REVIEW ON THE DIAGNOSIS AND TREATMENT OF PSORIASIS DISEASE

Miss. Sharavani Bappasaheb Chaudhar, Sakshi Bhivsen Shinde, Vaibhav Arjun Ubale
Gajanan Maharaj Collage Of Pharmacy, chh.sambhajinager
shravani0503@gmail.com
Prof. Shubhangi Bhojgude Department Of Pharmaceutics
Dr.kavita kulkarni(PhD.M pharma), Department Of Quality Assurance**,**
Gajanan Maharaj Collage Of Pharmacy, chh.sambhajinager

ABSTRACT

Psoriasis is a common, chronic inflammatory skin disease affecting many people of the world now a days. Psoriasis is Principally an immunological T lymphocyte-driven disease, relating both the distinctive and T-cell-mediated immune Systems. The mostly affected sites comprise the scalp, extensor surfaces of the knees and elbows, umbilicus, genitalia, Anterior lower legs and nails. Psoriasis (PsO) is a common, systemic, chronic, inflammatory disease characterized by key clinical symptoms, including itching, pain, and scaling. PsO is associated with a high prevalence of comorbidities, including other autoimmune diseases and malignancies. Smoking, obesity, metabolic Syndrome, hypertension, dyslipidemia, diabetes And reduced physical activity are associated With psoriasis, increasing cardiovascular risk. Epidemiologically there are about 125million peoples affected around the world and 10million cases in India are observed annually. Basically many effective therapies are available to manage the symptoms of psoriasis which include topical, systemic, biological, natural herbs as well as phototherapy and combination therapy.

Psoriasis is a chronic inflammatory disease that is characterized by plaque, inverse, guttate, pustular, and erythrodermic variants. This review focuses on the epidemiology, diagnosis, and treatment of cutaneous psoriasis. Psoriasis is a rather common inflammatory skin disease that is characterized by the appearance of red scaly. Plaques and may affect any part of the body. There are certain factors that make psoriasis a challenge for physicians, these include: high Prevalence, disability, chronicity, disfiguration, and associated comorbidities. The approach to the management of Psoriatic patients should Also take into account the dermatological clinical features. In African American Population purplish coloured patches with grey scales on very dark skin have been reported. There is no Cure for psoriasis, but treatment can significantly reduce symptom, even in severe cases. A treatment Regime is curated by the physician keeping in mind the area of the rash, age, overall health and other Factors. Retinoid and steroids in the type of creams, ointments, gels etc. are prescribed. Various treatments are also available for patients with modest to severe psoriasis.

KEYWORDS:

Psoriasis, Investigation, Pustular, Stress ,Reduction, Lifestyle changes, diagnosis; general practice, primary care, Cardiovascular risk, Comorbidities, Biologic treatments, T-Lymphocytes, Treatment, Patient Counseling.

Introduction:

Psoriasis is a chronic inflammatory disease of the skin that affects almost 2% of the world's total population. It is Characterized by skin cells that multiply up to 10 times faster than normal. Psoriasis was first recognized and Described accurately by England's Dr. Robert Willan at around 1809, as a specific clinical entity Psoriasis is a complex disease

with lifelong emotional and social consequences for affected patients. It also lessens the Patients' quality of life and needs a long-term supervision. For this reason, self management strategies are essential in Addition to appropriate treatment of the disease to improve patient health and quality of life . It is a chronic skin disease that may develop at any age. The United States and Europe propose that psoriasis account For 4% of skin diseases in kids [1]. Psoriasis (PsO) is a chronic, inflammatory, Autoimmune disease affecting the skin, with an Estimated prevalence of 2–4% (7.4 million peoPle) in the USA. PsO can be difficult to Treat as it is frequently accompanied by Comorbidities that confound diagnosis and Complicate management. Secukinumab selectively targets interleukin (IL)-17A, a cytokine that is considered a key Player in the pathogenesis of PsO [3], and was The first and only fully human IL-17A inhibitor Approved for PsO in both the USA and Europe. The efficacy and safety profile of secukInumab has been studied for up to 5 years in PsO clinical trials. However, clinical trials Typically provide evidence from patients with Out certain comorbidities or active infections That may render them immunocompromised. In the real-world setting, clinicians may be Confronted with challenging scenarios, such as Prior malignancies and recurrent, chronic Infections, for which there are few data to rely. Upon for guidance. Here, we review the use of secukinumab in Patients with PsO who are often excluded from Clinical trials because of pregnancy, age (those 17 years or younger, and those 65 years or Older), manifestation of erythrodermic PsO, or Chronic illnesses, including latent tuberculosis Infection (LTBI), infection with hepatitis B and C virus (HBV and HCV, respectively) or HIV, Multiple sclerosis (MS), and malignancies. We conducted a literature search on secukInumab treatment in PsO populations of interEst. Individual searches were performed for Specific populations (search terms: ‘‘pregnan*,’’ ‘‘pediatric OR paediatric,’’ ‘‘special pop*,’’ ‘‘elderly,’’ ‘‘erythroderma’’) or patients with Comorbid chronic conditions (‘‘cancer OR Malign*,’’ ‘‘comorbid*,’’ ‘‘hepatic,’’ ‘‘hepatitis,’’ ‘‘[human immunodeficiency virus] OR HIV,’’ ‘‘multiple sclerosis,’’ ‘‘tuberculosis OR TB’’), Together with ‘‘secukinumab’’ and ‘‘psoriasis.’’The search was further refined by only selecting Articles published in English, and results with More than 45 citations were further restricted For ‘‘treatment’’ or ‘‘quality of life.’’ Articles of potential interest were reviewed after manual Curation (i.e., consolidation of duplicates, Removal of articles not directly relevant); addi Tional references found in these manuscripts Were also used. This article is based on previOusly conducted studies and does not contain Any studies with human participants or animals Performed by any of the authors.

SPECIAL POPULATIONS

Pregnancy

There is a need for long-term treatment of PsO In patients of childbearing age, but data Regarding efficacy and safety of biologics for PsO During pregnancy are limited to small retroSpective studies and case studies.

Because T helper 17 (Th17) cells are downregulated During pregnancy, and because PsO is a Th17 Cell immune-mediated disease, amelioration of PsO-related symptoms may occur during pregNancy. Twice as many patients with PsO Reported improvement in symptoms during Pregnancy than reported worsening . TraDitional treatments, including methotrexate And acitretin, are contraindicated in pregnancy, And cyclosporine should be limited to the lowest dose for the shortest duration possible. Animal studies indicated that secukInumab is not harmful to embryonic or fetal Development, parturition, or postnatal development, including immune response (Novarti Data on file). The manufacturer's global safety database, Which records maternal or paternal exposure to Secukinumab during pregnancy in all indications, reported 291 pregnancies between December 26, 2017, and December 25, 2018, in Which pregnancy outcomes were analyzed.Rates of spontaneous abortion (31 of 291;10.7%) were similar to that of an earlier Analysis (30 of 292; 10.3%) [15] and to rates in The general population (15–20% for women With a mean maternal age of 30.6 years). Among patients with known outcomes, the rate Of congenital abnormalities was similar to that In the general population. Because antibodies were expected to cross the placenta is The third trimester, most patients discontinued Secukinumab during the first trimester. One case study reported a 45-year-old Woman who developed amenorrhea after 3 months of secukinumab therapy. She had Tubal sterilization at age 43 and was treated with Other tumor necrosis factor inhibitors (TNFis) From age 40 to 44 prior to receiving secukInumab. Clinicians determined that she had a Miscarriage at 6 weeks of gestation. However, She previously had one spontaneous abortion, Was multiparous, and was 45 years old—each a Major risk factor unrelated to PsO; therefore, it Was inconclusive whether secukinumab was Related to the spontaneous abortion. Although risks to pregnancy are likely low, There are insufficient data with secukinumab Use in pregnant women to advise continuation Of secukinumab treatment during pregnancy. At least one group, the Australian Psoriasis Collaboration, has recommended discontinuation of treatment 19 weeks prior to intended Conception, enough time to achieve secukInumab washout, to avoid potential risk of teratogenicity, embryo toxicity, or any negative Effects on male fertility [20]. Of note, in a study Of patients who achieved 75% reduction in their Psoriasis Area Severity Index (PASI75) score after 1 year of treatment with secukinumab and then Relapsed after treatment discontinuation, 94% Of patients regained a PASI75 score by 16 weeks, Suggesting that secukinumab may be the optimal treatment for patients who discontinue Secukinumab during pregnancy [21]. Secukinumab is not recommended for use during Pregnancy unless the potential benefits of Treatment outweigh the potential risks.

Pediatric Population:

The prevalence of PsO is low (0.55%) in children aged less than 10 years. In patients aged More than 20 years, PsO prevalence increases With age more rapidly in women than in men; Differences between the sexes equilibrate after Age 20. Options for treatment of children with PsO are relatively limited. While etanercept is approved by both the US Food and Drug Administration (FDA) and European Medicines Agency (EMA) for children with PsO, adalimumab is approved by the EMA for children, And ustekinumab is approved by the FDA for Adolescents, most systemic treatments are used Off-label. Although secukinumab is in Ongoing development for the treatment of Children aged more than 6 years with PsO, Available evidence of the safety and efficacy of Secukinumab in children is currently limited to Case studies. Secukinumab was deemed safe and effective For the treatment of a rare, life-threatening Autoimmune condition characterized by deficiency of the IL-36 receptor antagonist (DITRA) In a 4-year-old boy refractory to methotrexate, TNFis, and IL-1 inhibitors [24]. The patient Presented with generalized pustular rash, fever, And elevated markers of inflammation and was Initially diagnosed with generalized pustular PsO. He received topical and systemic corticosteroids, methot turexate, and etanercept, leading To moderate improvement; however, by the Time DITRA was diagnosed within the next 4 months, the patient had experienced three Disease flares with fever and new pustular skin Eruptions. Despite treatment with anakinra at Doses as high as 6 mg/kg per day, he continued To experience fever with new skin eruptions; he Then started secukinumab 75 mg/week, which Led to rapid improvement of skin disease and Blood parameters. At last follow-up 2 months Later, while receiving secukinumab and pred nisolone 2.5 mg/day, the patient presented with Completely cleared skin, normalized blood Parameters, and normal ultrasound findings on The liver. The authors presented this case as an Example of the important benefit of an IL-17 Antagonist in a child with DITRA, with rapid And complete improvement of skin and systemic inflammation. Additionally, secukinumab treatment was Associated with remission in a 13-year-old boy With recalcitrant chronic erythrodermic Pso. The patient presented with PsO at age 6, Which progressed to erythroderma at age 9. He Was initially treated with methotrexate and Multiple cycles of cyclosporine, acitretin, and Phototherapy for 4 years, with minimal Improvement and recurrent flares. He started on Secukinumab after it became available in his Country; following his first injection of secukinumab 300 mg, his PASI score improved from 50 to 11.4, his Dermatology Life Quality Index Score improved from 27 to 8, and he experienced relief from his erythroderma. He achieved complete remission by 8 weeks with no adverse Effects and went on to complete 1 year of Monthly treatment with secukinumab. Currently, two phase 3 clinical trials evaluating the efficacy and safety of secukinumab for the treatment of PsO in a pediatric population (age 6–17 years) are ongoing (NCT03668613 And NCT02471144), with preliminary results Anticipated shortly. Given that children do not Endure injections well and that

secukinumab Has more injections than other biologics, such As IL-23 and IL-12/23 inhibitors, this may be Considered a disadvantage for secukinumab in Pediatric populations. However, while adalimumab, etanercept, and ixekizumab are associated with the highest incidences of injection Site reactions (ISRs) [26], in the FIXTURE study, Only 0.7% of patients receiving secukinumab Experienced ISRs compared with 11% of Patients receiving etanercept, suggesting that Secukinumab would be safe and advantageous For the treatment of pediatric patients. While insufficient safety data exist to make Specific treatment recommendations in pediatric populations, these trials will provide dermatologists with additional information in this population[2].

Elderly Population

Elderly patients (aged 65 years or more) with PsO are challenging to treat as this population Has a higher prevalence of comorbidities, which May lead to inadequate treatment responses And higher risk of adverse events . Although Elderly patients with moderate to severe PsO in Clinical trials had a higher baseline frequency of Cardiovascular and metabolic disorders, the Efficacy of secukinumab was comparable to that In a younger population at 52 weeks (percentage of patients experiencing PASI75, 81.8% vs 79.4%, respectively), with a safety profile consistent with that described in other studies of Biologics in elderly populations[2].

CURRENT THERAPEUTIC SCENARIO OF PSORIASIS:

Psoriasis treatment depends on its severity. Thus, adequate disease assessment plays a key Role. Currently, several tools are used in clinical Practice, also for special areas (e.g., nail Psoriasis). Globally, Psoriasis Area Severity Index (PASI) And body surface area (BSA) are the most used Tools in clinical practice and clinical trials. Specifically, the PASI score evaluates both lesion Severity and affected area, amalgamating them Into a singular score from 0 (absence of disease) To 72 (most severe), with the body divided into Four regions (head: 10%, arms: 20%, trunk: 30%, legs: 40%) and each area scored individually. These individual scores are then aggregated to yield the final PASI. Skin involvement for each region is assessed, graded from 0 (no Involvement) to 6 (90–100% involvement), Alongside severity measured via three clinical Indicators ranging from 0 (none) to 4 (maximum): erythema (redness), induration (thickness) and desquamation (scaling). The Cumulative severity parameter for each skin Section is calculated, factoring in the respective Area score and section weight (0.1 for head, 0.2 For arms, 0.3 for body and 0.4 for legs)[4].

MATERIAL AND METHODS:

Psoriasis Pathogenesis:

This section will provide a quick overview of the Immunopathogenesis and histological features of Psoriasis (Fig. 1). Both psoriasis and psoriatic Arthritis (PsA) are complicated hereditary Inherited diseases. Research on families, twins, Linkage, and population-based relationships Supports the genetic basis of psoriasis. These Studies demonstrate the significance of the Immune system and

keratinocytes in the Aetiology of psoriasis. It has been found that Psoriasis and psoriatic arthritis share a group Of unique CARD14 polymorphisms, and that CARD14 modulates NF-kB-mediated responses in The skinkawada. In order to gather data on a Connection between psoriasis and various Immune syst

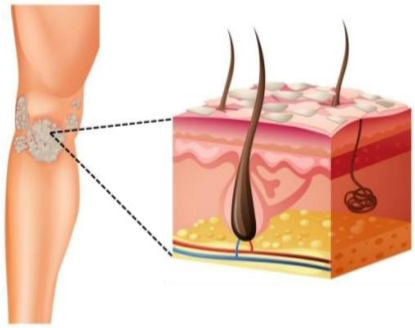


Fig. 1: Psoriasis.

em loci, Elder and his colleagues Assessed the SNP analyses of multiple significant studies in this field[11]

EPIDEMIOLOGY OF PSORIASIS:

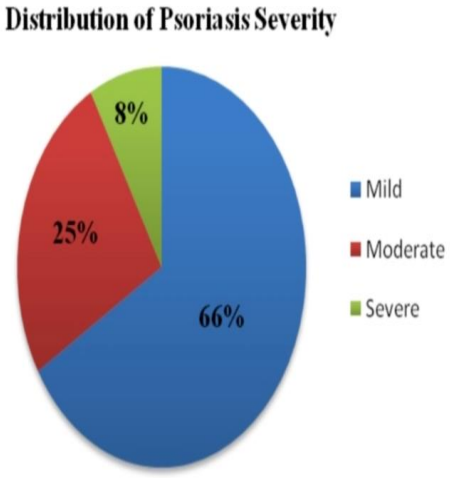
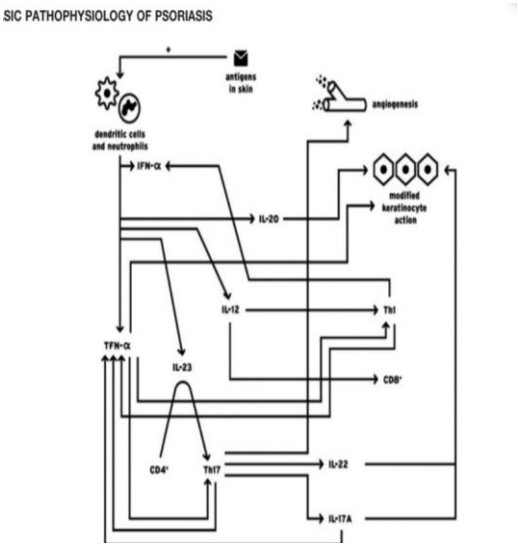


Figure: Psoriasis severity chart from 278 adult's patients [17].

In the survey of the International Federation of Psoriasis Associations (IFPA) there is almost 3% of the world's Population affected with some types of psoriasis and That's approx. 125 million people. When we studied for India, the range of more than 10 million cases of Psoriasis annually observed. That's why it comes under a Very common category and it is our moral duty to work More on the effective treatment and management of Psoriasis. According to research the occurrence of Psoriasis in the USA is around 2%. The increasing rates Of psoriasis have been investigated in peoples of Faroe Islands; the prevalence in the population is about 2.8% The occurrence of psoriasis is less in some ethnic groups Like Japanese and likely it could be absent in indigenous Australians and Indians from the origin of South America[5].

PATHOPHYSIOLOGY



Psoriasis is a chronic autoimmune disease with multiple leukocytes And cytokines interacting to produce the disease process (Figure 1). The inflammatory cascade of

psoriasis begins when antigens in The skin activate dendritic cells and neutrophils, which release Cytokines including tumor necrosis factor ! (TNF-!), interleukin 23 (IL-23), and IL-12. These cytokines participate in positive feedback loops by activating leukocytes, which then release more Cytokines, resulting in continuous inflammation. For example, IL-23 converts cluster of differentiation 4–positive cells into T-helper 17 (TH17) cells that release IL-17A; TH17 cells and IL-17A Act to upregulate TNF-!. These cytokines also exert effects on the Skin with IL-17A, IL-20, and IL-22, and TNF-! Contributing to The modified keratinocyte function and the TH17 cells promote Angiogenesis[7]. The pathophysiology involves hyper-proliferation Of epidermis, abnormal differentiation of Epidermal keratinocyte, inflammation and Alteration in the immune system due to multiple Factors. [12] An increase in DNA synthesis is the key Character of hyper-proliferation and a Significantly lower turnover rate for the Epidermis. In psoriasis, there is a lag in Appearance of keratins (1 and 10) that are Expressed in normal physiology of differentiating Skin. [13] Abnormal keratinocyte differentiations Involve increased expression of keratins such as Six and 16. Neutrophils in filtrate the epidermis And the layers above it. T-cells infiltrate the Dermis with a majority of CD8+ cells[7].

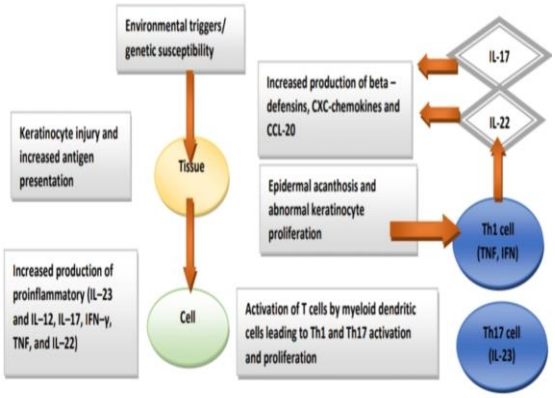


Figure 1: Pathophysiology of psoriasis"

1.1. Types of Psoriasis

There are different types of psoriasis and it's possible to have more than one type[1].

1.2. Chronic plaque Psoriasis

Among different types of psoriasis, the most common one is plaque psoriasis or psoriasis vulgaris. Almost 85 percent of People with psoriasis have plaque psoriasis which is characterized by thick red patches of skin, often with a silver or White flaking layer[1].

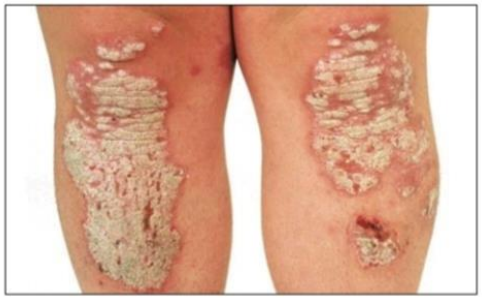


Figure 1 Plaque psoriasis

Guttate Psoriasis Streptococcal infection like pharyngitis or perianal infection classically triggered a distinct variant of psoriasis called Guttate psoriasis which is more common in kids and adolescents than

adults. In this case, patients severely present small Drop like lesions which respond well to topical treatments and phototherapies[1].

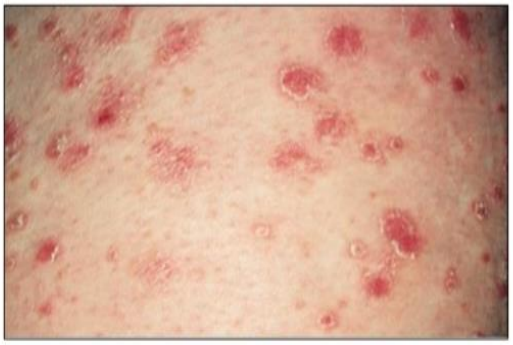


Figure 2 Guttate psoriasis

1.3. Flexural Psoriasis The quality of life of a psoriatic patient may be impaired considerably by facial and flexural psoriasis. This type Of psoriasis is an extrapolative marker indicating a poor prediction of psoriasis. Facial and flexural psoriasis cannot be Considered as dissimilar disease entities but rather as site differences[1].

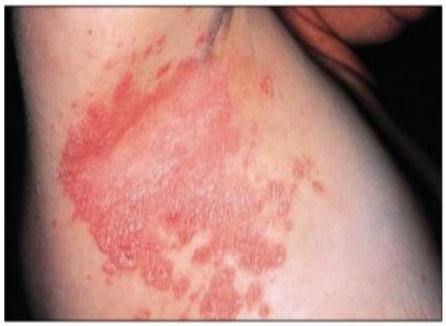


Figure 3 Flexural psoriasis

1.4. Erythrodermic Psoriasis

Erythroderma is a scaly erythematous dermatitis that involves 90% or more of the cutaneous surface. The most mutual Dermatoses underlying erythroderma are psoriasis and eczema. Erythroderma may be also caused by cutaneous TT Celllymphomas[1].

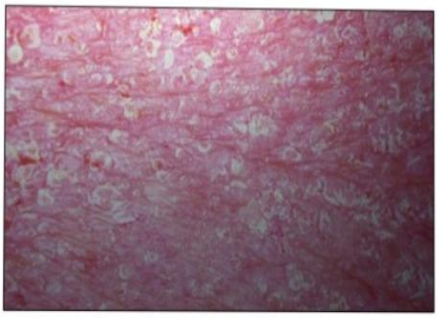


Figure 4 Erythroderma psoriasis

1.5. Pustular psoriasis

The patients who are suffering from pustular psoriasis or related pustular diseases may genetic abnormalities which Impair the function of crucial players of the innate skin immune system. Detection of these irregularities has changed The paradigm of these diseases recently[1]

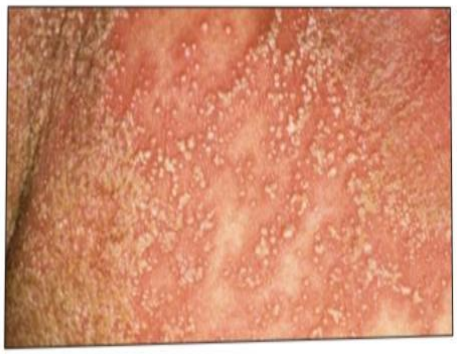


Figure 5 Pustular psoriasis

1.7 Palmoplantar Psoriasis

Plaque psoriasis that involves the palms and soles is characterized as palmoplantar psoriasis. This type of psoriasis is a Challenge for dermatologists that is difficult to be treated with topical and systemic

therapies[1].

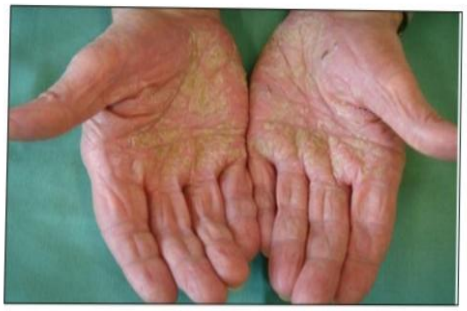


Figure 6 Palmoplantar psoriasis

1.8 Scalp Psoriasis

Scalp psoriasis can affect patients' lives harmfully and is often resistant to the treatment that is not been a major focus Of a scientific study. The activity of secukinumab of patient-reported outcomes of scalp psoriasis is evaluated by this Analysis[1].

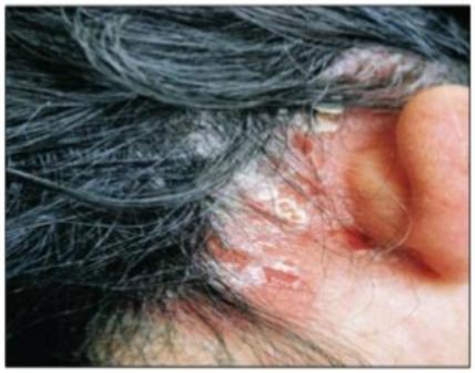


Figure 7 Scalp psoriasis

1.9. Nail Psoriasis

About 80% patients with psoriasis are likely to develop nail psoriasis as a result of the conditions of their nails as nails Are considered epidermal appendages. Psoriasis can cause nail disorders of two patterns[1].

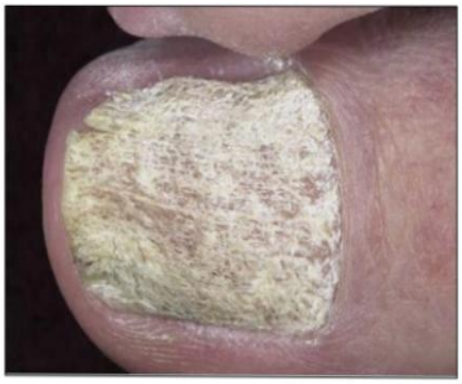


Figure 8 Nail psoriasis

1.10. Psoriatic Arthritis

An inflammatory rheumatic disorder of unknown etiology occurring in patients with psoriasis is named as psoriatic Arthritis. An authenticated set of classification criteria for psoriatic arthritis having specificity of 98.7% and sensitivity Of 91.4% and has recently established by The Classification Criteria for Psoriatic Arthritis group[1].

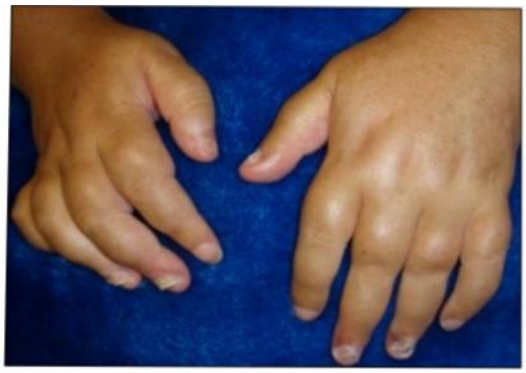


Figure 9 Psoriatic arthritis

Causes of Psoriasis:

The causes of psoriasis are yet not fully cleared, but There are many factors which are responsible for the Development of psoriasis.

These factors include genetics, Environment and the immune system[5].

Genetic factors:

In our body genes play an important role to control Everything in the body, from height to eye colour. The Normal function of cells in the body is controlled by Genes, so it is important to work genes normally. Around 10% of the normal population has predisposed the genes To psoriasis; but from 10% only 1-3% of the populations Develop psoriasis. Also families with some history of Psoriasis have been more likely to develop psoriasis[5].

Environmental factors:

Many environmental factors are generally causing Psoriasis. These factors activate genes to produce Psoriasis. Some of the responsible factors related to the Environment are established below[5].

Infections:

Streptococcal throat infection, tonsillitis on the throat And some other skin related infection may harm the skin And that can result in produce guttate (small, salmonPink droplets). The other major types of psoriasis can Develop after the time period of two of three weeks During the infection. Injury on skin, like cuts or scrapes Can also lead to developing more chances of psoriasis[5].

Stress:

Stress is very common and it can cause mental illness Which may affect the body to produce many products. The stress enhances the inflammatory response to our Skin. According to researchers it is the important factor For stress induced psoriasis[5].

Medication:

Ace-inhibitors, Beta-blockers, Lithium, Synthetic Antimalarial medications, Quinidine, Indomethacin, NSAIDS, Interferons etc. So, the group of these Medications can activate the disease condition[5]

Life style:

Our skin is a major part of the body and also it is the Major organ of our body, it works as a protective tool From external harms on the body. So, due to less Awareness regarding personal care in our daily life style Which include diet, exercise, sleep and obesity, air Pollution, more alcohol consumption, smoking. Consuming alcohol in small or extra amounts may Enhance the production chances of psoriasis and Continuously smoking can also make the disease worse[5].

Other factors: Cold weather, Obesity, Folate and vitamin B12 Deficiency[5].

Treatment and Management

The condition is slight and can be treated by creams in most cases. However, a low percentage of kids have moderate to Severe disease requiring drugs, such as ciclosporin or methotrexate, and few will need injections with newer biological Agents, such as anti-TNF (tumour necrosis factor) drugs. Anti-TNF drugs (among them etanercept, infliximab, and Adalimumab) are intended to decrease inflammation in the body produced by tumour necrosis factor. There is lacking Of evidence for the safety and efficacy of

these biological mediators in paediatric psoriasis. Systemic treatments, including methotrexate and cyclosporin, are associated with potential hepatotoxicity, due to either Direct liver damage or suppression of immunity or both immunomodulated and a direct liver damage; therefore, Treatment of patients with psoriasis poses a therapeutic challenge. Psoriasis treatments reduce inflammation and clear the skin. Treatments can be divided into three main types: topical Treatments, light therapy and systemic medications.

Topical psoriasis treatments include:

Topical corticosteroids, vitamin d analogues, anthralin, topical retinoids, calcineurin inhibitors, salicylic acid and coal Tar. Oral or injected medications include retinoids, methotrexate and cyclosporine. Medications like Thioguanine (Tabloid) and hydroxyurea (Droxia, Hydrea) can be used if other drugs can't be given Psoriasis management may involve systemic and topical medication, phototherapy, stress reduction, climatotherapy, And various aides such as moisturizers, sunlight, salicylic acid, and other keratolytics like urea. Taking daily baths, using moisturizer, exposure of skin to small amounts of sunlight, avoiding psoriasis triggers and Drinking alcohol are some steps to manage psoriasis[1].

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Table No.2: List of Topical agents used in the Treatment of Psoriasis.

Drug Name	Formulations	Dose
Corticosteroids:		
Clobetasol propionate	Ointment, Spray, foam, Lotion, Shampoo.	Lotion, Spray, shampoo, 0.05%, Ointment 0.05% applied on affected area.
Halobetasol propionate	Ointment	
Betamethasone	Cream, Gel, Lotion, Foam	
Mometasone	Cream, Ointment, Gel.	
Vitamin D3 analogues:		Apply a thin layer of formulation (0.005%) on affected skin twice daily.
Calcipotriol	Ointment, Cream, Solution.	
Calcitriol	Ointment	
Tacalcitol	Ointment	
Retinoids:		0.1% of cream is applied on affected skin and for vulgaris cleans the skin gently.
Tazarotene	Gel, Cream and Foam.	
Coal Tar	Ointment, Gel, Solution, Shampoo, Soap.	Use with the combination of other drugs twice a day.
Calcineurin inhibitors:		Apply a thin layer of 0.03% cream or ointment on affected skin twice a day.
Tacrolimus	Ointment	
Pimecrolimus	Cream	
Dithranol:		(0.1-1%) is applied once a day and washed off carefully after 10 minutes to one hour.
Anthralin	Cream, Shampoo, Gel.	

CONCLUSION:

It is clear that psoriasis has a major impact on an individual's psychological well-being and consequently affects their Quality of life. Proper counseling and management can improve the mental condition of a psoriatic patient[1]. The management of PsO is frequently compli Cated among special populations of patients, Including those with comorbid chronic ill-Nesses, elderly patients, and pregnant women, Fully recognizing that comorbid conditions are Common in patients with moderate-to-severe PsO. Treatment of these patients, as well as Those with the rare and potentially fatal erythrodermic form of PsO, requires careful consideration and frequent monitoring to achieve Optimal management.

REFERENCE:

1. *Myers WA, Gottlieb AB, Mease P. Psoriasis and Psoriatic arthritis: clinical features and*

disease Mechanisms. Clin Dermatol. 2006;24(5):438–47.

2. *Lonnberg AS, Zachariae C, Skov L. Targeting of Interleukin-17 in the treatment of psoriasis. Clin Cosmet Investig Dermatol. 2014;7:251–9.*
3. *Blauvelt A, Prinz JC, Gottlieb AB, et al. Secukinumab administration by pre-filled syringe: efficacy, safety and usability results from a randomized Controlled trial in psoriasis (FEATURE). Br J Dermatol. 2015;172(2):484–93.*
4. *Bissonnette R, Luger T, Thaci D, et al. Secukinumab Demonstrates high sustained efficacy and a favourable safety profile in patients with moderate-to-Severe psoriasis through 5 years of treatment (SCULPTURE Extension Study). J Eur Acad Dermatol Venereol. 2018;32(9):1507–14.*
5. *Strober BE, Clay Cather J, Cohen D, et al. A Delphi Consensus approach to challenging case scenarios. In moderate-to-severe psoriasis: part 2. Dermatol Ther (Heidelb). 2012;2(1):2-012-0002-x.*
6. *Porter ML, Lockwood SJ, Kimball AB. Update on Biologic safety for patients with psoriasis during Pregnancy. Int J Womens Dermatol. 2017;3(1): 21–5.*
7. *Kaushik SB, Lebwohl MG. Psoriasis: which therapy For which patient: focus on special populations and Chronic infections. J Am Acad Dermatol. 2018;80(1):43–53.*
8. *Warren RB, Reich K, Langley RG, et al. Secukinumab in pregnancy: outcomes in psoriasis, psoriatic arthritis and ankylosing spondylitis from the Global safety database. Br J Dermatol. 2018;179(5): 1205–7.*
9. *. Nybo Andersen AM, Wohlfahrt J, Christens P, Olsen J, Melbye M. Maternal age and fetal lose population based register linkage study. BMJ. 2000;320(7251):1708.12*
10. *Gelfand JM, Weinstein R, Porter SB, Neimann AL, Berlin JA, Margolis DJ. Prevalence and treatment of Psoriasis in the United Kingdom: a population Based study. Arch Dermatol. 2005;141(12):1537–41.*
11. *Henderson Berg MH, Carrasco D. Injection site Reactions to biologic agents used in psoriasis and Psoriatic arthritis. J Drugs Dermatol. 2017;16(7):695–8.*
12. *Korber A, Papavassilis C, Bhosekar V, Reinhardt M.Efficacy and safety of secukinumab in elderly subjects with moderate to severe plaque psoriasis: a Pooled analysis of phase III studies. Drugs Aging. 2018;35(2):135–44.*
13. *Ferna´ndez-Torres RM, Paradela S, Fonseca E. Long Term response to etanercept monotherapy in moderate to severe psoriasis: assessment in daily practice By the maintenance of low values of PASI and BSA.J Dermatol Treat. 2014;25(1):54–6. https://doi.org/10.3109/09546634.2012.755254.*
14. *Finlay AY, Khan GK. Dermatology Life Quality Index (DLQI)—a simple practical measure for routine clinical use. Clin Exp Dermatol. 1994;19(3):210–6. https://doi.org/10.1111/j.1365-2230.1994. Tb01167.x.*
15. *Nast A, Smith C, Spuls PI, et al. EuroGuiDerm Guideline on the systemic treatment of Psoriasis Vulgaris—part 1: treatment and monitoring recommendations. J Eur Acad Dermatol Venereol. 2020;34(11):2461–98. https://doi.org/10.1111/jdv.16915.*
16. *Megna M, Camela E, Battista T, et al. Efficacy and Safety of biologics and small molecules for psoriasis In pediatric and geriatric populations Part II: focus On elderly patients. Expert Opin Drug Saf. 2023;22:1–16 (Published online February).*
17. *Martora F, Fabbrocini G, Marasca C. Pityriasis rosea After Moderna mRNA-1273 vaccine: a case series. Dermatol Ther. 2022;35(2): e15225. https://doi.org/10.1111/dth.15225.*
18. *Camela E, Potestio L, Fabbrocini G, Pallotta S,Megna M. The holistic approach to psoriasis Patients with comorbidities: the role of investigational drugs. Expert Opin Investig*

Drugs.2023.https://doi.org/10.1080/13543784.2023.2219387.

19. *Armstrong AW and Read C. (2020) Pathophysiology, Clinical presentation, and treatment of psoriasis: A Review. JAMA 323(19): 1945-1960.*
20. *Sanjay kumar Rout1*, Bankim Chandra Tripathy2 and Vikash Ranjan Kar3. Natural Green Alternatives to Psoriasis Treatment- A Review. Glob J Pharmaceut Sci. 2017; 4(1): 555631.*
21. *Ladizinski B, Lee KC, Wilmer E, Alavi A, Mistry N, Sibbald RG. A review of the clinical Variants and the management of psoriasis. Adv Skin Wound Care 2013;26(6):271-84.*
22. *Katherine Brind'Amour. Palque Psoriasis: Symptoms, Treatments and Complications. Healthline. 2019.Available from https://www.healthline.com/health/plaque-psoriasis-pictures*
23. *[7] Van de Kerkhof PC, Murphy GM, Austad J, Ljungberg A, Cambazard F, Duvold LB. Psoriasis of the face and flexures, Journal of Dermatological Treatment. 2007; 18(6): 351-60.*
24. *[8] Zattra E, Belloni Fortina A, Peserico A, Alaibac M. Erythroderma in the era of biological therapies. European Journal of Dermatology. Mar-Apr 2012; 22(2): 167-71.*
25. *[9] Bachelez H. Pustular psoriasis and related pustular skin diseases. British Journal of Dermatology. 15 Jan 2018;178(3):614-618.*
26. *[10] Rocamora V, Garcías-Ladaria J. Complete response of secukinumab in palmoplantar psoriasis, Dermatology Online Journal. 15 Oct 2017; 23(10): PMID: 29469799.*
27. *[11] Feldman SR, Green L, Kimball AB, Siu K, Zhao Y, Herrera V, Nyirady J, Alexis AF. Secukinumab improves scalp Pain, itching, scaling and quality of life in patients with moderate-to-severe scalp psoriasis, Journal of Dermatological Treatment. Dec 2017; 28(8): 716-721.*
28. *[12] Karen Regina Rosso Schons, Cristiane Faccin Knob, Nádia Murussi, André Avelino Costa Beber, Walter Neumaier Odirlei André Monticielo. Nail psoriasis: a review of the literature, Anais Brasileioros De Dermatologia. Mar-Apr 2014; 89(2): 312–317.*
29. *Cantini F, Niccoli L, Nannini C, Kaloudi O, Bertoni M, Cassarà E. Psoriatic arthritis: a systematic review, Int J Rheum Dis. Oct 2010; 13(4): 300-17.*
30. *[14] Stephanie S. Gardner and MD.WebMD. Psoriasis. 2021.Available from https://www.webmd.com/skin-problems-and-treatments/psoriasis/understanding-psoriasis-Basics.*
31. *Krueger G, Ellis CN. Psoriasis- recent advances in understanding Its pathogenesis and treatment. Journal of the American Academy Of Dermatology. 2005 Jul 31; 53(1): S94-100.7.*
32. *Bowcock AM and Krueger JG. Getting under the skin: The Immunogenetics of psoriasis. Nature 2005; 5: 699-711.*
33. *Nickoloff BJ and Nestle FO. Recent insights into the Immunopathogenesis of psoriasis provide new therapeutic Opportunities J. Clin Invest 2004; 113: 1664-75.*
34. *Fiore M, Leone S, Maraolo AE, Berti E, Damiani G. Liver Illness and Psoriatic Patients. Biomed Research International. February 2013; 2018(3): 1-12.*