Research Progress of Atopic Dermatitis in Traditional Chinese and Western Medicine

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Abstract: Atopic dermatitis (AD), also known as atopic eczema, is a chronic, relapsing, inflammatory skin disease. Its etiology is not yet fully understood, but it is believed to be associated with various external factors, including genetics, immune dysregulation, environmental influences, and skin barrier dysfunction. By reviewing the literature, this article summarizes the pathogenesis, triggering factors, and integrated traditional Chinese and Western medicine treatment approaches, aiming to provide the latest research progress for the treatment of AD.

***Keywords*:** atopic dermatitis; integrated traditional Chinese and Western medicine; efficacy; research progress.

1. Clinical Manifestations

Atopic dermatitis is a skin disease related to heredity and allergic constitution, mainly characterized by pruritus and exudative tendency. Its clinical manifestations vary with age. In the early stage, erythema and papules appear on the skin with indistinct boundaries. Scratching may lead to erosion, exudation, and crusting, with a tendency to spread. After treatment, the condition may improve or resolve, but some cases persist into adulthood, with lichenified skin lesions. Severe pruritus may result in excoriation and bleeding.

In the 21st century, the one-year prevalence of adult AD in Asia is 1.2%, and in Europe it is 17.1%. In Asian children, the prevalence ranges from 0.96% to 22.6%. In Italy, the one-year incidence is 10.2%, while in Scotland it is 95.6%[1]. In recent years, the prevalence of AD among preschool children in China is 2.78%, and among children aged 1–7 years it is 12.94%. About 30.48% of newborns in China are affected by AD, and the prevalence is increasing year by year[2]. There are few studies on the incidence of AD in the 21st century, especially in adults. Therefore, further epidemiological studies are needed for both children and adults across different continents.

2. Etiology and Pathogenesis

Atopic dermatitis is an inflammatory disease mediated by Th2 cells. The skin is composed of various cell populations that maintain homeostasis. Recent studies have revealed the complexity of the immune pathogenesis of AD, showing a close relationship between Th2-specific dendritic cells and skin chemokines (ITGA4, ITGB1, and CCR2)[3]. CD103 (ITGAE) levels are low, while CD11c (ITGAX) levels are high. Several AD-specific immune cells have been identified in lesional skin, including macrophages expressing CCL13 and CCL18, T cells expressing CCR7, and fibroblasts expressing COL6A5 and CCL19. Memory B cells expressing IL4R, FCER2, and IgG are increased in AD and are associated with allergic diseases through IgE production. Overactivation of the NF-κB pathway may lead to chronic inflammation in AD skin. Deletion of inhibitory IKKβ in fibroblasts increases CCL11 expression, promoting eosinophilia and shifting the immune response toward type 2 immunity. Fibroblasts lacking IKKβ in facial skin exhibit AD-like features, including scratching behavior.

According to the immune theory, most AD patients have elevated serum IgE levels. Th2 cells are significantly increased in skin lesions, and the interleukins (IL-4, IL-5) they produce can increase IgE levels and eosinophilia. Abnormal numbers of Langerhans cells in the skin can activate Th2 cells, leading to their proliferation and differentiation[4]. Mutations in the high-affinity IgE receptor in some patients result in abnormal IgE-mediated hypersensitivity reactions. In AD, epithelial cytokines such as IL-25, IL-33, and TSLP can directly promote Th cell cytokine secretion (e.g., IL-4, IL-5, IL-13, IL-31) or indirectly via dendritic cell polarization. IL-4 binds to the IL-4 receptor, activating JAK1 and JAK3, which in turn activate IL-4Rα and STAT-6. Phosphorylated STAT-6 dimerizes and functions as a transcription factor by binding to specific DNA sequences in IL-4-responsive genes[5].

The JAK-STAT signaling pathway also plays a role in AD pathogenesis. It regulates various downstream cytokines and growth factors involved in biological development and immune modulation. JAK kinases include JAK1, JAK2, JAK3, and TYK2; the STAT family includes seven proteins: STAT1–6 and 5A/5B[6]. The JAK-STAT cascade is initiated by cytokine-receptor binding. JAKs bind to type I and II cytokine receptors, activating both JAKs and receptors[7-8]. Pruritus

is transmitted via unmyelinated C-fibers and myelinated Aδ-fibers from the dorsal root ganglia. STAT3 activation mediates astrogliosis in the spinal dorsal horn, amplifying chronic itch. Lipocalin-2 (LCN2) enhances itch signaling and triggers the vicious itch-scratch cycle. JAK1 is involved in AD pathogenesis via IL-4, IL-5, IL-13, IL-22, TSLP, and IFN-γ signaling. Overactivation of JAK1 leads to overexpression of skin serine proteases, impairing skin barrier function[9]. STAT3 is a key transcription factor regulating keratinocyte differentiation and skin integrity. In mouse models, the JAK inhibitor delgocitinib inhibits JAK1/2/3, suppresses STAT3 activation, and improves skin barrier function by increasing epidermal proteins such as filaggrin (FLG), loricrin, and natural moisturizing factors[10].

3. Drug Therapy

3.1 Traditional Chinese Medicine (TCM) Treatment

3.1.1 External TCM Therapy

Common TCM treatments for AD include acupuncture, wet compresses, cotton moxibustion, and oral herbal medicine. Acupuncture modulates T-cell activation and cytokine secretion to maintain immune balance and reduce inflammation[11]. Fire acupuncture, a modified form[12], can also be used. It warms meridians, dispels cold, clears heat, detoxifies, and promotes tissue regeneration. For heat-type AD, fire acupuncture can clear heat and expel toxins. For exudative acute-stage patients, herbal wet compresses can be applied after syndrome differentiation and herbal preparation. Cotton moxibustion[13] involves burning thin cotton layers over the skin, providing immediate itch relief without pain, and is well-tolerated. For refractory AD, TCM treatment based on syndrome differentiation is recommended, combining internal and external therapies.

3.1.2 Oral TCM Therapy

AD can be classified into different TCM syndromes:

(1) Wind-Dampness Accumulation Type: Characterized by severe itching, blisters, erosion, and exudate. Modified Guomin Decoction[14] is recommended.

(2) Wind-Dampness-Heat Toxin Type: Based on wind-dampness with heat toxins. Symptoms include itching, exudate, fever, irritability, dry mouth, and constipation. Qu Feng Bai Du San[15] is used.

(3) Blood Deficiency and Wind-Dryness Type: Recurrent due to blood deficiency and wind-dryness. Treatment focuses on nourishing blood and dispelling wind. Jian Pi Yang Xue Qu Feng Tang[16] is used.

(4) Liver Qi Stagnation Type: Due to chronic recurrence, patients may experience anxiety, depression, and insomnia. Modified Xiao Yao San[17] is used.

3.2 Western Medicine Treatment

3.2.1 Conventional Therapy

The main principle is to relieve symptoms and avoid triggers. Topical corticosteroids are first-line treatments[18], selected based on severity. Calcineurin inhibitors (e.g., tacrolimus, pimecrolimus) are used for T-cell-mediated inflammation[19]. Phototherapy[20] is also applied, sometimes in combination with calcineurin inhibitors and UVB[21]. Antihistamines (e.g., loratadine, cetirizine) are used for severe itching. Antibiotics or antivirals are used for secondary infections. Immunosuppressants (e.g., cyclosporine, methotrexate) are considered for severe cases[22].

3.2.2 Biologics

For patients unresponsive to conventional therapy, biologics such as dupilumab, tralokinumab, and secukinumab are used[23]. These target type 2 helper T-cell-mediated inflammation and have shown significant improvement in pruritus and skin lesions in clinical trials.

3.2.3 Gut Microbiota Regulation

Dysbiosis of skin and gut microbiota may influence AD onset and progression. Probiotics (e.g., Bacillus licheniformis, Bacillus coagulans) can regulate microbiota, improve immunity, and alleviate symptoms[24-25].

3.2.4 Moisturizers and Skin Barrier Repair

Genetic mutations in barrier-related genes (e.g., filaggrin, protease inhibitors) impair skin integrity[26]. Medical moisturizers (e.g., Collgene, Winona, Yuze) can restore the barrier[27], relieve dryness and itching, and prevent secondary infections[28].

3.3 Integrated TCM and Western Medicine Treatment

Routine care focuses on moisturization. Topical corticosteroids or tacrolimus are used for inflammation. Acute stages

are managed with corticosteroids, and maintenance with tacrolimus and moisturizers. Oral TCM decoctions can be added based on syndrome differentiation. For example, Qing Re Li Shi Fang (Heat-Clearing and Dampness-Removing Decoction) [29] has anti-inflammatory and microbiota-regulating effects, especially in children.

4. Conclusion

Atopic dermatitis is a chronic inflammatory disease with genetic predisposition, influenced by environmental, dietary, emotional, and lifestyle factors. TCM emphasizes individualized treatment based on syndrome differentiation, while Western medicine offers rapid symptom control. Integrated treatment combines the strengths of both systems, using TCM for internal regulation and Western drugs for external control, gradually tapering off medications. Daily moisturization and probiotic supplementation can enhance skin barrier function and immunity, forming a virtuous cycle of reduced damage and improved recovery.

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