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Literature Review

A New Approach to Symptom Relief in Atopic Dermatitis: The Role of Upadacitinib

Humaira Shamim[1], Maryam Raza[2], Muhammad Hassaan Zia[3],
Usman Rasheed[4], Asad Ullah Khan[5] and Nadia Ali Azfar[6]
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dr.homaira@gmail.com

Abstract:

Background

Atopic dermatitis (AD) is a common, chronic inflammatory disease that affects millions of individuals worldwide. It is characterized by intense itching, recurrent eczematous lesions, and a relapsing-remitting course, all of which significantly impair quality of life. Conventional treatments, including topical corticosteroids, calcineurin inhibitors, and systemic immunosuppressants, often provide only partial relief or carry risks of adverse effects with prolonged use. The limited efficacy and safety concerns associated with existing therapies have driven interest in targeted approaches that address the underlying immune dysregulation. One such strategy involves inhibition of the Janus kinase (JAK)–signal transducer and activator of transcription (STAT) pathway, which plays a key role in transmitting inflammatory cytokine signals involved in AD pathogenesis. Upadacitinib, a selective JAK1 inhibitor initially approved for immune-mediated conditions such as rheumatoid arthritis and inflammatory bowel disease, has recently gained approval for moderate-to-severe AD, representing a promising addition to the list of current treatment options.

Methods

This review is based on evidence from published clinical trials and comparative studies assessing the efficacy and safety of Upadacitinib in patients with moderate-to-severe atopic dermatitis. While relevant literature is discussed, specific details about the search strategy and inclusion/exclusion criteria were omitted for the sake of brevity, which limits the methodological transparency typically expected in systematic reviews.

Results

Clinical trials and comparative studies have demonstrated that Upadacitinib offers a quicker and more effective response than other JAK inhibitors and various drug classes. However, certain limitations—such as small sample sizes, short follow-up durations, and differences in study populations—must be considered before drawing definitive conclusions. The use of Upadacitinib may also be restricted due to its rare but potentially serious adverse effects, which necessitate caution in specific patient populations. Periodic laboratory monitoring may be necessary, and regulatory agencies have issued warnings about the potential, sometimes fatal, adverse effects of prolonged use.

Conclusion

Despite these concerns, Upadacitinib remains a valuable addition to the treatment options available for refractory atopic dermatitis and other dermatological conditions, highlighting its broad therapeutic potential. However, further research is needed to establish its long-term safety and effectiveness in real-world settings. The limitations of current studies should be addressed in future research.

Keywords: Upadacitinib, Atopic Dermatitis, JAK Inhibitors, Eczema Treatment, Inflammatory Skin Disease, Clinical Efficacy and Safety.

Introduction:

In the United States, the one-year prevalence of Atopic Dermatitis was reported at 12.98% among children (2007–2008) and ranged from 7.2% to 10.2% in adults (2010–2012) [1]. Atopic dermatitis, also known as Eczema, is a common lifelong skin condition characterized by erythematous, itchy, eczematous lesions [2,3]. Skin Lesions can range from pruritic, red, scaly patches to fluid-filled vesicles to thickening and lichenification of the skin.

The pathogenesis of AD is a widely studied phenomenon that involves multiple factors, including genetic, environmental, and unregulated immune responses [2,4]. It may begin with a genetically preordained flaw in the skin barrier, leading to the activation of pro-inflammatory cytokines and antigen-presenting cells, such as Th2 and Th22. Interleukins such as IL-4 and IL-

13 lead to the mobilization of eosinophils and mast cells, and the secretion of IL-31 causes itchiness [5].

Symptoms of painful, itchy lesions may result in sleep deprivation and deteriorating performance in school and may lead to multiple limitations on clothing choices, using soaps and shampoos, keeping pets, and swimming in chlorinated water, which may irritate the skin and trigger acute flares [6].

Due to its persistent nature, various treatment options have been introduced, including topical steroids, topical calcineurin inhibitors, and biologics. The mainstay of treatment is the use of emollients and the avoidance of factors that aggravate the condition. Lately, Janus Kinase (JAK) inhibitors have been gaining popularity for treating atopic Dermatitis. Upadacitinib is a Janus Kinase inhibitor commonly used to treat rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, and inflammatory bowel disease [7]. The inhibition of JAK suppresses cytokine signaling in crucial inflammatory pathways, making it a viable option for treating atopic dermatitis as well [8].

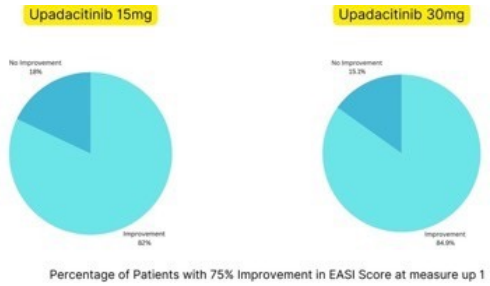
While these developments mark progress, further investigation is warranted to evaluate the long-term safety, efficacy, and real-world application of Upadacitinib in patients with Atopic Dermatitis. This study aims to address these gaps and contribute to the development of evolving therapeutic strategies for the condition.

Discussion:

Upadacitinib is a Janus kinase inhibitor with more excellent selectivity for JAK 1 than other JAK isoforms [9]. Inhibition of JAK leads to inhibition of the JAK-STAT signaling pathway, which is one of the most integral signaling pathways downstream of cytokine receptors [10]. About 60 cytokines, including many interleukins, colony-stimulating factors (CSFs), hormone-like cytokines, and growth factors, use the JAK-STAT pathway as their chief mode of initiating transcription [11]. Th2 cytokines such as interleukin (IL)-4, IL-5, IL-13, IL-31, and thymic stromal lymphopoietin contribute to chronic inflammation and itchiness in atopic dermatitis through JAK-STAT signal transduction. Moreover, the JAK-STAT pathway contributes to regulating the epidermal barrier and peripheral nerve activity associated with the transduction of pruritus. Focusing on the JAK-STAT pathway may lead to the inhibition of these signals and yield therapeutic effects in patients with atopic dermatitis [12].

Efficacy in Clinical Trials

A retrospective cohort study conducted between July 2021 and August 2022 analyzed the effectiveness and tolerance of Upadacitinib for severe atopic dermatitis in 29 adolescents and adults with a median follow-up of 54.4 weeks. At the end of the follow-up, 23 patients (79.3%) achieved either full or almost complete clearance, and 24 patients (82.7%) achieved improvement of at least 75% on the Eczema Area and Severity Index (EASI). It also proved effective in patients with treatment-resistant disease following the failure of biologics or Baricitinib [13]. Numerous Meta-analyses published in 2023 and 2024 analyzed multiple randomized controlled trials and found that Upadacitinib drastically improved signs and symptoms in patients with moderate to severe atopic dermatitis. Notably, 30 mg of Upadacitinib proved more efficacious than 15 mg of Upadacitinib [14]. However, most of these studies had a small number of patients, with some having already undergone treatment with other drug agents like immunosuppressants and immunomodulators, which may have contributed to better patient outcomes [15,16].



Percentage of Patients with 75% Improvement in EASI Score at measure up 1

Simpson EL, Papp KA, Blauvelt A, Chu CY, Hong HC, Katoh N, Calimlim BM, Thyssen JP, Chiou AS, Bissonnette R, Gold LF. Efficacy and safety of upadacitinib in patients with moderate to severe atopic dermatitis: analysis of follow-up data from the measure up 1 and measure up 2 randomized clinical trials. JAMA dermatology. 2022 Apr 1;158(4):404-13.

Figure 1: Efficacy of Upadacitinib in Atopic Dermatitis [28].

Comparative Effectiveness

Other studies have also been conducted to compare the efficacy of JAK inhibitors with anti-interleukin-receptor antibodies such as Dupilumab for atopic dermatitis. Dupilumab works against two specific interleukins, IL-4 and IL-13, in atopic conditions [17,18,19]. A meta-analysis published in 2023 in Heliyon demonstrated that improvement in EASI-75 was rapidly evident with Abrocitinib and Upadacitinib compared to Dupilumab, with noticeable distinction as early as week 2 of treatment. More patients achieved EASI-75 and EASI-90 at week 12 and the end of therapy. EASI (Eczema area and severity index) defines the severity of atopic dermatitis based on clinical signs and the percentage of body surface area affected. An EASI-75 score is at least a 75% reduction from baseline in EASI response. Similarly, the EASI-90 score is at least a 90% reduction from the baseline in EASI response. IGA response (Investigator's Global Assessment grade number), which ranges from worst (4 points) to the best (0 points), that evaluates the severity of atopic dermatitis, was more rapid and remained superior throughout

treatment with Abrocitinib/Upadacitinib. Pruritus was also more controlled by week 2 [20,21]. While this indicates that Upadacitinib may be superior to Dupilumab in achieving a rapid relief response, several factors must be considered. However, the 12-week duration of the study does not assess the efficacy of Upadacitinib for long-term disease control, whereas Dupilumab has been proven safer for long-term use [22].

Comparative assessments of the safety and efficacy of Upadacitinib with other JAK inhibitors, such as Abrocitinib and Baricitinib, have also shown that Upadacitinib is superior. Network meta-analysis has suggested that Upadacitinib 30 mg achieves higher IGA and EASI response than other regimens. Moreover, Upadacitinib 15 mg also considerably increased IGA or EASI response compared with Abrocitinib and Baricitinib. But it also concluded that Upadacitinib 30 mg caused more treatment-emergent adverse events (TEAEs) [23].

Adverse Effects and Safety Profile

Many adverse effects have also been reported where JAK inhibitors have proved to be very beneficial for treating atopic dermatitis—these range from standard to severe. Common adverse effects include acne, cough, headache, nasopharyngitis, oral herpes, and upper respiratory tract infections. Severe adverse effects, although rare, include infections such as herpes zoster, herpangina, tuberculosis reactivation, venous thromboembolism, cardiovascular events, and increased risk of malignancy [24,25]. The US Food and Drug Administration (FDA) placed a black box warning on all approved JAK inhibitors in 2021, but did not include the novel Tyk2 inhibitors such as Deucravacitinib. Hence, the question arises whether Tyk2 inhibitors such as Deucravacitinib are a potentially safer alternative because they do not inhibit JAK1/2/3 [26]. Similarly, the European Medicines Agency (EMA) and Health Canada have ordered boxed warnings for the entire class of oral JAK inhibitors. This decision was based on safety findings from clinical trials, particularly a post-marketing study of tofacitinib in rheumatoid arthritis, which showed raised risks of these adverse events [27].

An analysis of follow-up Data from the Measure Up 1 and Measure Up 2 randomized clinical trials published in JAMA Dermatology followed the efficacy and safety of Upadacitinib used in patients with moderate to severe atopic dermatitis. In the combined studies, the exposure-adjusted event rate (EAER, events/100 patient years [PYs]) of serious infections was higher with Upadacitinib 30 mg (3.6/100 PYs) compared to 15 mg (2.2/100 PYs). The EAER of opportunistic infections (excluding tuberculosis and herpes zoster) was similar between groups (1.9/100 PYs for 15 mg vs. 2.0/100 PYs for 30 mg). Deep vein thrombosis, pulmonary embolism, and stroke were reported, with all events classified as serious but considered unconnected to the study drug. The EAER of cancer was 0.6/100PYs with Upadacitinib 15mg and 0.9/100PYs with Upadacitinib 30 mg. Nonmelanoma skin cancer (NMSC) was the most reported in both treatment groups [28].

Clinical Considerations and Contraindications

Patients with atopic dermatitis are usually started on topical corticosteroids and are leveled up if unresponsive to the initial treatment. Upadacitinib can be given to patients 12 years or older, but only with recalcitrant, moderate-to-severe disease. However, a thorough history and physical exam should be performed to evaluate patients who may have contraindications to the use of the drug. Laboratory investigations may include a Complete blood count, Lipid profile, D-dimer levels, Hepatitis B and C antibodies, Liver and kidney function tests, Tuberculosis screening, HIV screening, etc. Patients should also be periodically monitored to assess for any undesirable effects that may require discontinuation of the drug. [29]. Patients with moderate to severe renal impairment may require a dosage reduction by half or more of the original dose, but no dosage adjustment is needed for mild impairment [30]. Patients with severe hepatic impairment are advised not to use Upadacitinib (Child-Pugh C), but dosage adjustment is not necessary for those with mild or moderate impairment (Child-Pugh A and B) [31]. There is limited data on the use of newer systemic therapies such as JAK inhibitors for the treatment of atopic dermatitis in pregnant and lactating women, which can make managing atopic dermatitis difficult as there are no extensive clinical studies on the potential impact and adverse reactions of JAK inhibitors on female fertility, conception, pregnancy and lactation [32]. Before starting any immunomodulatory and immunosuppressive medication, a vaccination history should be thoroughly reviewed to recommend mandatory age-appropriate vaccinations. For patients on JAK inhibitors, these include yearly Influenza, pneumococcal, and Shingrix vaccinations [33].

Wider Dermatological Applications

JAK inhibitors have demonstrated promising results in their application to treat various other dermatological conditions. This includes alopecia areata, an autoimmune condition that targets hair follicles and results in hair loss [34]. Tofacitinib, which was initially approved for rheumatoid arthritis, has shown effectiveness in treating psoriasis. In the Phase I trial, 59 adults with mild-to-moderate psoriasis received tofacitinib for 14 days. Significant improvements were seen at 30 and 50 mg twice daily ($p < 0.01$). In Phase II, 67% of patients on 15 mg twice daily achieved PASI 75 (Psoriasis Area and Severity Index) at week 12 ($p < 0.0001$), compared to only 2% with placebo. Phase III results showed PASI 75 in 63.6% of patients on 10 mg twice daily, surpassing etanercept (58.8%) and placebo (5.6%) [35]. Ruxolitinib has also been approved for

treating atopic dermatitis [36]. JAK inhibitors, which block IFN-γ signaling, have become popular for treating vitiligo because they help improve pigmentation. The most commonly reported JAK inhibitors used in vitiligo treatment are Ruxolitinib, Tofacitinib, and Baricitinib. A double-blind Phase II trial (NCT03099304) with 157 patients showed that those using 1.5% Ruxolitinib cream (twice daily, once daily) and 0.5% once daily had significantly better results, achieving F-VASI 50 (Facial-Vitiligo Area Scoring Index) at week 24. At week 52, these groups maintained significant repigmentation and showed good tolerance, suggesting topical Ruxolitinib as an effective vitiligo treatment [37]. Numerous case reports and case series have documented the safety and efficacy of Upadacitinib, Tofacitinib, Baricitinib, and Ruxolitinib in treating various lichen planus (LP) variants. The most common variants that JAK inhibitors improved were lichen planopilaris, nail lichen planus, and erosive lichen planus [38,39]. Cases have been reported in patients with refractory amyopathic dermatomyositis successfully managed with Upadacitinib [40,41]. In recent years, the interest in using JAK inhibitors to treat Hidradenitis Suppurativa (HS) has increased. However, there still isn't much data to understand the effectiveness of JAK inhibitors for HS. Only one clinical trial was published in the literature (Janus kinase 1 inhibitor INCB054707), a study with 15 patients up to week 24 with Upadacitinib, and a case series where Tofacitinib was effectively used. Meanwhile, there are several ongoing clinical trials [42]. The JAK/STAT signaling pathway plays a fundamental role in rosacea, which is why it has shown promise in treating refractory rosacea. Their possible mechanisms of action may include suppressing the inflammatory response, modifying vascular permeability, inhibiting new blood vessel growth, and strengthening the skin's protective barrier [43].

Pharmacokinetics

In vitro metabolism studies suggested that Upadacitinib is a weak substrate for cytochrome P450 (CYP) 3A, with minimal metabolic contribution from CYP2D6 [44]. Potent CYP3A inhibitors lead to a moderate (75%) increase in Upadacitinib exposure, while rifampin, a CYP inducer, decreases Upadacitinib exposure by approximately 50% [45]. Therefore, Upadacitinib should be used cautiously in patients receiving long-term treatment with potent CYP3A4 inhibitors such as ketoconazole, and concomitant use with potent CYP3A4 inducers such as rifampin is not recommended [46].

Future Research Goals

Although Upadacitinib is an effective therapeutic option in cases of moderate to severe AD, further research is needed to determine its safety when used long-term, efficacy in specific populations (pediatric and pregnant), and its comparative performance with other JAK inhibitors and biologics.

Conclusion:

This paper examined the comparative efficacy, safety, and clinical application of Upadacitinib in the treatment of moderate to severe atopic dermatitis. The findings indicate that Upadacitinib demonstrates rapid symptom relief and greater improvement in disease severity scores (EASI-75, EASI-90, and IGA responses) compared to other JAK inhibitors and biologics. However, its use is accompanied by an increased risk of adverse events, particularly at higher doses, which highlights the need for cautious patient selection and monitoring.

By synthesizing current clinical evidence, this review highlights the significant therapeutic potential of Upadacitinib while also emphasizing the importance of proper patient selection for its use. This paper aims to provide clinicians with a better understanding of the benefit-risk profile and a framework for integrating it into existing treatment algorithms.

Future research should focus on defining long-term safety profiles, optimizing dosing strategies, and identifying patient subgroups most likely to benefit from treatment. Additionally, ongoing trials in other dermatologic and immune-mediated diseases may expand Upadacitinib's indications and further refine its clinical utility.

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