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Review on Psoriasis Disease

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Abstract: *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. This review would discuss and focus on recent updates in the management of Psoriatic patients and its common related issues as well as the clinical picture of psoriasis in order to understand and inform medical practitioners and develop their knowledge of the etiology of the condition, immune and environmental factors, has led to the development of precision-targeted therapies that alleviate patient morbidity*

Keywords: Psoriasis, management of psoriatic patients, treatment, skin disease, therapies.

I. INTRODUCTION

As one of the frequently encountered chronic inflammatory skin diseases, psoriasis affects 2–3% of the global population. It adversely burdens one's quality of life with disfiguring lesions, itching, and even serious mental disorders such as helplessness, embarrassment and anger.[1] However, there is still a lack of effective approaches for psoriasis therapy. Great efforts have been made by researchers in the way to understand the disease.[2] It has been intensively demonstrated that psoriasis is an immune-driven inflammatory skin disease, which involves Th17 secreting IL-17A, IL-17F and IL-22 to trigger epidermal hyperproliferation. Upon stimulation, keratinocytes do not only expand rapidly with aberrant differentiation, but also secrete inflammatory cytokines (e.g., IL-1b, IL-6, and TNF-a) and chemokines (e.g., CXCL8, CXCL9, and CXCL10) to induce neutrophil infiltration and further Th17 cell recruitment into the epidermis, thus self-amplifying skin inflammation. This makes psoriasis fundamentally different from dermatitis. Dermatitis is also a frequently encountered chronic inflammatory disease whose initiation is ascribed to the activation of the Th2 immune response.[3]

II. THE 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.10 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 (Figure 1). 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 naive T cells to TH1 cells. IL-23 is central to the survival and proliferation 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.Among these pathways, IL-23–mediated activation of the TH17 pathway is thought to be predominant. IL-23 signaling is mediated intracellularlyvia Tyk2-Jak2andSTAT3,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.[4]

III. DISEASE EVALUATION

Psoriasis skin disease impact involves both objective and subjective dimensions. Among objective dimensions are the extent, the severity (induration, scaling, erythema), and the localisation of skin involvement. Such aspects can be

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evaluated by ocular examination of the skin. Subjective dimensions may involve feelings of itch and pain but also impact on quality of life and perceived stigmatisation.[5]

When it comes to disease severity, it is surprising that an international consensus on what can be considered mild, moderate, and severe psoriasis is missing. The most ambitious quest to finally unite different perceptions under an international consensus is arguably the one published by the International Psoriasis Council (IPC) in 2019 (35).[6] Disappointingly, the final statement did not involve the terms mild, moderate, or severe disease. Instead, psoriasis severity was suggested to be dichotomized into the following:

Psoriasis patients should be classified as candidates for topical therapy or candidates for systemic therapy (including phototherapy). The latter are patients who meet at least one of the following criteria:

1. Body surface area involvement (BSA) > 10%
2. Disease involving special areas (e.g. face, palms, soles, genitalia, scalp, or nails)
3. Failure of topical therapy

In Sweden, the Swedish Society for Dermatology and Venereology (SSDV) has defined the boundaries for mild, moderate and severe psoriasis using psoriasis area and severity index (PASI) and dermatology life quality index (DLQI) in an attempt to capture both objective and subjective aspects of the disease. Severe disease is defined as PASI > 10 and/or DLQI > 10. Mild disease as PASI < 3 and DLQI ≤ 5. Moderate disease as 3 ≤ PASI ≤ 10 and/or 5 < DLQI ≤ 10. DLQI consists of 10 questions concerning patients´ perception of the last weeks impact of skin disease on different aspects of their health-related quality of life. It takes 2 minutes to fill in and scores 0 (no impact) to 30 (maximum impact). PASI is the golden standard for skin psoriasis assessment in Sweden but there are other scores such as body surface area (BSA) and physician's global assessment (PGA).[7]

IV. CLASSES OF THERAPY

Treatments for psoriasis can be usefully classed into four groups:

1. Topical agents requiring in-patient therapy such as dithranol or tar, which when used intensively have the ability to clear most or all of the plaques, e.g. Ingram's regime on in-patients.
2. Topical agents that are more 'patient-friendly' but less efficacious than those in class 1 and do not show a high rate of clearance, e.g. calcipotriol.
3. Ultraviolet radiation (UVB) or photochemothearpy (PUVA).
4. Systemic agents such as cyclosporin, methotrexate or retinoids.

As a general rule most patients would start off with agents in class 2, and then move on to classes 1, 3 and 4 when they are seen in hospital. The reason for placing class 1 agents before class 2 is historical, but does provide a necessary perspective to understand the limitations in efficacy of class 2 agents.[8]

V. LOCAL TREATMENT OF PSORIASIS: SIGNIFICANCE AND CHALLENGES

Currently, the treatment of psoriasis is achieved either systematically or topically. In systemic administration, a more efficient interaction of the drug with the target is achieved due to the circulation of the medication. This ensures that pathogenic factors are more specifically targeted. Even so, the cumulative organ toxicity or various side effects go along with the improvement of the primary disease on a long-term basis. Systematic administrations will also increase the overall drug consumption with low local drug retention in the skin tissue. Moreover, in some special cases, psoriasis patients are too weak or have too complex conditions to receive systemic treatment with high risks of systemic side effects. In such cases, the topical treatment must be applied. Actually, the topical therapy for psoriasis is of more clinical significance, not only because of the abovementioned issues of systematic therapy but also because of the following reasons:

Most psoriasis cases involve less than 5% of body surface area,[9] making topical treatment practicable

Most psoriatic cases are of mild or moderate severity, and thus the topical therapeutics are recommended to avoid systemic side effects

The local treatment of medicines, especially biologics, is proved to be more effective due to their direct action on the disease sites while the systemic administration may lead to the loss of activities of biologics during circulation before reaching the action sites

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the complex causal relationship between psoriasis and its comorbidities (e.g., metabolic syndrome and cardiovascular diseases) remains controversial. It is speculated that topical lesions release proinflammatory cytokines into circulation thus negatively affecting other systems or even the entire body. It was reported that in the blood of psoriasis patients, several inflammation biomarkers are detected in relation to disease activity,[10] which suggests the importance of an early and active control of local limited psoriatic inflammation.

DRUGS AVAILABLE[11]

Drug	Mechanism	Application
Methotrexate	Dihydrofolate reductase inhibition blocks purine biosynthesis; induction of lymphocyte apoptosis	s.c./oral
Cyclosporin	Calcineurin inhibition leading to reduced IL-2	Oral
Acitretin	Normalization of keratinocyte proliferation/differentiation through retinoid receptor binding	Oral
Fumarate	Intracellular glutathione, modulation of Nrf2, NF-κB, and HIF-1α; promoting a shift from a pro-inflammatory Th1/Th17 response to an anti-inflammatory/regulatory Th2 response.	Oral
Apremilast	PDE4 inhibitor increases intracellular cAMP levels in immune and non-immune cells modulating inflammation	Oral
Etanercept	Dimeric human fusion protein mimicking TNF-αR	s.c.
Infliximab	Chimeric monoclonal IgG1κ antibody that binds to soluble and transmembrane forms of TNF-α	i.v.
Adalimumab	Human monoclonal antibody against TNF-α	s.c.
Certolizumab	Fab portion of human monoclonal antibody against TNF-α conjugated to polyethylene glycol	s.c.

VI. CONCLUSION

The significance and challenges of local treatment of psoriasis are firstly discussed. Then biomaterials, including microneedles, nanoparticles,nanofibers and hydrogels, are extensively reviewed for their capacity in psoriasis treatments. The fabrication strategies, and the advantages and disadvantages of these biomaterials in treating psoriasis are particularly discussed. Finally, clinical trials of these biomaterials for psoriasis therapy are summarized. In fact, biomaterial-based strategies for psoriasis treatments are still in the very early stage. More investigations are highly demanded to expedite the clinical translation of biomaterials.

ABBREVIATION

Th17 T helper cell 17
IL-17A Interleukin-17A

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IL-17F Interleukin-17F
IL-22 Interleukin-22
IL-1b Interleukin-1b

REFERENCES

[1]. Y. Liu, J. G. Krueger and A. M. Bowcock, Genes Immun., 2007, 8, 1–12.
[2]. H. Valdimarsson, R. H. Thorleifsdottir, S. L. Sigurdardottir, J. E. Gudjonsson and A. Johnston, Trends Immunol., 2009, 30, 494–501.
[3]. A. M. Bowcock, Annu. Rev. Genomics Hum. Genet., 2005, 6, 93–122.
[4]. Alwan W, Nestle FO. Pathogenesis and treatment of psoriasis: exploiting pathophysiological pathways for precision medicine. Clin Exp Rheumatol. 2015;33(5)(suppl 93):S2-S6.
[5]. Gourraud PA, Le Gall C, Puzenat E, Aubin F, Ortonne JP, Paul CF. Why statistics matter: limited inter-rater agreement prevents using the psoriasis area and severity index as a unique determinant of therapeutic decision in psoriasis. J Invest Dermatol. 2012;132(9):2171-5.
[6]. Otero ME, van Geel MJ, Hendriks JC, van de Kerkhof PC, Seyger MM, de Jong EM. A pilot study on the Psoriasis Area and Severity Index (PASI) for small areas: Presentation and implications of the Low PASI score. J Dermatolog Treat. 2015;26(4):314-7.
[7]. Muramatsu S, Kubo R, Nishida E, Morita A. Serum interleukin-6 levels in response to biologic treatment in patients with psoriasis. Mod Rheumatol. 2017;27(1):137-41.
[8]. Mihatsch MJ, Wolff K. Consensus conference on cyclosporin A for psoriasis. Br J Dermatol 1992; 126(6):621–3.
[9]. A. Menter, N. J. Korman, C. A. Elmets, S. R. Feldman, J. M. Gelfand, K. B. Gordon, A. Gottlieb, J. Y. Koo, M. Lebwohl, H. W. Lim, A. S. Van Voorhees, K. R. Beutner, R. Bhushan and American Academy of Dermatology, J. Am. Acad. Dermatol., 2009, 60, 643–659.
[10]. W. H. Boehncke, S. Boehncke, A. M. Tobin and B. Kirby, Exp. Dermatol., 2011, 20, 303–307.
[11]. Micha R, Imamura F, Wyler von Ballmoos M, Solomon DH, Hernan MA, Ridker PM, et al. Systematic review and meta-analysis of methotrexate use and risk of cardiovascular disease. Am J Cardiol. 2011;108(9):1362-70.