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Janus Kinase Inhibitors: A Paradigm Shift in Contemporary Dermatologic Practice

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Dear Editor

Over the past decade, the introduction of Janus kinase (JAK) inhibitors has profoundly reshaped the therapeutic landscape of dermatology. By selectively targeting intracellular signaling pathways central to immune dysregulation, these small-molecule agents have offered a level of efficacy, rapidity of response, and disease control that was previously unattainable for many chronic inflammatory and immune-mediated skin disorders. Collectively, emerging clinical trial data and real-world experience support the view that JAK inhibitors represent a genuine paradigm shift in contemporary dermatologic practice [1].

Several JAK inhibitors are now approved for dermatologic indications, most notably atopic dermatitis, alopecia areata, and vitiligo, with agents such as baricitinib, upadacitinib, abrocitinib, and ruxolitinib demonstrating robust efficacy across multiple disease severity spectra. These approvals have validated the JAK-STAT pathway as a critical therapeutic target in skin inflammation and autoimmunity. Importantly, the clinical impact of JAK inhibition extends well beyond currently approved indications. An expanding body of evidence-including case reports, small series, and mechanistic studies-has highlighted their off-label utility in a wide range of dermatologic conditions such as lichen planus, dermatomyositis, chronic pruritus, hidradenitis suppurativa, granulomatous dermatoses, autoimmune blistering diseases, and immune-mediated photodermatoses [2].

In this context, JAK inhibitors have demonstrated particular promise in conditions characterized by treatment resistance or limited therapeutic options. Their oral administration, predictable pharmacokinetics, and relatively rapid onset of action contrast favorably with conventional systemic immunosuppressants and biologics. For many patients, especially those with refractory disease, JAK inhibitors have shifted treatment goals from partial symptom control toward meaningful disease modification and improved quality of life [3].

Despite these advances, access to JAK inhibitors remains highly uneven across different regions of the world. In Iran, as in many low- and middle-income countries, availability of these agents is limited, regulatory approval for dermatologic indications is incomplete, and cost represents a substantial barrier. Consequently, only a small proportion of dermatologists currently prescribe JAK inhibitors, often in highly selected patients after exhaustion of conventional therapies. This reality creates a significant gap between global therapeutic progress and real-world practice, underscoring

the need for region-specific data and experience to guide rational, safe, and equitable use.

Within this constrained setting, our group has accumulated growing clinical and academic experience with JAK inhibitors across several challenging dermatologic conditions. We have explored the role of JAK inhibition in immune-mediated photodermatoses, a heterogeneous group of disorders-including polymorphous light eruption, actinic prurigo, solar urticaria, chronic actinic dermatitis, and hydroa vacciniforme-characterized by aberrant immune responses triggered by ultraviolet radiation. These conditions often respond poorly to conventional therapies and impose a significant burden due to the necessity of strict photoprotection and chronic immunosuppression. Our work has highlighted the potential of JAK inhibitors as a targeted therapeutic option capable of addressing both inflammatory signaling and pruritus in refractory cases [4].

In addition, we have reported successful clinical outcomes with baricitinib in pediatric actinic prurigo, illustrating the potential role of JAK inhibition even in younger patients with severe, persistent disease when standard treatments fail [5]. Beyond photodermatoses, our randomized clinical trial comparing baricitinib with azathioprine in adults with moderate-to-severe atopic dermatitis demonstrated favorable efficacy and an acceptable safety profile for JAK inhibition in a real-world, single-center setting [6]. Such comparative data are particularly relevant in regions where traditional immunosuppressants remain the mainstay of systemic therapy due to cost and accessibility.

Moreover, emerging case-based evidence from our group and others suggests that JAK inhibitors may offer effective alternatives in rare or treatment-resistant autoimmune skin diseases, such as dermatitis herpetiformis unresponsive to dapsone and dietary modification [7]. These observations further support the concept that JAK inhibition can modulate shared inflammatory pathways across diverse dermatologic entities, transcending traditional disease classifications.

Nevertheless, enthusiasm for JAK inhibitors must be balanced with careful patient selection, vigilant safety monitoring, and an awareness of potential adverse effects, including infections, laboratory abnormalities, and thromboembolic risk. In resource-limited settings, these considerations are magnified by restricted access to laboratory monitoring and follow-up. Therefore, pragmatic guidelines informed by real-world experience are urgently needed to optimize benefit while minimizing harm.

In conclusion, JAK inhibitors have undeniably transformed modern dermatologic therapeutics, offering new hope for patients with chronic, severe, and refractory skin diseases. However, their full potential will only be realized if issues of access, affordability, and education are addressed, particularly in regions such as Iran where clinical need is high but availability remains limited.

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