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Review Article

# Role of topical tofacitinib in autoimmune skin disorders: An expert opinion

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ABSTRACT

Autoimmune skin diseases, caused by immune system dysfunctions, have complex mechanisms. Biologicals are widely used to treat these chronic conditions. These available treatments for autoimmune skin diseases have several drawbacks, including related adverse effects, issues with adherence to long-term therapy, and the need for better management strategies. Recently, JAK inhibitors, like tofacitinib, have shown promise in treating vitiligo, alopecia areata (AA), atopic dermatitis (AD), and psoriasis, among other autoimmune dermatological conditions. Conventional systemic therapies require monitoring for adverse events (AEs) and risk of drug interactions, thus resulting in reduced compliance. Topical JAK inhibitors have a superior safety profile due to low systemic absorption, provide tailored therapy, and limited systemic effects. Topical tofacitinib is being investigated for off-label autoimmune dermatological disorders and has shown promising outcomes. According to the opinion of the experts, topical tofacitinib (2%), twice daily (BID), is suggested as an effective treatment for autoimmune skin diseases with minimal AEs.



## 1. Introduction

Autoimmune skin diseases are caused by a malfunctioning immune system, resulting in skin tissue death. Several distinct components are thought to interact in a complex and poorly understood manner to cause these autoimmune skin diseases and other inflammatory skin illnesses.[1] India has reported the highest incidence of common autoimmune skin disorders in Southeast Asia.[2] Recently, biologicals, either first-generation (anti-TNF) or second-generation (anti-IL-17/anti-IL-23), have been widely used to treat individuals with chronic inflammatory illnesses.[3] Patients with psoriatic arthritis and rheumatoid arthritis have been treated in clinics using the first inhibitors of signaling proteins that are directly connected to cytokine receptors, such as tofacitinib and baricitinib.[3,4]

Janus kinases (JAKs) of four types (TYK2, JAK1, JAK2, and JAK3) are the target of these signaling protein inhibitors. The cytokines bind to the cytokine receptors on the cell surface, resulting in the phosphorylation of JAK proteins, causing the dimerization of transcription factors known as signal transducer and activator of transcription (STAT), located intracellularly. These STAT proteins translocate into the nucleus and positively or negatively alter the gene expression of several inflammatory mediators.[3] Currently, JAK inhibitors appeal to dermatologists and are tested as systemic and/or topical treatments for various skin diseases, with options for oral administration and topical application.[5] JAK inhibitors, also known as Jakinibs, have proven to be effective in treating various autoimmune

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dermatological disorders, such as vitiligo, alopecia areata (AA), atopic dermatitis (AD), and psoriasis.

Tofacitinib, a first-generation JAK inhibitor, blocks the JAK1 enzyme, responsible for transmitting signals for the initiation of the inflammatory immune response.[6,7] The first approved indication of tofacitinib was to prevent allograft rejection, which was withdrawn owing to adverse events (AEs) associated with high dosage.[8,9] Currently, tofacitinib is widely used to treat rheumatoid arthritis, adult and juvenile psoriatic arthritis, and polyarticular juvenile idiopathic arthritis, which are FDA-approved indications for the drug. Similarly, ulcerative colitis, systemic lupus erythematosus, ankylosing spondylitis, and recently, COVID-19 pneumonia are the indications wherein tofacitinib is used.[6,8] Additionally, tofacitinib has also been used in various off-label dermatologic conditions such as AD, alopecia, psoriasis, and vitiligo.[6,10]

## 2. Unmet Needs in the Treatment of Autoimmune Skin Disorders

Current therapies for autoimmune skin disorders have limitations, including side effects, challenges with adherence to long-term therapy, limited access to management strategies, and a lengthy drug approval process.[11,12] Moreover, there is a lack of awareness, an educational gap, and a need for advocacy for newer, improved therapeutic options.[11,12] A better understanding of therapeutics, public education, and support is needed for newer management strategies.[11] National clinical practice guidelines based on standardized evidence-based care medicine are lacking in practice and present only limited approved treatment options.[13] These unmet needs have necessitated the development of newer therapeutics that could be used for the long term with minimal side effects.

Conventional systemic therapies have limited efficacy in some patients,[14] and non-compliance is a significant factor.[15] Furthermore, physicians often avoid systemic therapies due to the need for monitoring AEs and the potential risk of drug interactions.[15] Topical JAK inhibitors have a better safety profile due to limited systemic absorption.[16] Topical administration of JAK inhibitors (like tofacitinib) has the potential to be a targeted therapy, restrict unintentional systemic effects,[17] and may help reduce the AEs associated with oral JAK inhibitors.[6]

## 3. Topical use of Tofacitinib

Tofacitinib was initially developed as an oral medication for rheumatoid arthritis,[10] and its efficacy prompted exploration into other autoimmune conditions, including psoriasis and ulcerative colitis.[8] The topical formulation of tofacitinib was developed to mitigate the potential side effects from its long-term oral administration.[6] Clinical trials have exhibited promising results for topical tofacitinib in treating autoimmune skin diseases.[10]

## 4. Use of Tofacitinib in Autoimmune Skin Disorders

### *4.1. Alopecia areata (AA)*

Alopecia Areata (AA) is a prevalent autoimmune condition characterized by non-scarring hair loss, ranging from scalp patches to total body hair loss. In a normal, healthy hair follicle, the major histocompatibility complex (MHC) is reduced with high levels of transforming growth factor-beta (TGF-β) and other interleukins (IL). However, in AA, there is increased expression of MHC I and MHC II, a significant number of CD8 cells, and activation of the JAK/STAT pathway. During active disease, T-cells infiltrate the hair follicles, creating a microenvironment skewed towards active inflammation with interferon-gamma (IFN-$\gamma$) activating IL-2, IL-7, and IL-15. The JAK/STAT inhibitor blocks this IFN-$\gamma$ activity, making them a suitable therapeutic option for treating AA.[18]

Topical tofacitinib (2% ointment) twice daily (BID) for 6 months was studied in a 24-week, open-label, single-center study consisting of 10 patients with AA. Results indicated that 30% of patients reported hair regrowth with an average SALT (Severity of Alopecia Tool) reduction of 34.6%. The observed side effects included scalp skin irritation (40%) and folliculitis (10%), which resolved without treatment.[19] Further, a case series of 11 pediatric patients with AA, alopecia totalis, and alopecia universalis reported the efficacy and safety of topical tofacitinib (2% ointment) applied for an average duration of 34.5 weeks. The average reduction in SALT score was 32.3%, with 8 out of 11 patients showing improvement in SALT score and 3 patients demonstrating cosmetically acceptable regrowth. The treatment was tolerated without any AEs.[20] Another case series of 4 patients reported successful treatment of severe AA with 2% topical tofacitinib cream, applied BID for 7 months. One patient demonstrated a 93.3% change in SALT score from baseline and had hair regrowth; no AEs were reported.[21] Furthermore, an interesting case was registered with topical tofacitinib 2% cream in a 23-year-old female with a history of AA. Complete hair regrowth was observed in 4 weeks, and positive response was maintained even after 4 months post-treatment discontinuation.[22] These studies indicate the positive impact of topical tofacitinib in AA.

### *4.2. Vitiligo*

Vitiligo is an acquired and idiopathic autoimmune disease characterized by patchy depigmentation of the skin, hair, or both. The depigmentation results from progressive melanocyte destruction, mainly due to cytotoxic T-cell infiltration that further leads to the secretion of IFN-$\gamma$. This triggers the JAK/STAT pathway at the cellular level and mediates melanocyte destruction and loss of clinical

pigment. When the JAK/STAT pathway is suppressed, the dissociation of melanocytes that produce low levels of E-cadherin in the basal layer of the epidermis, a crucial step before melanocyte apoptosis, is impaired. Additionally, it has been demonstrated to reduce matrix metalloproteinases (MMP)-9, the most potent factor released by keratinocytes in response to TNF-$\alpha$ and IFN-$\gamma$, which was discovered to be elevated in patients with vitiligo in both blood and skin and induced disruption of E-cadherin. Since several activation and transcription processes for the disease progression are mediated via the JAK/STAT pathway, it is an exciting target for interrupting the disease and potentiating repigmentation.[23]

A pilot study of 2% tofacitinib cream (BID; 3 months) with narrowband ultraviolet B (NB-UVB) for treating facial vitiligo in 11 patients reported good to excellent repigmentation in all patients with a mean improvement of 70% in facial VASI score without any AEs.[24] Several case studies have reported the efficacy of topical tofacitinib in vitiligo patients. A 4-year-old boy with segmental vitiligo, treated with 2% tofacitinib cream BID along with NB-UVB, demonstrated freckling and 3 depigmented linear macules on the chin after 4 weeks and complete repigmentation at 6 months.[25] Additionally, a 17-year-old boy with stable nonsegmental vitiligo having acrofacial involvement (with a 15-year evolution) was treated with 2% topical tofacitinib ointment BID on lesions in combination with NB-UVB thrice a week, for 9 months. The study reported near complete facial repigmentation in the periorbital region, around the lips, and above the clavicle, with only minor AEs such as erythema and transient acne.[26] Another case concerning a 17-year-old girl with a 4-month history of vitiligo and depigmented patches on bilateral upper eyelids with associated leukotrichia found 2% tofacitinib cream treatment (BID; 5 months) to be effective. There was near-complete repigmentation of eyelashes without any AEs.[27]

### 4.3. Atopic dermatitis

Atopic dermatitis (AD) is a prevalent skin condition characterized clinically by persistent inflammation and histologically by infiltration of inflammatory cells, most notably lymphocytes, eosinophils, and mast cells. The JAK-STAT pathway is one of the essential signaling pathways in various inflammatory diseases, including AD. Th2 cells stimulate immature B cells via JAK-STAT, leading to mature B cells and plasma cells producing IgE, which binds to skin mast cells, triggering the release of histamine and increasing AD symptoms. Moreover, IL-4 from the Th2 immune milieu causes the epidermal cells to release chemokines, pro-inflammatory cytokines, and angiogenic factors, worsening AD pathophysiology. Further, IL-5 released through the JAK-STAT pathway activates eosinophils, attracting them to the skin via chemokine ligand (CCL)26 and worsening AD. Furthermore, IL-31 induces pruritus, thus intensifying AD symptoms. Therefore, all these pathways are activated, causing a chronic cycle that could be interrupted by inhibiting the JAK/STAT pathway.[28]

Topical tofacitinib has demonstrated efficacy in AD. A 4-week, phase IIa, randomized, double-blind, parallel-group, vehicle-controlled study in 69 adults with mild to moderate AD evaluated topical tofacitinib (2% ointment) for 4 months. The study reported a rapid onset of efficacy with increasing improvement up to week 4. The mean change in EASI score was significantly improved with tofacitinib, and patients with a clear or almost clear PGA score were substantially higher in the tofacitinib-treated group. Moreover, tofacitinib ointment was well-tolerated and had an acceptable safety and local tolerability profile.[29]

### 4.4. Psoriasis

Psoriasis is a chronic inflammatory condition that causes scaly erythematous lesions on the skin. Psoriatic lesions often have epidermal hyperplasia, parakeratosis, and an accumulation of inflammatory cells in the dermis. Further, T-cells, particularly Th17 cells, drive the inflammatory response, which is mediated by several cytokines, including TNF, IL-17, and IL-23. IL-23 binds to the IL-23 receptor, attracting a JAK2 and TYK2 heterodimer that auto-phosphorylates and activates the receptor, attracting STAT proteins. The STAT proteins bind, phosphorylate, and translocate to the nucleus to regulate gene transcription, further leading to cytokine production. TYK2 inhibitors suppress intracellular signal transduction downstream of the receptor, inhibiting TYK2 function and thus reducing cytokine production during psoriatic inflammation.[30]

A phase IIa, randomized, double-blind, parallel-group, vehicle-controlled study evaluated topical tofacitinib's efficacy in treating 71 patients with chronic plaque psoriasis. Tofacitinib ointment (2%), administered BID for 4 weeks, significantly improved the least squares mean along with significant improvement in induration and scaling. Mild or moderate AEs were reported in 35% of patients, which included nasopharyngitis and urinary tract infections.[31] Further, a phase IIb, randomized, double-blind, parallel-group, vehicle-controlled study evaluated topical tofacitinib ointment (1% and 2%) in 435 patients with mild to moderate chronic plaque psoriasis. Patients were divided into two treatment regimens: once a day (QD) and two times a day (BID) for 12 weeks. Patients receiving 2% tofacitinib QD, 1% tofacitinib QD, and 2% tofacitinib BID achieved a PGA-C response of clear/almost clear and $\geq$ 2-grade improvement from baseline compared to vehicle. Also, a response rate of 18.6% and 22.5% was observed for 2% tofacitinib QD and 2% tofacitinib BID, respectively, with a significantly improved PASI score in the 2% tofacitinib QD group. However, the highest incidence of AEs (which included nasopharyngitis and upper respiratory

tract infection) was reported in the vehicle QD group.[32]

## 5. Expert Opinion

Panel discussions were conducted by Eris Oaknet Healthcare Pvt. Ltd., Mumbai, India, and involved experts in autoimmune skin disorders across the country's four regions (east, west, north, and south). These experts deliberated on the best practices of using topical tofacitinib in their clinical practice and responded to the moderators' queries about its topical application.

### *5.1. Monitoring and evaluation of the effectiveness of topical tofacitinib*

Experts recommended baseline blood work for monitoring the side effects during the treatment, including complete blood count, liver enzymes, lipid profile, QuantiFERON-TB gold test, and Hepatitis B viral status. However, these tests are more relevant for oral tofacitinib and may not be necessary for topical application. Thus, monitoring and evaluating the effectiveness of topical preparation is challenging in clinical practice.

In AA, effectiveness is monitored using a reduction in SALT score and hair regrowth. In vitiligo, monitoring involves observing skin repigmentation or a decrease in body surface area (BSA) involvement. For AD, improvement of symptoms or physical appearances, such as complete or partial improvement in patches or reduction in pruritus, should be monitored. In psoriasis, monitoring involves observing the clearing of skin, decreasing scores (such as PGA-C, PASI), or reduction in psoriasis patches in terms of BSA.

### *5.2. Overview of experts' discussion*

Experts discussed the role of topical tofacitinib in various autoimmune diseases, agreeing on its beneficial effects in AA and vitiligo. However, they mentioned challenges in its application for AD and psoriasis due to their involvement in larger BSA. Many experts have either not used topical tofacitinib for psoriasis or AD or have not found it effective. They emphasized that topical tofacitinib should be reserved for localized, refractory cases that do not involve larger BSA. The experts further recommended supplementing topical tofacitinib with PUVA, NB-UVB therapy, or excimer light therapy for better results in vitiligo and AA. All the experts came to a common consensus that topical application is more tolerable and less stressful compared to oral medications in pediatric patients. Topical formulations also improve compliance, specifically in patients with steroid phobia. Some experts even recommended treatment initiation with topical tofacitinib rather than as a secondary option after other failed therapies. However, there was consensus on initiating treatment with oral therapy and using topical preparation as maintenance to prevent relapse once the disease has stabilized (in cases of vitiligo or AA).

Most experts suggested lipophilic formulations, such as gels and creams, for topical applications due to their effective penetration. Gel formulations were recommended for small patches on the scalp, beard, and other areas in AA, while ointments were preferred for extensive body surfaces. Moreover, ointment formulation is less irritating, more potent, and remains on the skin longer, resulting in less wastage than gel formulations. Gels or lotions are preferred for treating hairy areas, whereas foam formulations are recommended for scalp vitiligo, white hair repigmentation, or larger surface areas, necessitating the need for all formulation types (cream, gel, foam, and lotion). A formulation with improved efficacy and applicability is required, along with a larger pack size and affordable pricing. Cost-effectiveness is crucial, so experts recommended selecting formulations based on the affected area. One of the experts stated that tofacitinib is more economical for facial vitiligo compared to FDA-approved ruxolitinib.

Further, the experts recommended long-term clinical and pharmacokinetic studies on topical tofacitinib to assess its safety and efficacy. With limited evidence from a few case reports or case series, larger cohort studies are required, particularly for delicate areas like eyelashes, leukotrichia, eyelids in AA, and vitiligo. Moreover, comparative studies between calcineurin inhibitors and tofacitinib are needed to determine usage preference in autoimmune skin disorders like vitiligo.

Experts agreed that topical tofacitinib is safe, with negligible systemic absorption, even after long-term use. Therefore, laboratory investigations may not be required in limited applications (such as AA on the face or localized atopic patches). Further, systemic immunosuppression will be minimal as the treatment is recommended in small areas. However, some AEs reported by these experts have included folliculitis and skin irritation.

Experts have also expressed concern that better-penetrating agents, if applied to extensive surfaces, might cause systemic side effects. However, the experts have used topical tofacitinib for varied durations, ranging from one month to two years, without experiencing any serious AEs.

### *5.3. Best practices of using topical tofacitinib in autoimmune skin disorders*

Experts expressed that penetration remains challenging with the ointment formulation, suggesting a gel form would be more effective for facial, stable vitiligo. As per the experts, best practices include using topical tofacitinib (2%) on a few patches on the scalp, mustache, or eyebrows in AA for at least 6-12 weeks. Topical tofacitinib may benefit recalcitrant psoriasis patches following phototherapy, biologics, or cyclosporine. Systemic tofacitinib can be considered for AD, but local remnant patches need to be treated with the

maintenance dose. It is effective for early-stage vitiligo (<10% BSA) and can be applied for 12 to 16 weeks with NB-UVB.

Additionally, the acceptance rate of tofacitinib ointment is very high, owing to its tolerability. Experts recommend topical tofacitinib along with oral or phototherapy in AA and vitiligo. Experts came to a common consensus that topical use on limited BSA does not exhibit AEs as seen with the systemic route. Some common AEs include upper respiratory infections, nasopharyngitis, and urinary tract infections if systemic absorption occurs. For larger BSA and longer duration, decreased neutrophil counts, reduced hemoglobin, and increased mean cholesterol levels can be expected.

## 6. Conclusions

Tofacitinib, a JAK inhibitor, prevents JAK phosphorylation and STAT activation. It has found off-label application in autoimmune skin diseases like AD, psoriasis, alopecia, and vitiligo. The topical formulation of tofacitinib was developed to combat the systemic adverse effects of the drug and to achieve a targeted therapy. Topical tofacitinib (2%), BID, is usually the recommended dosage for autoimmune skin diseases with minimal AEs. Some limitations associated with topical tofacitinib include lack of awareness, standardization via national clinical practice guidelines, regulatory approvals, and non-adherence to therapy.

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None.

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