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Psoriasis Beyond the Skin : An Interdisciplinary Review of Pathophysiology and Treatment

Vaishnavi Vijay Jambhulkar, Payal Subhash Ghode, Sunil Baban Rathod, Kuldeep P. Waidya
Sahakar Maharshi Kisanrao Varal Patil College of Pharmacy Nighoj, Maharashtra , India
jambhulkarvaishnavi5@gmail.com , payalghode11@gmail.com , sunilrathod5335@gmail.com

Abstract: *Psoriasis is widely recognized as a chronic inflammatory skin disorder characterized by keratinocyte hyperproliferation and immune dysfunction. However, emerging evidence emphasizes its systemic nature, with effects on multiple organ systems and contributions to a variety of comorbidities. This interdisciplinary review delves into the evolving understanding of psoriasis beyond its dermatologic manifestations, focusing on its complex pathophysiological mechanisms, which include immune pathways, metabolic disturbances, and cardiovascular risk factors. We discuss the most recent advances in the diagnosis of psoriatic arthritis, metabolic syndrome, renal and hepatic involvement, and neuropsychiatric conditions associated with psoriasis. The review also assesses new and existing therapeutic approaches that try to change disease processes systemically, highlighting the significance of a multidisciplinary approach for the best possible care. The need for integrated care models to enhance patient outcomes and quality of life is highlighted by the recognition of psoriasis as a multisystem disorder.*

Keywords: Autoimmunity, Cytokines, Interleukin, Keratinocytes, NF-κB signaling pathway, Psoriasis, STAT signaling pathways, TNF

I. INTRODUCTION

Psoriasis is a chronic autoimmune skin disease that manifests as epider-mal hyperplasia and dermal inflammation, which can range from mild to disabling. It is a condition that has significant genetic associations and it tends to wax and wane, with flare-ups that can be triggered by a num-ber of environmental factors including stress and skin trauma. There are several forms of psoriasis, with the most common form being plaque psoriasis. Plaque psoriasis is characterized by the presence of sharply demarcated, thick, erythematous plaques that are usually covered by dry silvery white scales . The plaques range in size from 1 square centimeter to several square centimeters. In mild-to-moderate cases these plaques cover less than 5% of the body surface area, but in more severe cases, they can cover more than 20% of the body. Therapies may target inflammation and the abnormal immune response, as well as epidermal hyperproliferation.

Psoriasis is an unpredictable chronic inflammatory disease with remis-sions and exacerbations that often require prolonged therapy, Although plaque psoriasis is not life-threatening but with constant relapses it can significantly affect the quality of life. Patients with psoriasis may present with psychological manifestations due to poor esthetics and require spe cal attention and counseling. Stress, family history, and early onset of symptoms are some of the poor prognostic factors. Both warm weather and sunlight improve the symptoms. Therefore, treatment must be tailored according to the age of the patient, quality of life issues, and the long-term side effects of therapy particularly with topical steroids and other immuno-suppressive agents. Treatment can be categorized broadly as topical applications, phototherapy, systemic drugs, and other modalities. Topical therapy and phototherapy are used in mild-to-moderate psoriasis, and Systemic therapy with methotrexate or cyclosporine is indicated for more severe disease. Biologics should be used by qualified experts only when indicated. Topical therapy for scalp psoriasis includes the use of emollients and moisturizers, corticosteroids, keratolytics, tar, anthralin, vitamin D3 analogs, and calicineurin inhibitors. Sometimes, a combination of topical agents is more effective than monotherapy,

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Severe psoriasis requires systemic drugs such as acitretin, methotres ate, cyclosporine, tacrolimus, hydroxyurea, 6-thioguanise, mycophenolste fumaric acid esten, apramilast, and biologic agents. Among the available biologic agents, etanercept, adalimumab, infliximab, secukinumab, uste numab, tirakizumab, and wekizumab are are indicated for plaque psoriasis Etanercept is approved to be used in children with proper monitoring. Many studies have suggested that omega-3 fatty acids may improve vari ous signs and symptoms of psoriasis by reducing symptoms, and limiting the spreading of the inflarnimatory process.

Types of psoriasis :

Psoriasis comes in a variety of forms, each with unique features (Fig. 1). Plaque psoriasis is the most prevalent and is distinguished by elevated, inflammatory skin lesions covered in thick, silvery scales. Small, drop-like lesions dispersed across the body are the first signs of guttate psoriasis. Skin folds, such as those in the groin and armpits, are impacted by inverse psoriasis . The appearance of pus-filled blisters distinguishes pustular psoriasis from erythrodermic psoriasis, which is characterized by widespread inflammation and scale shedding. Pitting, discoloration, and detachment from the nail bed are just a few of the changes that nail psoriasis can make to the texture and look of the nails, for those who suffer from this ailment, knowing the various forms of psoriasis aids in precise diagnosis and effective treatment planning.

1. Plaque psoriasis : About 80–90% of instances of psoriasis fall into this category, making it the most prevalent kind. Raised, scaly, red patches of skin that are frequently covered with scales of silver give it away. The elbows, scalp, knees, and lower back are the areas of plaque psoriasis that are most typically affected. Plaque psoriasis is the most common type of psoriasis and has been extensively studied . The following are some key findings on this type of psoriasis: Plaque psoriasis has a significant genetic component, as evidenced by the identification of many genes associated with its onset. For example, a study discovered a substantial correlation between plaque psoriasis and a specific genetic variant in the CARD14 gene. Furthermore, the illness stems from an overactive immune system that produces inflammatory molecules known as cytokines. According to one study, cytokines including interleukin-17 and interleukin-23 were found to be more prevalent in patients with plaque psoriasis than in healthy controls. Additionally, several comorbidities, such as metabolic syndrome, psoriatic arthritis, and cardiovascular disease, are frequently present in conjunction with plaque psoriasis. Studies have indicated that compared to healthy people, persons with plaque psoriasis are more likely to develop metabolic syndrome.

2. Guttate psoriasis : This psoriasis appears in kids, teenagers and adults after a bacterial illness like strep throat. Small, scaly, red spots on the skin that can arise unexpectedly and spread quickly across the body are its defining features. Guttate psoriasis is strongly linked to streptococcal infections, which are far more common in people with the condition than in healthy controls . Research has shown that some genes, including a variant in the IL23R gene, raise the likelihood of developing guttate psoriasis, even if the exact genetic basis of the disorder is still unclear . According to 41% of patients, stress is the main cause of guttate psoriasis, with other triggers including streptococcal infections, skin traumas, and some drugs.

3. Inverse psoriasis : Inverse psoriasis primarily affects skin folds such as the groin, armpits, and areas beneath the breasts. It is characterized by red, smooth, shiny areas that are often inflamed by friction and perspiration. According to study, the most frequently affected locations are the groin (60%), axilla (40%), inframammary folds (25%), and intergluteal cleft (15%) . Inverse psoriasis can be challenging to diagnose since it resembles other skin conditions such fungal infections and intertrigo; studies show that the average diagnostic delay for this condition is 4.4 years . Metabolic syndrome is more common in affected people than in healthy people, and it is frequently associated with comorbid conditions including diabetes, obesity, and metabolic syndrome .

4. Pustular psoriasis : This is an uncommon variety of psoriasis that manifests as tiny, pus-filled blisters on the skin. Generalized pustular psoriasis (GPP), which can affect the entire body, and localized pustular psoriasis (LPP), which

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affects particular body parts like the palms and soles, are the two forms of pustular psoriasis . With a prevalence of 0.1% to 0.5% in the general population, pustular psoriasis is extremely rare, accounting for less than 5% of all cases. LPP is more localized, with tiny blister patches on particular body areas, whereas GPP is characterized by painful blisters, fever, and chills . Streptococcal infections are commonly cited as a cause, and other common triggers include stress, infections, and several drugs.

5. Erythrodermic psoriasis : Erythrodermic psoriasis is a rare but extreme form of psoriasis that can occur in individuals with pre-existing psoriasis or can be the first presentation of psoriasis. It is characterized by widespread red, inflamed skin that sheds scales in sheets. The skin may also be itchy, painful, and tender to the touch. Other symptoms can include fever, chills, rapid heartbeat, and fluid and electrolyte imbalances. It is a medical emergency that requires prompt diagnosis and treatment to prevent complications.

6. Nail psoriasis : The nails are impacted by this type of psoriasis, which makes them thick, discolored, and pitted. The nails may potentially separate from the nail bed in extreme circumstances. It affects the nails, causing changes in their appearance and texture. It can occur in individuals with other types of psoriasis or as an isolated condition. The most common nail changes seen in nail psoriasis include pitting, ridging, discoloration, and thickening. In severe cases, the nails may become deformed and detached from the nail bed, a condition known as onycholysis.

Sr.No.	Type	Clinical features
1	Plaque psoriasis	Red, scaly patches of skin that can be itchy or painful
2	Guttate psoriasis	Scaly, small, teardrop-shaped, red or pink spots on the skin.
3	Inverse psoriasis	Smooth, red, and shiny patches of skin.
4	Pustular psoriasis	Pus-filled blisters that appear on the skin.
5	Erythrodermic psoriasis	Red, itchy, and painful skin
6	Nail psoriasis	Nails are thick , discoloured and pitted

Table : Symptoms and clinical features of different types of psoriasis.

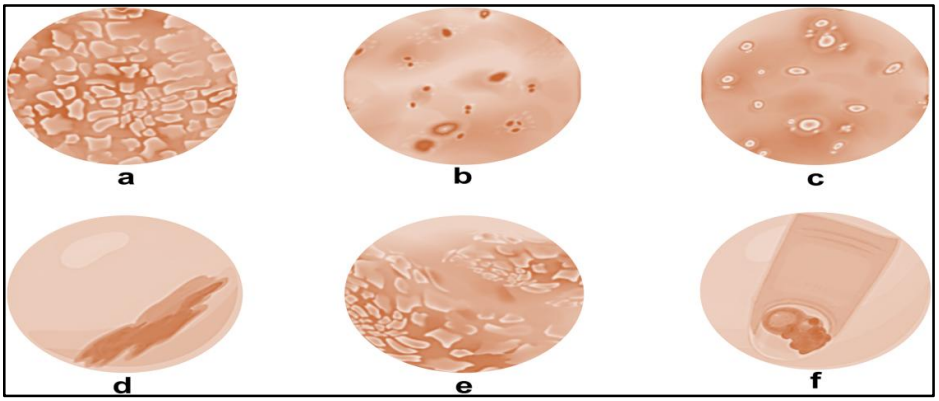

Fig 1. Types of psoriatic skin conditions . (a) Plaque psoriasis ; (b) Guttate psoriasis ; (c) Pustular psoriasis ; (d) Inverse psoriasis ; (e) Erythrodermic psoriasis (f) Nail psoriasis

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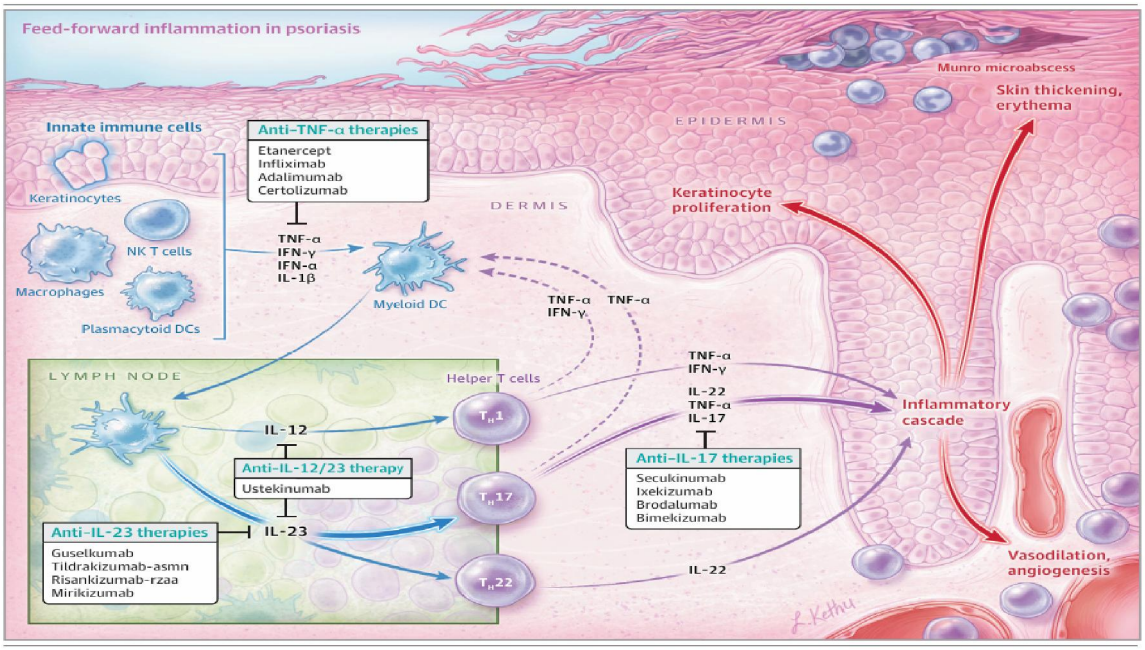
Pathogenesis of Psoriasis

The pathogenesis of psoriasis is complex and not fully elucidated.

Excessive activation of parts of the adaptive immune system is thought to be central to the pathogenesis of psoriasis. In the initial steps of psoriasis pathogenesis, a variety of cell types, including plasmacytoid dendritic cells, keratinocytes, natural killer T cells, and macrophages, secrete cytokines that activatemyeloid dendritic cells. For example, DNA-LL37 complexes stimulate plasmacytoid dendritic cells to secrete interferon alfa (IFN-α) which, in turn,activates myeloid dendritic cells. Once activated, myeloid dendritic cells secrete IL-12 and IL-23. IL-12 induces differentiation of native T cells to TH1 cells. IL-23 is central to the survival and prolifera-tion of TH17and TH22 cells. TH1 cells secrete interferon gamma (IFN-γ)and TNF-α; TH22 cells secreteIL-22; and TH17 cells secreteIL-17,IL-22,and TNF-α.11 Among these pathways, IL-23–mediated activation ofthe TH17 pathway is thought to be predominant.11 IL-23 signaling is mediated intracellularly via Tyk2-Jak2 and STAT3,which leads to transcription of key inflammatory mediators. These cytokines lead to downstream keratinocyte proliferation, increased expression of angiogenic mediators and endothelial adhesion molecules, and infiltration of immune cells into lesional skin.



Sustained inflammation that results in unchecked keratinocyte proliferation and defective differentiation is the defining feature of psoriasis. The inflammatory infiltrates of the psoriatic plaque are formed of dermal dendritic cells, macrophages, T cells, and neutrophils. They are overlaid by acanthosis (epidermolysis), according to the histology of the condition . The inflammatory mechanisms involved in plaque psoriasis and the other clinical variants are similar, but they also show distinct distinctions that are responsible for the variations in phenotype and therapeutic response . Psoriasis pathogenesis is a complex process with several interrelated components. In afflicted skin regions, it starts with the aberrant behavior of keratinocytes, which causes fast proliferation and improper differentiation . Cytokines and their receptors impact this process by initiating complex signaling pathways including MAPK, STAT, and NF-κB, which intensify the inflammatory response. In addition, the significance of the microbiome has become more and more clear, with dysbiosis playing a part in the imbalance. Psoriatic lesions persist because of pro-inflammatory conditions that are created by microbial changes, immune system disruptions and disruptions in cell signaling pathways. The chronic inflammation and aberrant skin cell proliferation associated with psoriasis are caused by the ongoing interaction of keratinocytes, cytokine signaling, and the microbiota. This highlights the disease's complexity and the requirement for focused therapy approaches.

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The role of keratinocytes in psoriasis pathogenesis : Both the initial stages of psoriasis and its ongoing maintenance depend heavily on keratinocytes. Keratinocytes have a variety of stimuli they might react to as part of the innate immune system. The actions of keratinocytes are modulated, and psoriasis is influenced by a variety of variables including genetics, cytokines and receptors, metabolism, cell signaling, transcription factors, non-coding RNAs, antimicrobial peptides, etc. Self-nucleotides and antimicrobial peptides are released by stressed keratinocytes, which aid in pDC activation. Then, IFN-a, IFN-γ, TNF-a, and IL-1β are produced by the activated and matured mDCs . In addition to taking part in the initiation phase, keratinocytes also contribute to the maintenance phase by amplifying psoriatic inflammation. Once activated by proinflammatory cytokines synergistically, keratinocytes are highly proliferative. They can produce copious chemokines (e.g., CXCL1/2/3, CXCL8, CXCL9/10/11, CCL2, and CCL20) to recruit leukocytes (such as neutrophils, Th17 cells, dendritic cells, and macrophages), antimicrobial peptides (e.g., S100A7/8/9/12, hBD2, and LL37) to induce innate immunity, and other inflammatory mediators to amplify inflammation. Additionally, keratinocytes, f ibroblasts, and endothelial cells cause tissue reorganization by promoting the growth and activation of endothelial cells as well as the deposition of extracellular matrix. Keratinocyte hyperproliferation and aberrant differentiation, dilated and hyperplastic blood vessels, and infiltration of inflammatory cells such as leukocytes are all consequences of the interaction between keratinocytes and immune cells, particularly Th17 cells, which leads to the production and maintenance of psoriasis.

The role of cytokines and their receptors in the pathogenesis of psoriasis : The development of psoriasis depends on cytokines and their receptors, which allow immune cells and keratinocytes to communicate. TNF-a, IFN-γ, IL-23/IL-17A, and IL-22 are immune-derived chemicals that stimulate keratinocytes, which set off several signaling pathways. Antimicrobial proteins, cytokines, chemokines, and growth factors are released due to aberrant keratinocyte development. To treat psoriasis, therapies targeting TNF-a, IL-17A, and IL-23 specifically work very well . Researchers' focus has recently increased on cytokines produced by keratinocytes or receptors expressed in those cells . Psoriasis is thought to be primarily caused by the IL-23/IL-17 cytokine axis. Immune cells are thought to need to produce IL-23 to maintain and grow immune cells capable of generating IL-17. However, keratinocytes can also generate IL-23, however it is unknown if this IL-23 plays a part in psoriasis. Scientists recently showed, using a transgenic mouse model, that keratinocyte-derived IL-23 was sufficient to activate IL-17-producing immune cells, induce them to release IL-17, and result in a persistent skin inflammatory response. Further research revealed that IL-23 expression in keratinocytes was controlled by epigenetic regulation through H3K9 dimethylation, which may contribute to psoriasis.

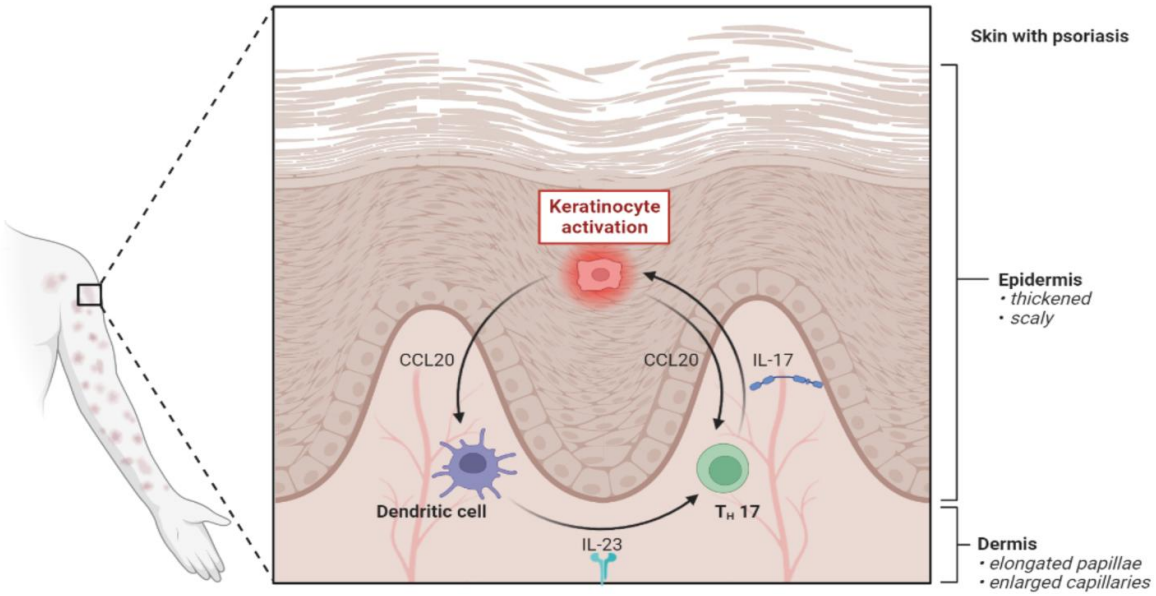

Fig 2. Pathogenesis of psoriasis

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Another important cytokine generated mostly by CD4+ T cells and group 3 innate lymphoid cells (ILC3) after IL-23 is IL-22. Non-hematopoietic cells including keratinocytes, epithelial cells, and hepatocytes express its receptor, known as IL-22R. By preventing keratinocytes from differentiating to their final state, IL-22 promotes the development of psoriasis and promotes the production of proinflammatory chemokines and antimicrobial peptides. IL-22 binding protein (IL-22BP) is a naturally occurring IL-22 inhibitor that binds specifically to IL-22, preventing it from performing its biological role. elevated levels of epidermal thickening and elevated production of inflammatory cytokines and IL-22-inducible antimicrobial peptides were seen in IMQ-induced psoriasis-like skin condition, which was aggravated by both hereditary IL-22BP deficiency and anti-IL-22BP neutralizing antibody.

TNF-like weak inducer of apoptosis (TWEAK), a crucial cytokine in psoriasis, has recently come to light. TWEAK deficiency reduced psoriatic dermatitis brought on by IMQ. Furthermore, animals lacking fibroblast growth factor-inducible 14 (Fn14, the TWEAK receptor) also had less severe illness. Scientists have most recently shown that keratinocytes play a crucial role in TWEAK's function in psoriasis utilizing a mouse model with keratinocyte-specific deletion of Fn14. Mice were shielded against IMQ-induced inflammation and psoriasiform hyperplasia by Fn14 loss in keratinocytes. It is significant to note that inhibiting TWEAK had a comparable effect on reducing epidermal thickness, skin infiltrates, and inflammatory mediators as inhibiting TNF-a and IL-17A.

The pathogenic process of psoriasis is mostly shown in this image from the viewpoint of keratinocytes. Initial stimuli can excite keratinocytesand stressed keratinocytes produce self nucleotides and antimicrobial peptides, activate pDCs and later DCs, and participate in psoriasis beginning phase. Activated keratinocytes impact the pathogenesis of psoriasis after being stimulated by cytokines in ways such as inflammatory infiltration, epidermal hyperplasia, innate immunity, tissue reorganization, etc.

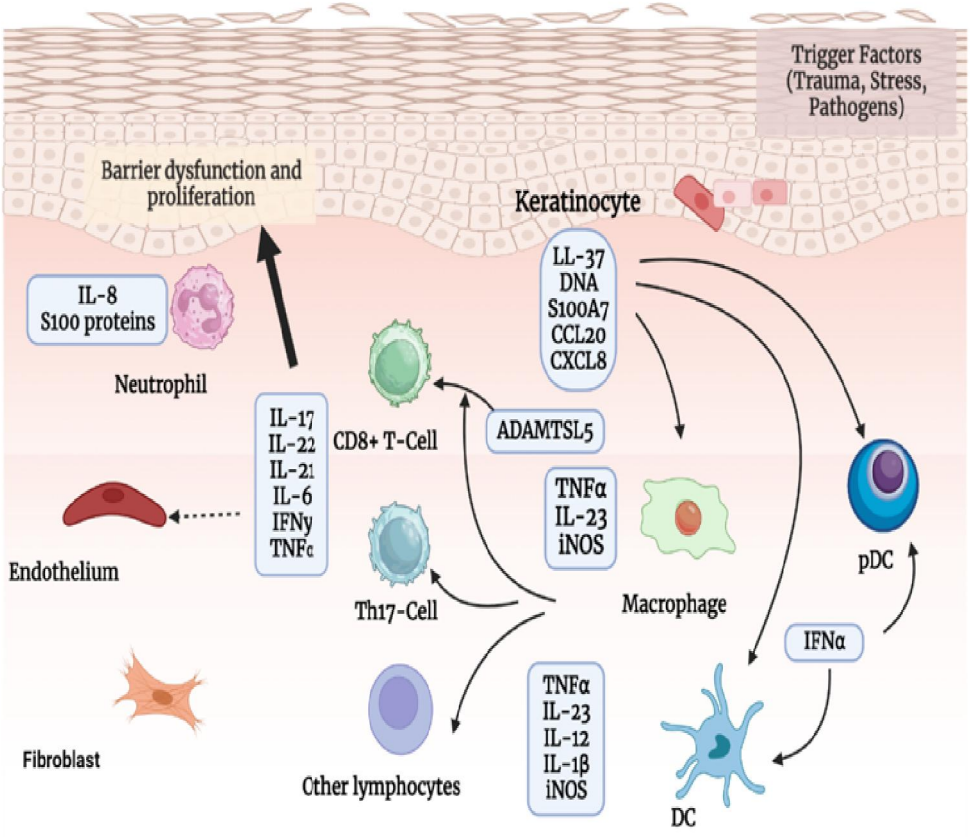

Fig 3. Pathogenic process of psoriasis

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Clinical Application : Therapy for Psoriasis

Plaque psoriasis can cause significant social morbidity, with patients potentially experiencing problems with work, activities of italy living, and socialization. Additionally, patients may teet unattractive and become depressed . Consequently, overall quality of life can suffer. Accordingly, in addition to a reduction or elimination of plaques and scales, and a reduction in flane-ups as important therapeutic outcomes, so is an improvement in quality-of-life scores. Among the Therapeutic strategies for prorizas care are monotherapy (which can limit side effects and improve adherencel, combination therapy (which can often be more effective and allow for lower dosing than monotherapy), and sequential therapy where stronger and sometimes more toxic agents are used extially to rapidly clear the lesions, followed by toxic agents for maintenance therapy) in addition to pharmacotherapy, nonpharmacologic approaches are valuable adjuncts to psoriasis care. These include managing sitestyle factors that may trigger exacerbations (for example, stress. smoking, obesity: Imiting alcohol consumption, and minimizing potential triggers to lesion formation scratching. piercings, tattoos, suntium, chemical irritants). Additionally, gentle cleansing, moisturization, sun protection, diet control, and engaging in physical activity can be valuable nonpharmacologie approaches for psoriasis care.

Drugs for Psoriasis

A. Topical Drugs :

Glucocorticoids are highly effective in mild, moderate and severe cases of psoriasis. The usefulness of topical corticosteroids is due to anti-inflammatory activity. The antimitotic effects of corticosteroids may account for an additional mechanism of action. Gradual response is seen over 3 weeks.

Keratolytics: Salicylic acid, urea, formalin and lactic acid can be used in combination with other drugs.

B. Systemic Drugs (Oral) :

1) Antimetabolites: Methotrexate and azathioprine are antiproliferative drugs which inhibit replica-tion and function of T cells. These are effective in moderate to severe psoriasis. Side effects: Myelosuppression and increase liver enzymes.

i. Immunosuppressants. Tacrolimus is an orally ef-fective drug in severe psoriasis. Cyclosporine in-hibits IL-2 production. Mycophenolate Mofetil is used (1-2 g/day) in severe psoriasis.

Agents for psoriasis

A. Apremilast : Apremilast [a-PRE-mi-last] is an oral agent approved for moderate-to-severe plaque psoriasis. It works by inhibiting phospho-diesterase-4, which ultimately leads to reduced production of sev-eral inflammatory mediators in psoriasis. The most common adverse effects are diarrhea, nausea, and headache. Depression may also occur. Strong CYP450 inducers (for example, carbamazepine and phenytoin) may reduce the efficacy of apremilast, and coadministration is not recommended.

B. Anthralin (Dithranol) : Anthralin is an anthracene derivative that has been reported to be the most effective topical treatment of stable plaque psoriasis. It has been reported to restore call differentiation by mitochondrial dys function, preventing the activation of T-cells, decreasing keratinocyte proliferation. Overnight therapy with 1% is indicated for the topical use. Short contact anthralin therapy (SCAT) is adopted (20 minutes to 1 hour before removal for the treatment of scaly plaques of psoriasis on the body or the scalp which are not responding to other treatments.

C. Coal tar : Coal tar is a thick, black lid obtained while distilling buminous vox It is reported to contain phenolic compounds such as bentene Naphthalene, phenols, and aniline On exposure to light, causes photoexcitation by UV-A 320 to 380 nm) and retards hyperpolitans Son of keratinocytes by suppressing DNA synthesis, it is convention ly acollied in an alcoholic solution containing 1% to 156 coal tar with salicylic acid on the poonatic plaques. A preperation containing crude cool tar is also frequently used as part of an inpatient or daily dressing regimen. It is used for ponosis of scalopaimoplantar. and chronic plaque. This therapy is expected to improve pooriasis 1 month after the initiation of the treatment and remission is also longer nan with any other agent. Common side effects include strong odor. severe skin irritation, stinging folliculitis, and formation of keratoac arthomas.

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D. Salicylic acid : Topical salicylic acid (2% to 6%) in a well-recognized keratolytic agent and has been used for psoriasis for many years. A combination com taining steroid with salicylic acid is used as a first line of treatment on thick, scaly plaques in palm, scalp, trunk, and soles but must not be used on eyes, genitals, and mucous membrane, It can also be combined with topical calcineurin inhibitors. Salicylic acid therapy tor psoriasis suffers from a major problem of a potential chronic or scute systemic intoxication, called "salicylism," which has symptoms of nausea, vomiting, tinnitus, metabolic acidosis, oral mucosa bum ing, etc. Therefore, it should be used more than 20% of the body surface area.

E. Retinoids : Retinoids normalize keratinocyte differentiation and reduce hyper proliferation and infiammation. Tazarotene is a topical retinoid used for the treatment of plaque psoriasis. Adverse effects are similar to ther topical retinoids described for acne. Acitretin [a-si-THE-tin second-generation retinoid used orally in the treatment of pustular toms of psoriasis. It is administered in the dose of 0.5 to 0.75 mg/kg.

G. Topical corticosteroids :Topical corticosteroids have been a mainstay of psoriasis therapy for over 50 years and are used in variety of other skin conditions as wel. The available agents differ in potericies and are formulated in a variety of dosage forms, including solutions, lotions, creams, ointments, gets, and shampoo (Figure 48.9. Upon binding to intracellular corticosteroid recep tors, these agents produce numerous effects that can be beneficial for psoriasis, including anti-inflammatory, antiproliferative, immunosuppres sive, and vasoconstrictive effects. Potential adverse effects, especially with the long-term use of potent corticosteroids, include skin atrophy, striae, acneiform eruptions, dermatitis, local infections, and hypopigmen tation. In children, excessive use of potent agents applied to a large sur face area can cause systemic toxicity, including possible depression of the hypothalamic-pituitary-adrenal axis and growth retardation.

Group 1 (Mild ;	Group 2 (Moderately potent ;	Group 3 (Potent ;	Group 4 (Super potent ;
Low Strength	Intermediate Strength	High Strength	Very High Strength
Alclometasone dipropionate 0.05% (c,o) Clocortolone pivalate 0.1% (c) Flucinolone acetonide 0.01% solution (s) Hydrocortisone base or acetate 0.25% to 2.5% (c,o) Triamcilonone acetonide 0.025% (c,o)	Betamethasone dipropionate 0.05% (c) Desonide 0.05% (c,o) Desoximetasone 0.05% Fluocinolone acetonide 0.025% (c,o) Flurandrenolide 0.025 to 0.5% (c, o) Fluticasone propionate 0.005% to 0.05% (c,o) Hydrocortisone	Amcinonide 0.1% (c,o) Betamethasone dipropionate, augmented 0.05% (c) Desoximetasone 0.05% (g) Diflorasone diacetate 0.05% (o,c) Fluocinonide 0.05% (c,g,o,s) Halcinonide 0.1% (c,o) Triamcinolone	Betamethasone dipropionate 0.05% (o,g) Clobetasol propionate 0.05% (c,g,o) Diflorasone diacetate 0.05% (o) Fluocinonide 0.1% (c) Flurandrenolide 0.1% (c) Halobetasol 0.05% (c,o)

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	butyrate 0.1% (c,o,s) Hydrocortisone valerate 0.2% (c,o) Mometasone furoate 0.1% (c,o) Triamcinolone acetonide 0.1% to 0.2% (c,o)	acetonide 0.5% (c,o)	

Table . Potency of various topical corticosteroids . c = cream ; g = gel ; o = ointment ; s = solution

II. CONCLUSION

The article emphasizes the critical role of the General Practitioner (GP) in diagnosis, severity assessment, and directing patients to appropriate specialist investigations. It stresses that due to its chronic nature, multi-system involvement, and associated comorbidities, psoriasis requires a multidisciplinary approach. This aligns with the holistic view of General Medicine, aiming to enhance treatment effectiveness and optimize patient care. Psoriasis is a chronic autoimmune skin condition that causes red, scaly areas that cause pain, discomfort, and social stigmatization. It affects 2 to 3 percent of people worldwide. Numerous varieties of psoriasis, including nail, plaque, guttate, inverse, pustular, and the underlying cause of erythrodermis, is the consequence of the interplay between immunological, environmental, and genetic variables. The clinical presentation is the primary basis for diagnosis; imaging, blood testing, and biopsy are also used. Psoriasis development is significantly regulated by keratinocytes, which are in turn impacted by Genetic factors , cytokines , metabolism and cell signalling .

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