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Integrated Management of Psoriasis: From Targeted Biologics to Adjunctive Traditional Chinese Medicine Therapies

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Abstract: ***Psoriasis is a chronic immune-mediated inflammatory skin disease. The advent of biologics has significantly improved treatment efficacy. However, with their increasing application, numerous drawbacks—such as adverse reactions, the generation of anti-drug antibodies, immune deviation, disruption of skin barrier function, and high recurrence rates after discontinuation—have limited their broader use. In the current era of biologics, Traditional Chinese Medicine (TCM) offers not only traditional advantages like synergistic effects, reduced adverse reactions, and lower relapse rates, but also features multi-angle, multi-target, and broad-spectrum therapeutic actions. Furthermore, it plays a significant role in the clinical management of psoriasis comorbidities.***

Keywords: Psoriasis, Biologics, Traditional Chinese Medicine (TCM).

1. Introduction

Psoriasis is an immune-mediated chronic inflammatory disease characterized histologically by epidermal hyperplasia with parakeratosis and infiltration of various inflammatory cells, including lymphocytes and neutrophils, in the dermis. Psoriasis is classified into four major subtypes: plaque psoriasis, psoriatic arthritis, erythrodermic psoriasis, and pustular psoriasis. Among these, plaque psoriasis accounts for 80%-90% of cases and presents with well-defined erythematous plaques with scaling, which can become widespread in severe cases. It is currently understood that psoriasis is a chronic disease resulting from the complex interaction of genetic, immune, and environmental factors. Genetic factors play a significant role, with over 80 psoriasis susceptibility loci identified, including IL23R. The positive feedback immune circuit involving IL-17 and IL-23 is a central component of the disease pathogenesis. Additionally, positive feedback circuits involving interferon-γ (IFN-γ) and IL-36 also play crucial roles. These three inflammatory circuits interact and amplify one another, perpetuating the inflammatory response. Numerous highly effective targeted therapies have been developed against these molecules. These include not only monoclonal antibodies targeting cytokines like TNF, IL-23, IL-17, and IL-36, which have been used clinically for some time with good efficacy, but also small-molecule drugs such as Janus kinase (JAK) inhibitors. The latter have become a new focus in psoriasis drug development due to advantages like diverse routes of administration and lack of immunogenicity.

2. Immunological Pathogenesis of Psoriasis

Psoriasis is an autoimmune disease mediated by immune cells such as dendritic cells, neutrophils, and T cells through three major, interconnected inflammatory circuits:

1) The IL-17 and IL-23 feedback circuit-driven T helper 17 (Th17) and cytotoxic T cell 17 (Tc17) response.

2) The type I and type II interferon circuit driven by plasmacytoid dendritic cells (pDCs).

3) The IL-36 and neutrophil feedback circuit driven by neutrophil chemokines.

IL-23 promotes the proliferation of Th17 cells, Tc17 cells, and group 3 innate lymphoid cells (ILC3s) and their secretion of IL-17. IL-17, in turn, upregulates C-C motif chemokine ligand 20 (CCL20), which recruits Th17 cells, establishing a positive IL-17 feedback loop. On one hand, IL-17 stimulates the abnormal proliferation of keratinocytes, forming psoriatic plaques. On the other hand, it induces keratinocytes to produce inflammatory cytokines, which further activate IL-23 production and recruit IL-17-producing cells, participating in the positive feedback loop.

pDCs produce IFN-α, a type I interferon, while Th1 and Tc1 cells secrete IFN-γ, a type II interferon. These interferons act on keratinocytes to trigger skin inflammation. Conversely, IFN-γ induces the expression of chemokine (C-X-C motif) ligand 9 (CXCL9) and ligand 10 (CXCL10), which recruit Th1 and Tc1 cells to the site of inflammation, forming a positive feedback loop involving the interferon response [1].

IL-36 is expressed by various cells, including keratinocytes, and acts on keratinocytes to promote the secretion of neutrophil chemokines. This attracts neutrophil infiltration, leading to skin pustules characteristic of generalized pustular psoriasis. IL-36 also induces the secretion of chemokines and cytokines such as IL-17 and TNF-α. Furthermore, the production of IL-36 can itself be induced by IL-17, TNF-α, and IL-36, forming a positive feedback immune circuit involving IL-36 and IL-17.

These inflammatory circuits mutually amplify each other: IFN-γ promotes the IL-23 and Th17 response, and the IL-17 response promotes the expression and activation of IL-36. This results in a complex, interacting, and self-sustaining inflammatory network in psoriasis.

The JAK-STAT signaling pathway plays a crucial role in the

pathogenesis of psoriasis, with different JAKs mediating signals from different cytokines. JAK2 and TYK2 mediate signaling downstream of IL-23: dendritic cell-derived IL-23 acts on T cells, binding to its receptor and activating JAK2 and TYK2. The activated JAKs lead to phosphorylation of STAT3 and its dimerization. The phosphorylated STAT3 dimer translocates to the nucleus to regulate gene transcription, subsequently inducing the production of IL-17A and IL-17F. IL-22 activates JAK1 and TYK2, leading to STAT3 phosphorylation, and is involved in keratinocyte proliferation and differentiation. Furthermore, IFN-α and IFN-β bind to their receptors and activate STAT1 via JAK1 and TYK2, while IFN-γ signaling functions through JAK1 and JAK2.

3. Latest Pharmacological Treatment Strategies for Psoriasis

3.1 TNF-Targeting Biologics

TNF-α is a pro-inflammatory cytokine produced by keratinocytes, dendritic cells, neutrophils, activated lymphocytes, and other cells, and is highly expressed in psoriatic lesions. TNF-α induces the expression of endothelial adhesion molecules, recruiting more inflammatory cells to the lesions, which in turn produce cytokines and amplify the inflammatory response. TNF-α inhibitors were the first class of biologics approved for treating moderate-to-severe psoriasis and psoriatic arthritis. They work by targeting TNF-α to reduce its bioactivity, inhibiting keratinocyte proliferation, Th17 cell differentiation, and cytokine release. Among them, etanercept was the first used for treating moderate-to-severe plaque psoriasis. It competitively binds to TNF-α in the blood, blocking its binding to TNF receptors, thereby reducing psoriatic inflammation. Other TNF-α inhibitors currently used to treat psoriasis include infliximab, adalimumab, golimumab, and certolizumab pegol. Real-world studies have further validated the long-term efficacy and safety of TNF inhibitors. A prospective single-center real-world study from the Department of Dermatology at West China Hospital, Sichuan University, showed that adalimumab was superior to methotrexate in terms of PASI75 response rate and safety [2]. Unlike other biologics, certolizumab pegol is a polyethylene glycol-conjugated monoclonal antibody lacking the IgG Fc segment, preventing it from crossing the placental barrier; thus, it can be used in pregnant and lactating women [3]. Recent studies indicate that TWEAK (TNF-like weak inducer of apoptosis), a member of the TNF superfamily, promotes the upregulation of CXC-type chemokines and psoriasis-characteristic inflammatory molecules such as S100A8/A9 and SERPINB1/B9 in keratinocytes. Animal experiments show that antibodies neutralizing TWEAK function have efficacy comparable to therapeutic antibodies against IL-17 and TNF-α, suggesting that antagonizing TWEAK may be a novel alternative strategy for psoriasis treatment [4]. However, the use of TNF-α antagonists can also exacerbate psoriasis or induce paradoxical psoriasis, which may be related to TNF-α inhibiting IFN-α secretion by dendritic cells [5-6].

3.2 IL-23-Targeting Biologics

IL-23 initiates the intracellular JAK/STAT signaling pathway by binding to the extracellular domain of its cell receptor complex, activating the expression of psoriasis-related genes such as vascular endothelial growth factor (VEGF), intercellular adhesion molecule-1 (ICAM-1), and IκB-α (inhibitor of NF-κBα). The IL-23/Th17 axis has long been considered a central component in psoriasis pathogenesis. IL-23 induces Th17 activation and the release of inflammatory factors that act on keratinocytes. Stimulated keratinocytes produce more cytokines, including IL-23, amplifying the inflammatory response and leading to psoriatic pathological changes. Current IL-23 inhibitors for psoriasis treatment include guselkumab, tildrakizumab, risankizumab, ustekinumab, and JNJ-77242113. Among these, guselkumab was the first FDA-approved biologic specifically targeting the IL-23 p19 subunit for moderate-to-severe plaque psoriasis, showing significant efficacy in treating both plaque psoriasis and psoriatic arthritis [1]. In a Phase III clinical trial, 85% of patients using guselkumab achieved an IGA score of 0 or 1 at week 16, and 73% achieved PASI90 response, significantly outperforming placebo and adalimumab [1]. Furthermore, both domestic and international real-world studies indicate that guselkumab demonstrates sustained high response rates, offering significant clinical benefits, especially in refractory lesion areas [7-8]. Additionally, biologics such as tildrakizumab, risankizumab, and ustekinumab have also demonstrated therapeutic advantages in real-world clinical practice. Related research shows these drugs combine rapid onset of action with long-lasting therapeutic effects [9-14]. JNJ-77242113, a novel oral peptide, selectively and effectively blocks proximal IL-23 receptor signaling and downstream cytokine production by antagonizing the IL-23 receptor. It has also shown significant efficacy in treating psoriasis, with advantages such as low immunogenicity and oral administration [15].

3.3 IL-17-Targeting Biologics

IL-17 transmits signals by binding to a transmembrane complex of IL-17 receptor A (IL-17RA) and IL-17 receptor C (IL-17RC), thereby promoting inflammatory factor transcription and expression, stimulating keratinocyte proliferation, and contributing to local inflammation. Current IL-17-targeting biologics primarily include: those neutralizing the biological activity of IL-17A, such as secukinumab, ixekizumab, xeligekimab, and vunakizumab / SHR1314; bimekizumab, which inhibits the biological functions of both IL-17A and IL-17F; and brodalumab, which blocks the signaling pathway of the IL-17RA receptor. Numerous domestic and international real-world studies indicate that secukinumab, ixekizumab, and brodalumab all demonstrate significant long-term efficacy and reliable safety profiles in treating plaque psoriasis [8, 16-19]. Vunakizumab and xeligekimab were approved in China in August 2024 for treating adult patients with moderate-to-severe plaque psoriasis [20-21]. Bimekizumab is the first dual inhibitor of IL-17A and IL-17F approved for treating psoriasis, first authorized in Europe for moderate-to-severe plaque psoriasis. In Phase III trials, over 90% of plaque psoriasis patients achieved PASI90 response after 16 weeks of bimekizumab treatment, demonstrating faster and stronger therapeutic effects [22]. Several international real-world studies also indicate that bimekizumab is highly effective in treating

plaque psoriasis and psoriatic arthritis, with rapid onset, sustained long-term efficacy, good safety, and significant therapeutic effects even in patients previously treated with other biologics [23-26]. However, similar to other biologics, bimekizumab is administered via injection, and patient adherence may not be as high as with oral small-molecule drugs. To date, among domestic IL-17A-targeting drugs for psoriasis, SSGJ-608, HB0017, JS005, and AK111 have all entered Phase III clinical trials (NCT05536726, NCT06477237, NCT05975268, NCT06066125).

3.4 IL-36-Targeting Biologics

IL-36 belongs to the IL-1 family of cytokines and plays a crucial role in the pathogenesis of generalized pustular psoriasis (GPP). In psoriasis, IL-36 expression is upregulated. It binds to its receptor, inducing the recruitment of IL-1RAcP and activating intracellular signaling pathways, exerting pro-inflammatory effects. Currently, two drugs targeting IL-36R, spesolimab and ANB019 (imsidolimab), have shown promising efficacy in international clinical trials. Spesolimab was the first IL-36R-targeting drug approved for treating GPP, demonstrating potent efficacy in Phase II trials [27]. Trial results showed that by the end of week 1, 54% of patients in the spesolimab group achieved a GPPGA (Generalized Pustular Psoriasis Physician Global Assessment) score of 0, compared to only 6% in the placebo group (NCT03782792) [27]. Furthermore, a foreign real-world study reported rapid and sustained efficacy for spesolimab, with 100% of patients (n=5) achieving a GPPGA score of 0 at week 1 and no treatment-related adverse events. However, the sample size was small, and large-scale, long-term studies are needed to verify its durability and patient relapse risk [28]. Additionally, domestically developed humanized monoclonal antibodies targeting IL-36R, HB0034 and Recibokibart, as well as IMG-008, in which China is involved in development, have also entered clinical trials [29-30]. Similarly, IL-36 plays an important role in the pathogenesis of psoriatic arthritis, but there is currently a lack of clinical trials for IL-36R-targeting drugs treating psoriatic arthritis [31].

4. Current Status of Traditional Chinese Medicine as an Adjunct to Biologic Therapy

4.1 Synergistic Enhancement

The most common current clinical application of TCM as an adjunct to biologics is synergistic enhancement. Wang Jue [32] treated patients with moderate-to-severe plaque psoriasis of the wind-heat accumulation toxin pattern using Baibi Sanhuang Ointment combined with adalimumab, increasing the biologic's effective rate from 84.37% to 90.63%. Zheng Mingjing et al. [33] treated psoriasis patients pattern-differentiated as blood-heat syndrome with Xijiao Dihuang Decoction combined with secukinumab injection, achieving a markedly superior effective rate of 93.33% compared to secukinumab alone (73.33%).

4.2 Reducing Adverse Reactions

Adverse reactions following biologic use are very common, and timely TCM intervention can effectively alleviate symptoms. Li Wen et al. [34], when treating primary failure and inflammatory adverse reactions after secukinumab use, employed oral Chinese herbs and external medicinal baths with heat-clearing, blood-cooling, toxin-resolving, and itch-relieving properties, leading to gradual improvement of the patient's skin lesions.

4.3 Reducing Post-Discontinuation Relapse Rates

TCM can effectively lower the high relapse rates associated with biologic discontinuation. Du Juan et al. [35] treated psoriasis with Quyin Mixture combined with biologics, increasing the PASI90 response rate from 53.1% to 72.6% and reducing the six-month post-discontinuation relapse rate from 66.3% to 50%. Li Wen et al. [34], when treating patients who relapsed after stopping biologics, used oral administration and medicinal baths primarily based on Liangxue Jiedu Decoction, leading to rapid improvement of skin lesions without recurrence in the short term.

However, high-quality research reports on combining biologics with TCM remain relatively scarce. Furthermore, research reports on combining biologics with other first-line Western medications are also insufficient. On one hand, the efficacy of biologics as monotherapy has significantly improved compared to traditional treatment models, reducing patient demand for other therapies. On the other hand, biologics are expensive, and the treatment and maintenance phases are prolonged, making them unaffordable for many patients. Consequently, collecting sufficient samples for research on combining biologics with other treatments is challenging. Additionally, the clinical use of biologics is still in an exploratory phase, lacking highly authoritative guidelines or consensus. Based on the current research landscape, the persistently high relapse rate remains the most significant limiting factor for biologics. In response to this, TCM formulas primarily focusing on three therapeutic effects—clearing heat and resolving toxins, clearing heat and cooling blood, and clearing heat and activating blood—demonstrate considerable advantages in enhancing efficacy, reducing side effects, and lowering relapse rates.

5. Summary

Although biologics demonstrate significant efficacy, their targets are relatively singular. Faced with the complex pathological mechanisms of psoriasis, this single-target approach for achieving sustained clinical efficacy is bound to face severe challenges. Currently, research on biologics, both domestically and internationally, requires further in-depth exploration. For instance, research on reducing the incidence of anti-drug antibodies (ADA) remains insufficient; studies on immune deviation are mostly concentrated on atopic dermatitis (AD); and there is a lack of research on effective solutions for the persistently high relapse rates after drug discontinuation [36]. Although TCM research has achieved a degree of breakthrough in enhancing efficacy, reducing side effects, and lowering relapse rates, there is a lack of in-depth investigation and discussion on its mechanisms of action. Moreover, TCM research predominantly focuses on blood-cooling, blood-activating, and heat-clearing methods, with few reports on combining biologics with TCM formulas possessing tonic, exterior-releasing, or yang-warming effects, or other external TCM therapies. This, to some extent,

restricts the full realization of TCM's therapeutic advantages.

Based on this, the approach of combining TCM and biologics for psoriasis treatment can fully leverage the advantage of TCM's "holistic concept." It can be developed in terms of therapeutic breadth, offering three new potential avenues: multi-angle, multi-target action; simultaneous action on pro-inflammatory and anti-inflammatory pathways; and leveraging TCM's advantages in treating psoriasis comorbidities. This could further enhance TCM's strengths in synergistic enhancement, toxin-clearing and side-effect reduction, and preventing relapse during stable disease. Unfortunately, although numerous TCM research reports touch upon these three new directions, there is still a lack of related reports on their combination with biologics for psoriasis treatment. Future research could focus on the aforementioned three aspects, delving deeper into the mechanisms by which TCM enhances efficacy, reduces side effects, and lowers relapse rates for biologics, thereby forming a complementary relationship in both depth and breadth with the single-target approach of biologics in clinical practice.

However, TCM's therapeutic advantages and research avenues extend far beyond this. Numerous psoriasis treatment theories are continuously evolving. For example, new theories such as the "Xuanfu Theory", "State-Target Pattern Differentiation" [37], and "Visceral Wind-Dampness" [38] are guiding TCM to make continuous progress in treating psoriasis. Therapeutic methods represented by warming yang and releasing the exterior and supporting vital qi and tonifying the kidney have already shown efficacy [39]. Biologics have brought a milestone improvement to the clinical treatment of psoriasis and a significant impact on traditional medical models. How to fully leverage TCM's therapeutic advantages in the era of biologics will be a worthwhile direction for future consideration and research.

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