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Psoriasis: An Overview on Management

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Abstract: A chronic systemic inflammatory disorder, psoriasis is characterised by distinctive skin lesions as well as a number of comorbidities. The manifestations of psoriasis are variegated namely psoriasis vulgaris, guttate psoriasis, pustular psoriasis, inverse psoriasis and palmoplantar psoriasis. The generation of inflammatory mediators and enhanced keratinocyte proliferation are two distinguishing characteristics. Topical, systemic, phototherapy, and biologics are some of the different therapeutic modalities. For mild to moderate psoriasis disorders, topical medicines are favored over systemic therapies, which are optimal in severe disease situations. Immunosuppressants and biological substances are a part of systemic therapy. Furthermore, symptomatic alleviation has also been achieved through the use of phototherapy. This review will summarize general knowledge about psoriasis and some potential treatments to cure/management of psoriasis.

Keywords: psoriasis, immunosuppressant, biologics, topical therapy, inflammation, photo therapy

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1. Introduction

Psoriasis is severe disease in which 3-2.2% of people worldwide suffer from the multifaceted multifactorial, which is triggered by a variety of genetic. environmental, and immunological factors. The clinical signs include symmetrical, well-defined erythematous plaques with adhering silvery scale. The scalp, elbows, knees, and presacral region of the back are typical afflicted sites [1]. Manifestations of psoriasis are varied: Psoriasis vulgaris, pustular psoriasis, guttate psoriasis, and inverse psoriasis are four different types of the skin condition (pus-filled, yellowish, small blisters). Palmoplantar psoriasis is the term used to describe the condition when it affects the palms and soles [2].

1.1 Psoriasis Vulgaris

Psoriasis vulgaris, which accounts for about 90% of cases, is the most common clinical variant of psoriasis. In the clinical setting, it manifests as erythematous plaques with distinct borders and iridescent squamae. The most common locations for lesions are the knees, elbows, scalp, and sacral area. Lesions have a symmetrical distribution. There may be a traumatic event that predisposes people to these lesions [3].

1.2 Guttate Psoriasis

Typically affects children and adolescents. It can develop independently (acute guttate psoriasis) or it can aggravate pre-existing, chronic plaque psoriasis, which is frequently relatively limited (guttate flare of chronic plaque psoriasis). It commonly manifests one and a half weeks after an episode of acute tonsillitis and is closely related with prior or ongoing streptococcal infection, evidence of which may be discovered in the majority of affected patients1. If neglected, guttate psoriasis may go away on its own or it may progress into chronic plaque psoriasis [4].

1.3 Pustular Psoriasis

Generalized pustular psoriasis (GPP) is a severe, multisystemic condition characterised by the abrupt, extensive eruption of superficial, sterile pustules. The systemic consequences of GPP have the potential to be life-threatening especially in older people, and they may or may not be preceded by a history of plaque psoriasis. The majority of GPP instances affect adults, while it can also affect kids and babies [5].

1.4 Inverse Psoriasis

Clinically, inverse psoriasis (IP) is characterised by well defined, erythematous patches. The inguinal folds, axillae, inframammary folds, perianal area, umbilicus, and retroauricular areas are the most often affected body parts. Interdigital spaces, popliteal and antecubital fossae, and these structures may also be involved. Involvement of the external genitalia is seen as a component of IP presentation. IP could signify the psoriasis's sole localization or, more typically, it could be accompanied with classical plaque lesions that are



localised in other body parts. Contrary to plaque psoriasis, the lesions surfaces tend to be moist, smooth, and shiny, and whitish scales are often minimal or absent [6]. Young newborns may frequently show signs of IP as clearly defined, hardly raised erythematous plaques in the diaper area, with the inguinal folds frequently being involved (napkin psoriasis) [7]. Itching, irritation from sweating, and soreness are typically observed as a result of frequent superficial erosions and maceration [8]. Superinfection by bacteria and fungi is common in IP because the wet skin creates a suitable habitat for their growth [9]. On the other hand, colonisation of flexural regions may potentially make people more susceptible to IP flares [6].

1.5 Palmoplantar Psoriasis

It mainly affects the skin on palms and soles. It has pustular, hyperkeratotic, mixed or morphologies. A possibly related dermatosis known for its small, sterile pustules palmoplantar pustulosis or pustular palmoplantar psoriasis—could be a subtype of palmoplantar psoriasis or a separate entity. Both conditions are chronic in nature and cause serious functional impairment [10].

2. Multi-morbidity and Psoriasis

Multi-morbidity, which is the existence of two or more chronic conditions, is typical in people with psoriasis. Up to 30% of people with psoriasis develop psoriatic arthritis (PsA), which is more frequent in people with nail dystrophy and scalp/intergluteal/perianal psoriasis [11].

Psoriasis patients as compared to the general population are more likely to have obesity, cardiovascular disease, non-alcoholic fatty liver disease, diabetes, and metabolic syndrome, with rates being notably high in individuals with more severe psoriasis [12]. This could be a result of common risk factors, inflammatory pathways, and genetic characteristics [13]. Furthermore, psoriasis has a negative psychological impact since incidence of mental health issues (such as anxiety and depression) are higher than in the general population [14].

3. Pathophysiology

Psoriasis development is mostly explained by two theories. The first theory regards psoriasis as being largely a disorder of excessive skin cell proliferation and reproduction, in which psoriasis is a symptom of a problem with the epidermis and Whereas according to the its keratinocytes. second theory, the condition is an immunemediated condition in which the immune system's effects on the skin's abnormal cell growth are a consequence. T cells (which normally aid in the body's defence against infection) become active, go to the dermis, and cause the release of cytokines, particularly tumor necrosis factor-alpha (TNF), which promotes inflammation and the rapid synthesis of skin cells. The reason of the T cells being triggered is unknown [15].



Environment Trigger/ Genetic Susceptibility Keratinocyte injury and increase Antigen presentation Increased Production of Proinflammatory Activation of T-Cells by myeloid dendritic Cell leading to Th1 and Th17 Activation and Proliferation Th1 Cells Th17 (TNF,IFN) Cells (IL-23) Increased production of **Epidermal** Beta-defensins CXC-Acanthosis and Chemokines and CCLabnormal

4. Treatment

Different traditional methods, including topical, oral, biological, and parenteral medicines, are employed to treat psoriasis. Symptomatic alleviation has also been achieved through the use of phototherapy.

4.1 Topical Therapy

Topical psoriasis treatment typically offers somewhat to unsatisfactory results and is frequently accompanied by irritation. The primary therapy for mild to moderate psoriasis is topical therapy.

Table 1: Topical drugs, their mechanisms, and associated adverse effects [16]

| Drug | Mechanism | Adverse Effects |
|-------------|--|------------------|
| Vitamin D | Interferes with the genes that are | It provokes skin |
| analogues | responsible for keratinization, | inflammation. |
| | inflammation, and epidermal | They are |
| | growth. | therefore |
| | | administered in |
| | | conjunction |
| | | with topical |
| | | corticosteroids. |
| Corticoste | It inhibits the transcription of | Skin shrinkage |
| roids | genes to prevent the production of | and suppression |
| | pro-inflammatory cytokines. | of the adrenal |
| | | axis are side |
| | | effects of long- |
| | | term treatment. |
| Dianthrol | It results in mitochondrial | It has staining |
| | malfunction, the restoration of | qualities and |
| | cell differentiation, and decreased | induce skin |
| | keratinocyte proliferation. | irritation. |
| Retinoids | particularly binds to β and α | Erythema, |
| | retinoic acid, both of which are | stinging, |
| | present on the cell membrane of | burning, and |
| | keratinocytes, to influence gene | scaling. |
| | transcription. | |
| Tacrolimus | Inhibit both calcineurin | mild, well- |
| | phosphatase activity and the | tolerated skin |
| | production of inflammatory | irritation. |
| | mediators. | |
| Keratolytic | Acts via corneocyte desquamation. | Burning and |
| agents | | skin irritation. |



However, in serious cases, systemic administration combined with topical therapy may be helpful. Topical treatments may cause unfavorable skin interactions, such as irritation and burning, which could result in patient non-compliance [16].

4.2 Oral Therapy

When topicals, phototherapy, or biologics fail to control moderate-to-severe or resistant psoriasis, oral treatments are preferred as a solo or complementary treatment. The standard oral treatments for psoriasis have been methotrexate, cyclosporine, and acitretin [17].

Methotrexate boosts the release of adenosine, a natural immunological and anti-inflammatory modulator [18]. The gastrointestinal side effects are the most prevalent and include anorexia, nausea, stomatitis, and diarrhoea. CNS toxicity, which include headaches, vertigo, lethargy, and mood swings [19].

Cyclosporine is a calcineurin inhibitor that has been used for years to treat moderate to severe psoriasis [20]. Additionally, it prevents the synthesis of gamma interferon. Hypertension, nauseousness, headaches, and heightened sensitivity are typical adverse effects. It offers rapid onset of action. Furthermore, it is reportedly safe to use while pregnancy [16].

Acitretin is a retinoid and prevent keratinocytes from proliferating. It is non-immunosuppressive and is derived from vitamin A [16]. Acitretin can

be used to treat moderate to severe plaque psoriasis that is resistant to topical therapies or phototherapy. Common side effects may include Nausea, abdominal pain, Headache, night blindness, sticky skin, retinoid dermatitis, hair pigmentation, curling, and in very rare cases peripheral edema, suicidal tendency, capillary leak syndrome. Although side effects are frequent and dose-limiting, they can be reduced with careful patient selection, dosing, and monitoring [21].

4.3 Biologics

Biologic medicines that are frequently used to treat psoriasis are divided into three groups: tumour necrosis factor (TNF) α -inhibitors, interleukin (IL)-23 inhibitors, and IL-17 inhibitors. Although approved biologic agents vary by country [22]. Etanercept, Infliximab, and Adalimumab all work by inhibiting tumour necrosis factor- α (TNF- α). Ustekinumab inhibits the interleukin-12/23 (IL-12/23) pathway, and alefacept targets and inhibits T cells [23].

4.4 Phototherapy

Since the development of biologics, the use of phototherapy for moderate to severe psoriasis has declined. Narrowband UV-B, broadband UV-B, psoralen and UV-A (PUVA) are the three main phototherapy modalities used to treat psoriasis.

UV-B exposure reduces DNA synthesis, which causes keratinocyte death and lowers T-cellcell production of proinflammatory cytokines. Both



the broadband (290-320 nm) and narrowband (311 nm) wavelengths of UV-B can be utilized to treat plaque psoriasis. Due to their higher effectiveness, longer length of remission, lower photocarcinogenic potential, and less erythema at the same physical dose, narrowband UV-B are more frequently utilised than broadband UV-B. Adverse effects include photoaging, photocarcinogenesis, blistering, erythema, and pruritus.

PUVA: A psoralen, such as methoxalen, is used in PUVA therapy and is used topically or orally before UV-A (320-400 nm) radiation. Psoralens are intercalated into DNA in order to inhibit DNA synthesis. Although oral PUVA therapy is more effective than UV-B therapy, it is no longer favoured since prolonged use can lead to skin cancer. Other adverse effects include photoaging, gastrointestinal distress, burning, pruritus, and hypertrichosis [24].

5. Conclusion

Psoriasis is a chronic systemic inflammatory disorder that causes a number of concomitant conditions in addition to its unique skin lesions. Two distinct include features increased keratinocyte proliferation and the production of inflammatory mediators. Some of the various therapeutic techniques include topical, systemic, phototherapy, and biologics. Topical medications are preferred over systemic therapy, which are best in cases of severe disease, for mild to moderate psoriasis conditions. Systemic therapy includes biological agents and immunosuppressants. Aside from that. phototherapy has also proven successful in relieving symptoms.

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Conflict Of Interest

There are no conflicts of interest between the authors.

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