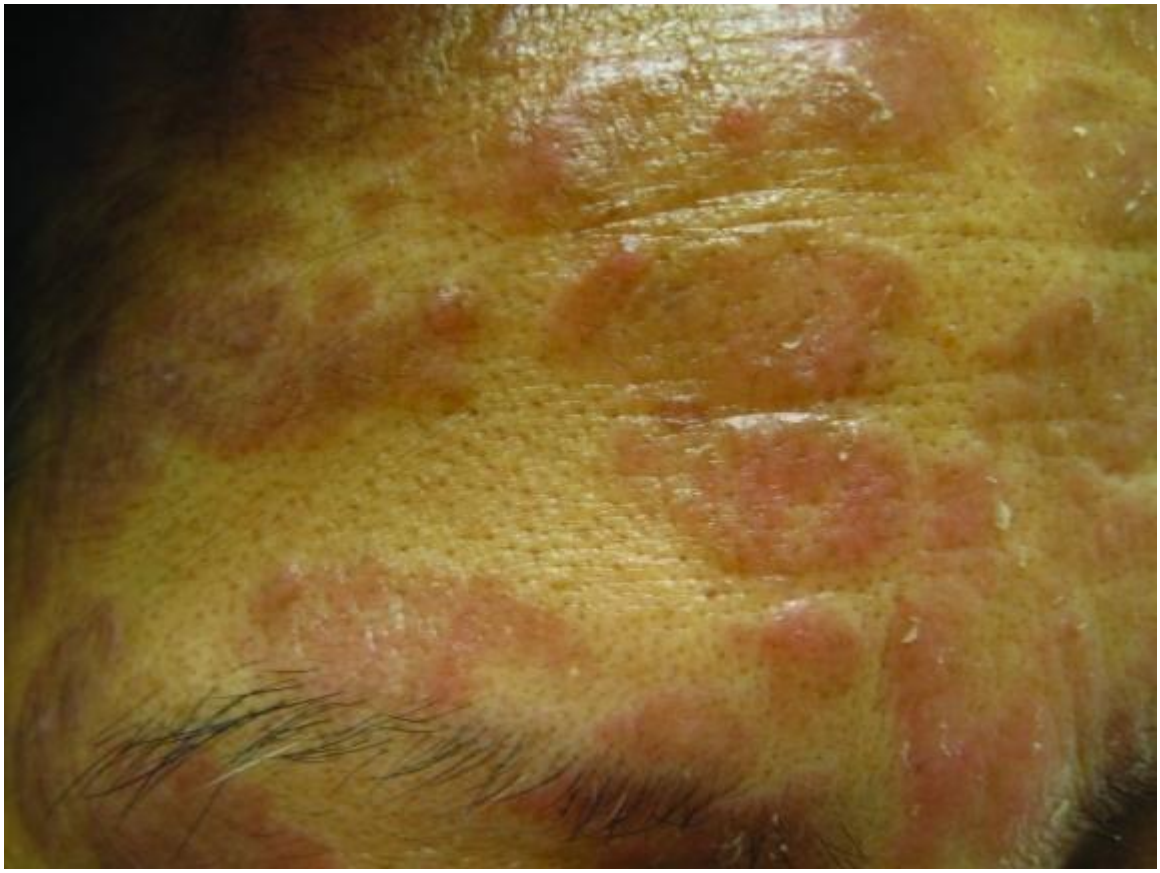

FIGURE 3: seborrheic dermatitis at forehead

Discoid, waxy, thin, scaly plaques on the face of the Filipino lady, 50 years old, with the aid of a seborrheic dermatitis

Differential Diagnosis:

Important Considerations in the Differential Diagnosis for Adult SD

Scalp

- Psoriasis - usually nonpruritic and tends to affect the occipital and frontal regions, whereas SD tends to affect the vertex and parietal regions
- Eczema (contact) - shampoo; hair dye
- Darier's disease - yellowish-brown clusters of rough dome-shaped papules in Seborrheic distribution; acanthosis
- Face: Psoriasis - rarely occurs in isolation; pitted nails
- Lupus erythematosus (LE) – discoid LE is associated with skin atrophy and scarring (alopecia)
- Rosacea – look for erythema and telangiectasia; it may cause Meibomianitis, along the posterior lid line
- Acne vulgaris – look for comedones, which are its hallmark
- Staphylococcal blepharitis (anterior lash line)
- Eczema (contact) - eyelids commonly involved (irritant - dry, scaly; or allergic - swollen, vesicular)
- Darier's disease- Nail changes

Trunk:

- Psoriasis - sharply-defined red plaques with a loose, silvery lamella scale
- Pityriasis rosea - herald spot; collarette scale; Christmas tree distribution
- Pityriasis versicolor - not symmetrical; hypo/hyperpigmentation
- Subacute lupus erythematosus - photosensitive distribution
- Eczema (nummular) - intense pruritus
- Tinea corporis - raised leading edges and central clearing; uncommon in infants
- Erythema annulare centrifugum - recurrent polycyclic lesions that slowly expand and disappear
- Darier disease - Greasy wart-like papules and plaques
- Grover disease (transient acantholytic dermatosis) - acanthosis

- Drug reaction - drug history (neuroleptic; immunosuppressant; PUVA; lithium)
- Parapsoriasis - elderly; very slow growing; resistant to treatment
- Pemphigus foliaceus - fragile, painful blisters - Nikolsky sign is positive
- • Primary syphilis – palm and single lesions; a history of chancre

Intertriginous Areas:

- Psoriasis (inverse) - sharply-defined border
- Dermatitis (Contact) - itchy; vesicular
- Tinea cruris - advancing border; very uncommon in infants
- Erythrasma - wood light coral-red fluorescence
- Candidiasis - satellite lesions; obesity; a history of immunodeficiency
- • Benign pemphigus population (Hailey-Hailey disease). - acanthosis

Important Considerations in the Differential Diagnosis for ISD

Cradle Cap

- Tinea capitis - (look for broken hairs or “black dots”); very uncommon in adults
- Impetigo - yellow, honey-colored crusting

Diaper Region:

- Tendency to avoid skin turns • Irritant touch dermatitis
- candidiasis – either secondary or from colonization with fecal yeast; look for satellite lesions

- Infantile psoriasis – sharply-defined red plaques with silver scale; most do not subsequently develop psoriasis
- Histiocytosis X (Langerhans cell histiocytosis) – tends to be confined to the skin folds with a purpuric rash on the body
- Acrodermatitis enteropathica – look for periorificial involvement and check zinc levels

Prognosis:

ISD typically has a moderate, minimal impact on the skin, whereas ASD has a persistent skin condition trend that is marked by relapses and remissions. ASD is extremely controlled but not curable.

31. ***Complications:***

Seborrheic dermatitis typically leads a mild path with very severe serious complications. Candida spp is especially valued in the intertriginal areas and eyelids for secondary bacterial disease particularly during the time of acute flares, with overcrowding in the diaper zone.

Erythroderma was identified with widespread SIDS in immunosuppressed neonates, but in adults with HIV-AIDS it is more commonly identified. Work has not therefore clearly founded, despite the preference of the SD for sebaceous-rich skin to be triggering erythrodermia per se, misdiagnosis is the most prevalent concern in both ISD and ASD.

Deterrence and Patient Education:

- Parental education is useful in allaying anxiety associated with ISD, and well-informed adults can learn to be confident at managing their condition
- For ASD, it requires emphasizing for the patient no remedy but can be managed well and managed primarily at home.[16]
- Many of the treatments for SD are available without a prescription, over the counter at the pharmacy, or increasingly on supermarket shelves. Directing the patient to the selection of such products may save consultation time and associated costs.

Enhancing Healthcare Team Outcomes:

SD is a difficult condition to manage in the adult, and its management is best by an interprofessional team.

- Consider the impact of SD on psychosocial functioning and quality-of-life and remember that SD may accompany neurological or psychiatric disease.[24]
- The dermatologist and pharmacist can help to promote the appropriate use of topical corticosteroids and employ steroid-sparing alternatives.
- New evidence suggests diet may play a role in SD, with a high fruit intake linked with a 25% reduced incidence of SD, relative to a 47% higher risk of SD in the West diet.
- atopic Dermatitis and ISD frequently coexist, whereas psoriasis often occurs in adults with SD, but there is usually no history of atopic dermatitis
- Review the diagnosis in recalcitrant cases of SD and consider immunodeficiency in sudden, severe cases
- Topical antifungals have an anti-inflammatory effect which contributes significantly to their efficacy and is a convenient steroid-sparing strategy

While primary clinicians treat this disorder, the complicated patient should be referred for advice by the dermatologist. Specialist dermatologists may also offer instruction in medicine and tracking and diagrammatics improvement by giving recommendations to the individual. Also, a pharmacist will provide aid in the collection of the most suitable drugs, checking of quantities, information for patients and conciliation of pharmaceutical items, and advise the prescriber of any issues. Near inter-professional team contact is critical to boost the results.

Other Treatments

I. Tar.

- The diagnosis of other dermatological disorders was traditionally Tar's diagnosis. Kaposi has shown its utility for seborrheic dermatitis in 1895.40 It is expected to use its methods of action to decrease the inflammatory reaction and to improve its antifungal properties.
- Tar was shown to be similar to ketoconazole for its fungistatic propensities,42 but there remain questions regarding its health profile. Studies have also shown that tar is capable of decreasing sebum output.
- Tar is widely used for superficial folliculitis, finger touch dermatitis, psoriasis aggravation in the individual, superficial Skin Atrophy, telangiectase, Pigmentation and keratoacanthoma exfoliative dermatitis. Kaposi has observed that the drug is poisonous to small children frequently afflicted by seborrheic dermatitis, including diarrhea, vomiting and tarry black urine..
- This can Also be related to an elevated likelihood of malignancy, in fact, squamous cell carcinoma43. Thus, tar needs to be used to manage seborrheic dermatitis with different issues.

II. Light Therapy.

- Phototherapy for severe seborrheic dermatitis was suggested as a viable remedy, although no randomised studies were conducted to prove its efficacy. Burning and burning symptoms, along with an elevated likelihood of malignancy following UV exposure, are raising adverse reactions with phototherapy.

Future therapies for SD could target improving skin barrier function or restoring the skin’s surface lipid composition.

Typical Formulary May Include:

i. Topical Creams, Ointments, and Lotions

- 2% salicylic acid + 2% sulfur in sorbolene cream or emulsifying ointment
- 2% ketoconazole cream

- 1% clotrimazole + 1% hydrocortisone cream
- 10% sulfacetamide + 5% sulfur lotion
- Betamethasone dipropionate 0.05% lotion
- 0.03% and 0.1% tacrolimus ointment

ii. Shampoos

- 1% zinc pyrithione
- 1% to .5% selenium sulfide
- 2% ketoconazole
- 1% ciclopirox
- 5% coal tar + 2% salicylic acid
- 0.1% and 0.03% tacrolimus

iii. Oral Medication

- Itraconazole
- Fluconazole
- Terbinafine

Summary

For those with seborrheic dermatitis (including topical selenium, zinc, ketoconazole, and cyclopirox), a variety of antivungal agents are beneficial for milder symptoms. Selenium and zinc are ideal because the presence of pathogens is mostly limited to the skin. Such drugs, such as topical corticosteroids or immunomodulators, are more successful in combination.45 Ciclopirox tends to be a safer option for more severe moderate diseases than other topical drugs. This is also simpler to use (as a shampoo, just two to three days a week) and it tends to have more effective anti-inflammatory properties than other antimicrobials45. It is often beneficial to provide a combined treatment, particularly as selenium to Zinc AND the introduction Of Topical Anti-fiction Agent.

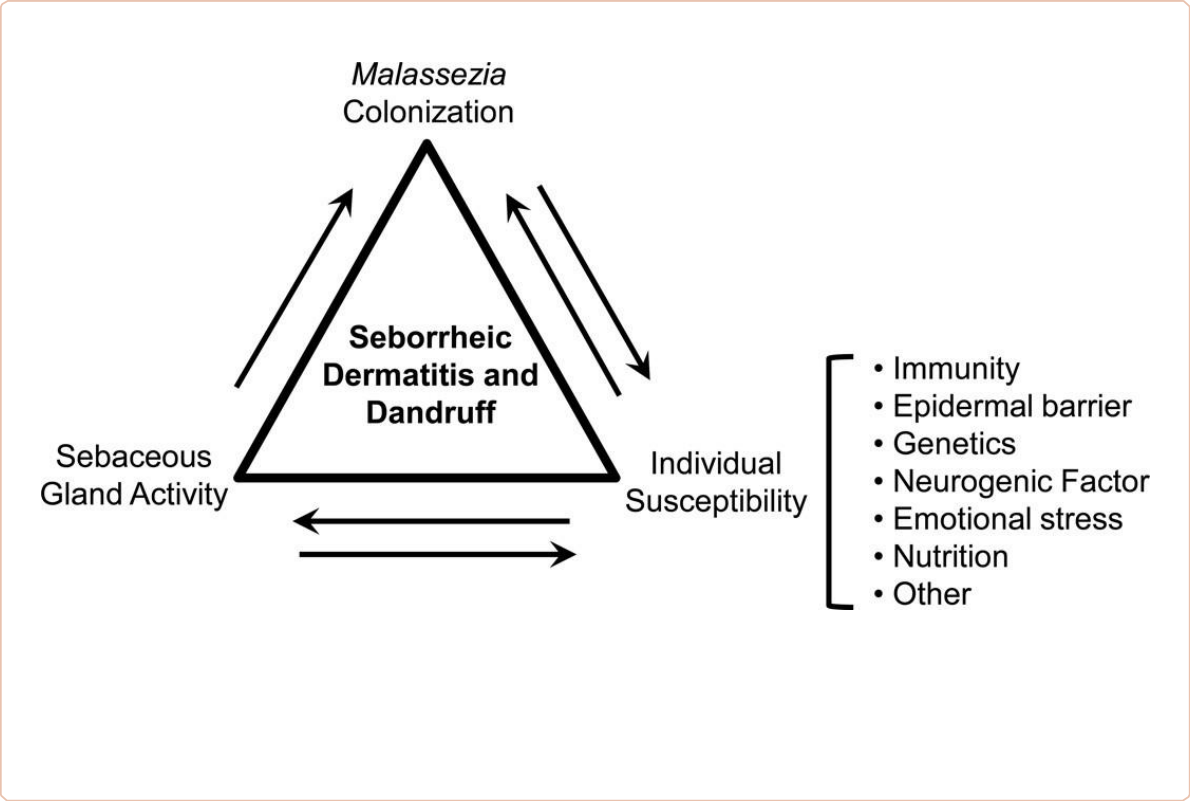

FIGURE 4: SUMMARY

Role of the Pharmacist

As a noticeable disorder, patients sometimes feel ashamed and disfigured as seborrheic dermatitis. The feedback of a pharmacist will significantly help patients. To check the extent of the conditions, the pharmacist will collect a background of treatment and then give guidance about the correct over-the-counter method. If required, your pharmacist can refer you to a specialist. The pharmacist will address medication goals, reasonable standards, length of care, the correct usage of drugs, how necessary it is to stick to the protocol and potential harmful effects.

The pharmacist will develop revised drugs databases, including medicinal products, nonprescription medications and dietary nutrients, for each individual in order to minimize the likelihood of medication reactions. The medications with a specific therapy index or products that may interfere with certain products will be checked by the pharmacist. Seborrheic dermatitis treatment may be of little benefit to individuals, so if visits with the dermatologist are required, a pharmacist may either locate or prescribe the correct treatment.

Regarding adverse events, medication complications, conformity and the utilization of their approved medications, pharmacists may be effective when treating patients.

Conclusions:

SD and dandruff are of the same condition as they tend to impact the body's regions of seborrhea (Table 4). They have several shared characteristics and respond to similar treatments. The pathogenesis may be affected by a number Intrinsically and biologically safe, such as Malassezia yeast, epidermal conditions feeding, sebaceous secretion, immune response and interconnections. Good SD and dandruff control includes removal of antifungal drug signs, enhancing underlying signs such as pruritus, overall Scalp or Skin quality To the better sustain Remission. studies of genetic or biochemical processes in humans and animal models can help define emerging targets for more effective diagnosis and improved care of fewer side-effects.

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