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Original Article

Effectiveness of Switching From Upadacitinib to Tralokinumab in Patients With Moderate-to-Severe Atopic Dermatitis: A Real-World Clinical Practice

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ABSTRACT

Background: Atopic dermatitis (AD) is a chronic eczematous disorder characterized by intense itchiness. Systemic therapies for AD include Janus kinase (JAK) inhibitors and various biological agents. The effects of transitioning from the JAK1 inhibitor, upadacitinib, to the anti-interleukin 13 antibody, tralokinumab, remain unclear.
Objective: This study evaluated the transition from 15 mg of upadacitinib to tralokinumab in patients with moderate-to-severe AD.
Methods: This analysis included 20 patients who switched from 15 mg of upadacitinib to tralokinumab due to an inadequate response or adverse events (AEs). We assessed the total and regional eczema area and severity index (EASI), which included assessments of the head and neck, trunk, and upper and lower limbs, along with erythema, edema/papulation, excoriation, lichenification, and the peak pruritus numerical-rating scale (PP-NRS), initially (start of 15 mg of upadacitinib), at the transition point (week 0), and during follow-up at weeks 4 and 12.
Results: The EASI, EASI of the four anatomical regions, and EASI of the four clinical manifestations significantly declined from baseline at weeks 4 and 12, with no substantial reductions from week 0. The PP-NRS score notably decreased from baseline at week 4. Achieving EASI of 50 and 75 improved post-switching.
Conclusion: Transitioning to tralokinumab substantially alleviated rash in patients with AD who experienced suboptimal responses or AEs to 15 mg of upadacitinib.

Keywords: Atopic dermatitis; Interleukin-13; Janus kinase 1; Real world clinical trials; Treatment switching

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by eczema and severe itching, with a variable disease course ranging from intermittent to persistent, particularly in severe cases[1,2]. Patients with AD often show a notable decline in quality of life due to symptoms, such as itching, which demands effective management. Presently, three oral Janus kinase (JAK) inhibitors (upadacitinib, abrocitinib, and baricitinib) and four biological agents (dupilumab targeting the interleukin [IL]-4/13 receptor, the IL-13 specific antibodies lebrikizumab and tralokinumab, and nemolizumab targeting the IL-31 receptor) have received approval in Japan for systemic AD management[3]. The introduction of these potent therapies has transformed the

management of AD, emphasizing the need for clinicians to optimize their application in practice. Persistent research and trials have indicated that upadacitinib is exceptionally potent and tolerable for the management of skin inflammation and itching in individuals with significant AD[4-7]. Previous investigations have confirmed the safety and effectiveness of upadacitinib administered at 15 and 30 mg in Japanese individuals with severe AD[8-11]. Tralokinumab, a high-affinity human monoclonal antibody, targets IL-13 effectively, as previous study has shown.[12] Both and real-world studies and controlled trials have consistently supported its efficacy and safety in treating serious AD cases[13-19].

Network meta-analyses of AD treatments suggested that 15 and 30 mg of upadacitinib demonstrated superior efficacy compared to tralokinumab 300 mg in managing both rashes and pruritus[20].

In real-world practice, patients who exhibit an insufficient response to 15 mg of upadacitinib or experience adverse events (AEs) may opt to switch to biologics as alternative therapies. However, the effects of transitioning from JAK inhibitors to anti-IL-13 antibodies have yet to be thoroughly studied. Specifically, the clinical outcomes associated with switching from 15 mg of upadacitinib to tralokinumab have not been explored in real-world settings or clinical trials.

This study was designed to evaluate the outcomes of the transition from 15 mg of upadacitinib to tralokinumab among Japanese patients with AD in a real-world clinical setting, with the anticipation of balanced safety and therapeutic benefits.

MATERIALS AND METHODS

Data collection and study design

Twenty Japanese individuals, aged ≥15 years with moderate-to-severe AD, participated in this study at our institution between August 2021 and April 2024. The diagnosis was based on the criteria set in the Japanese Atopic Dermatitis Guidelines 2021[21]. All the patients were initially prescribed 15 mg of upadacitinib orally once daily in combination with topical corticosteroids, applied twice daily, from the moderate to the strongest class, for a median duration of 12.75 months (range: 7.0–16.75 months). Following this, patients were transitioned to biweekly subcutaneous injections of tralokinumab at 300 mg, starting with an initial dose of 600 mg, without modifications to their existing topical treatments. This transition was prompted by either a lack of an adequate response or AEs during the upadacitinib treatment phase. The response to the 15 mg dose of upadacitinib was evaluated after at least 12 weeks of treatment.

Inclusion and exclusion criteria

Inclusion was contingent upon a diagnosis of moderate-to-severe AD, defined by an eczema area and severity index (EASI) score of ≥16, or a minimum EASI score of 2.4 in the head and neck area. Transition to tralokinumab was based on inadequate control of symptoms or AEs associated with upadacitinib treatment, without altering the topical regimen. The following inclusion criteria were used: AEs leading to discontinuation of upadacitinib, an EASI score >16, or an EASI score at the head and neck surpassing 2.4. Consent for switching to tralokinumab was obtained from all the participants. Patients whose upadacitinib dose was decreased from 30 to 15 mg, or who showed an insufficient response to the 30 mg dose were excluded. Additionally, patients who had been previously treated with both 15 mg of upadacitinib and dupilumab, among other therapies, were excluded from this study.

Therapeutic effectiveness

The EASI scores, including scores for four specific body regions (the head and neck, upper limbs, lower limbs, and trunk) and scores for four clinical manifestations (erythema, edema/papulation, excoriation, and lichenification), were assessed at the start of treatment with 15 mg of upadacitinib (baseline), at the moment of transition (week 0), and during weeks 4 and 12 following the switch. The severity of each clinical manifestation, as per the EASI, was evaluated using a scale from 0 to 3, with corresponding area scores ranging from 0 to 6. Key biomarkers, such as thymus and activation-regulated chemokine (TARC), immunoglobulin E (IgE), lactate dehydrogenase (LDH), and total eosinophil count (TEC) were quantified at the initial assessment and at weeks 0, 4, and 12. We calculated the percentage of patients achieving a 50%, 75%, or 90% reduction in their EASI scores from the baseline values (EASI 50, EASI 75, or EASI 90, respectively) at these intervals. Additionally, we determined the proportion of participants who attained a minimum improvement of four points on the peak pruritus numerical-rating scale (PP-NRS 4) at the same intervals.

Safety

Safety monitoring focused on recording treatment-emergent AEs (TEAEs) from the initiation of 15 mg of upadacitinib treatment for 30 days after the last tralokinumab dose. Treatment-emergent AEs were defined as any newly appearing AEs or worsening AEs after treatment initiation.

Ethic statement

All the participants provided written informed consent in accordance with the ethical standards stipulated in the Declaration of Helsinki (2004), and the protocol was approved by the Ethics Committee of Nippon Medical School Chiba Hokusoh Hospital (protocol code H-2023-123, approved on April 25, 2024).

Statistical analysis

Data are displayed as medians and interquartile ranges for

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nonparametrically distributed variables. We conducted the Friedman's test to compare data across multiple time points (baseline, week 0, week 4, and week 12) for both EASI and PP-NRS scores, as well as for comparisons across different anatomical regions and clinical signs (such as erythema, edema/papulation, excoriation, and lichenification). Bonferroni adjustments were applied to control for type I errors across these multiple comparisons. Statistical significance was defined as a p-value <0.05 after Bonferroni correction. All statistical analyses were performed using EZR (Easy R), a graphical user interface for R developed by the Saitama Medical Center, Jichi Medical University, Saitama, Japan, which was selected for its robust handling of non-parametric data and accessibility.

RESULTS

Patient demographics and baseline characteristics

Table 1 summarizes baseline patient characteristics. The reasons for switching therapies included higher EASI scores in 11 (55%) patients and AEs in 9 (45%). **Supplementary Table 1** provides detailed information on each patient's demographic and clinical backgrounds, including comorbidities, prior therapies, and AEs associated with upadacitinib.

Table 1. Baseline characteristics and demographics of patients with atopic dermatitis

Variables	Values
Male sex	16 (80.0)
Age (yr)	54 [45–59.3]
<18	1 (5.0)
Body mass index (kg/m^2)	23.4 [20.8–25.0]
Disease duration (yr)	43 [31.5–48.5]
Clinical indicators	
Whole EASI	24.3 [17.9–36.9]
EASI at face and neck	2.4 [1.2–3.8]
EASI at upper limbs	4.8 [3.8–8.0]
EASI at lower limbs	9.6 [7.2–13.6]
EASI at trunk	7.2 [6.2–11.5]
EASI of erythema	5.5 [3.2–8.1]
EASI of edema/papulation	5.5 [3.2–8.2]
EASI of excoriation	6.9 [4.4–8.9]
EASI of lichenification	7.4 [5.5–8.8]
Peak pruritus-numerical rating score	10 [7–10]
Laboratory parameters	
Immunoglobulin E (IU/mL)	1,379.0 [1,286.3–2,103.0]
Thymus and activation-regulated chemokine (pg/mL)	1,723.0 [1,180.3–3,630.3]
Lactate dehydrogenase (IU/mL)	254.0 [223.0–272.0]
Total eosinophil count (/mL)	452.4 [423.0–540.0]
Reasons for switching	
Total EASI ≥16 or head and neck EASI ≥2.4	11 (55.0)
Adverse events	9 (45.0)

Data provided as number (%) or the median [interquartile range].
EASI: eczema area and severity index.

Effectiveness of whole EASI and PP-NRS post-switch from 15 mg of upadacitinib to tralokinumab

Initially, there was a slight reduction in the whole EASI scores following treatment with 15 mg of upadacitinib at week 0 (**Supplementary Fig. 1A**), although this change was not statistically significant. Following the transition to tralokinumab, a marked reduction in the whole EASI was noted at weeks 4 and 12 compared to baseline levels. Although the PP-NRS scores (**Supplementary Fig. 1B**) decreased slightly at the time of switching, this change was not statistically significant. By week four, PP-NRS scores had significantly decreased relative to baseline, yet showed no notable change from week 0. By week 12, neither the whole EASI nor the PP-NRS scores exhibited significant deviations from baseline or week 0 levels. Significant alleviation in rash severity and a moderate but significant reduction in pruritus were observed following the switch to tralokinumab. In this study, we assessed the median percentage reduction in total EASI and PP-NRS scores from baseline to 12 weeks after transitioning from upadacitinib to tralokinumab treatment. **Supplementary Fig. 2** illustrates these reductions. By week 12, the median percentage reduction in total EASI was 75%, indicating an improvement in EASI. The median percent reduction in PP-NRS was 45%, reflecting an improvement in pruritus, although to a lesser extent than in skin symptoms.

Achieving EASI 75, EASI 90, and PP-NRS 4 after switching from 15 mg of upadacitinib to tralokinumab

Post-transition, the percentages of patients achieving EASI 50, 75, and 90 showed a progressive increase from baseline to week 12. Specifically, achieving an EASI score of 50 escalated from 47.4% at baseline to 64.7% at week four, reaching 75% at week 12. To achieve EASI 75, the rates increased from 17.6% at baseline to 20% at week four, and further to 36.4% at week 12. In contrast, achieving EASI 90 was initially 0% at both baseline and week four, but improved to 9.1% by week 12, as depicted in **Fig. 1**. The achievement of PP-NRS 4 also exhibited an upward trajectory from baseline to week four, with recorded percentages of 42.1% at baseline, 52.9% at week four, and a slight decline to 41.7% by week 12. The peak in achieving PP-NRS 4 observed at week four plateaued. Although achieving EASI 90 was initially stable, it only increased at week 12, highlighting a delayed but positive response to tralokinumab treatment over time.

Improvement in EASI at different anatomical regions after switching from 15 mg of upadacitinib to tralokinumab

Initial assessments showed that the EASI values in four anatomical regions (the head and neck, trunk, upper limbs, and lower limbs) did not exhibit significant changes at week 0 post-initiation of upadacitinib treatment compared to those at baseline (**Fig. 2**).

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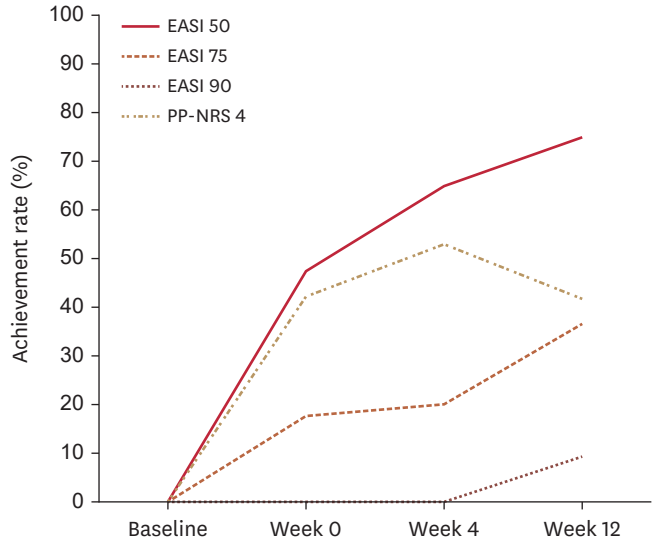

Fig. 1. Achievement rates of EASI 50, EASI 75, EASI 90, and PP-NRS 4 during treatment with 15 mg of upadacitinib and after switching to tralokinumab in patients with atopic dermatitis (n=20).
EASI: eczema area and severity index, PP-NRS: peak pruritus-numerical rating scale.

However, following the transition to tralokinumab treatment, notable reductions in EASI scores were observed across all sites at 4 and 12 weeks, relative to the initial baseline values (**Fig. 2**).

Specifically, EASI measurements in the head and neck regions showed a marked decrease by week 12 relative to their values at week four (**Fig. 2A**). Similarly, the scores for the upper limbs demonstrated a significant reduction at week12 when compared to those recorded at week 0 (**Fig. 2C**).

Improvement of EASI for clinical manifestations after switching from 15 mg of upadacitinib to tralokinumab

Initial evaluations demonstrated that the EASI scores for four clinical manifestations (erythema, edema/papulation, excoriation, and lichenification) remained largely unchanged at week 0 compared to baseline values (**Fig. 3**). Subsequent assessments post-transition to tralokinumab revealed significant reductions in these scores by weeks 4 and 12 relative to the baseline (**Fig. 3**). Notably, by week 12, there was a pronounced decline in all four clinical manifestations compared with week 0 values (**Fig. 3**).

By four weeks post-switching, the severity of erythema had substantially diminished from baseline (**Fig. 3A**). By week 12, the severity of lichenification also showed a significant reduction compared to that at week 4 (**Fig. 3D**).

No marked changes were observed in the scores of other clinical manifestations between weeks 4 and 12, indicating stable post-switch improvement.

Transition of laboratory parameters after switching from 15 mg of upadacitinib to tralokinumab

After switching to tralokinumab, there was a marked decrease in both IgE (**Fig. 4A**) and TARC (**Fig. 4B**) levels, which were notably lower at weeks 4 and 12 than those at week 0. In contrast, LDH levels, did not show significant fluctuations during any of the assessed time points (**Fig. 4C**). The TEC displayed a significant increase, being considerably higher at weeks 4 and 12 than the baseline values (**Fig. 4D**).

Safety

Throughout the course of treatment with 15 mg of upadacitinib, 29 TEAEs were reported in 15 patients, affecting 75% of the cohort. Additionally, nine AEs necessitated discontinuation of the drug in nine patients, accounting for 45.0% of cases (**Supplementary Table 2**). The AEs that prompted discontinuation included herpes labialis in two (10.0%) patients, herpes zoster in one (5.0%), Kaposi's varicelliform eruption in one (5.0%), peritonsillar abscess in one (5.0%), and anemia in four (20.0%).

Although three patients with herpes labialis and one with Kaposi's varicelliform eruption improved with antiviral treatment, two switched to tralokinumab due to multiple recurrences. Two patients with herpes zoster also improved with antiviral treatment. The patient's peritonsillar abscess improved with antibiotic treatment and hospitalization. The anemia in four patients revealed a decrease in hemoglobin (Hb) levels ≤9 g/dl, which led to the discontinuation of the 15 mg upadacitinib. After switching to tralokinumab, Hb levels normalized in two patients, and the other two patients were clinically assessed to have non-severe and manageable anemia.

During treatment with 15 mg of upadacitinib, elevated serum creatinine phosphokinase levels occurred in seven (35.0%) patients, nausea in one (5.0%), upper respiratory tract infection in one (5.0%), acne in four (20.0%), and headache in one (5.0%). All these conditions were mild and resolved spontaneously. Additionally, tinea corporis occurred in one (5.0%) patient, lupus miliaris disseminatus faciei in one (5.0%), arthritis in one (5.0%), and cystitis in one (5.0%), all of which improved with appropriate treatment.

After switching to tralokinumab, six TEAEs occurred in four (20.0%) patients, with no AEs leading to discontinuation or death, or serious AEs. During tralokinumab treatment, herpes labialis was observed in two (10.0%), which improved with appropriate treatment. Blepharitis was observed in one (5.0%) patient, COVID-19 infection in two (10.0%), and acne in one (5%), all of which were mild and resolved spontaneously. Conjunctivitis was not observed during the tralokinumab treatment.

Among the 15 patients (29 cases) who discontinued upadacitinib due to AEs, all AEs were resolved following the switch to

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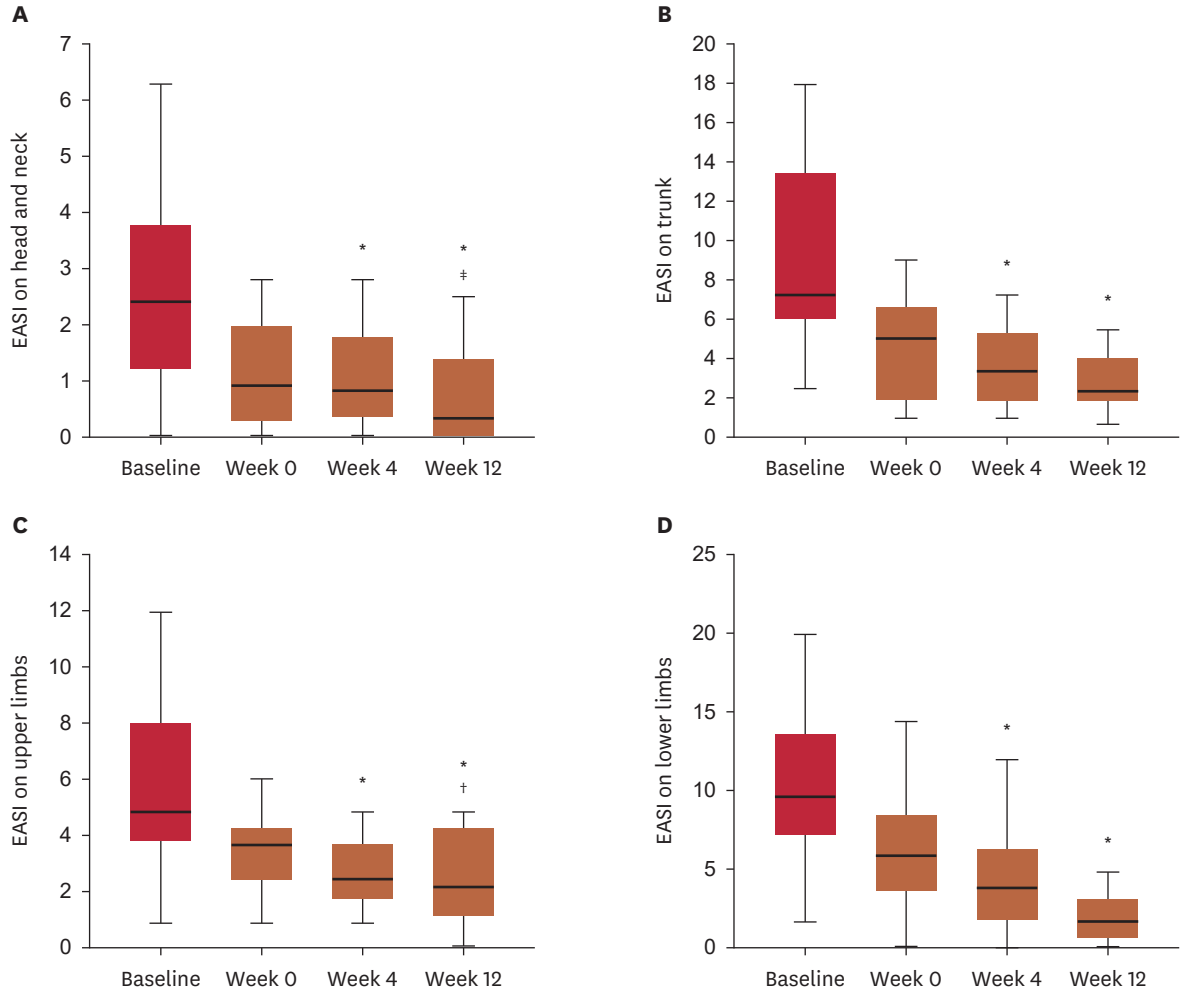

Fig. 2. Improvement in EASI scores at the head and neck (A), upper limbs (B), lower limbs (C), and trunk (D) after switching from 15 mg of upadacitinib to tralokinumab in patients with atopic dermatitis (n=20). Data shown as box plots are median [interquartile range]; upper and lower points are maximal and minimal values, respectively.
EASI: eczema area and severity index.
*p<0.05 vs. values at baseline; †p<0.05 vs. values at week 0; ‡p<0.05 vs. values at week four, according to the Friedman's test with Bonferroni post-hoc test.

tralokinumab. However, three patients (three cases) experienced a recurrence of AEs post-switch: one case of acne and two cases of herpes labialis. All recurrences were mild, with acne resolving spontaneously and herpes labialis improving with appropriate antiviral treatment. No patients experienced worsening of AEs after switching to tralokinumab.

DISCUSSION

In the current study, we aimed to clarify the outcomes of switching to the IL-13 antibody, tralokinumab, in patients with AD who did not achieve sufficient therapeutic effects or showed AEs with 15 mg of upadacitinib. We found that switching to tralokinumab significantly improved rashes, but had a limited effect on pruritus. Although network meta-analyses have shown that tralokinumab has lower efficacy against rashes and pruritus at 12 or 16 weeks compared to 15 mg of upadacitinib[20], our study demonstrated significant improvements in rashes after switching from upadacitinib to tralokinumab. Therefore, based on our results, we can infer that tralokinumab might still be a viable option for improving rashes in patients who do not respond to15 mg of upadacitinib.

Previous studies support our findings. A real-world clinical study involving 37 patients with AD who had inadequate responses or AEs with dupilumab or various JAK inhibitors showed that 22 (59%) patients responded to tralokinumab treatment[18]. In this study, a responder to tralokinumab was identified by any reduction in investigator's global assessment or NRS itch scores, along with patient satisfaction and a willingness to continue the treatment. The results of this investigation indicate that tralokinumab may be

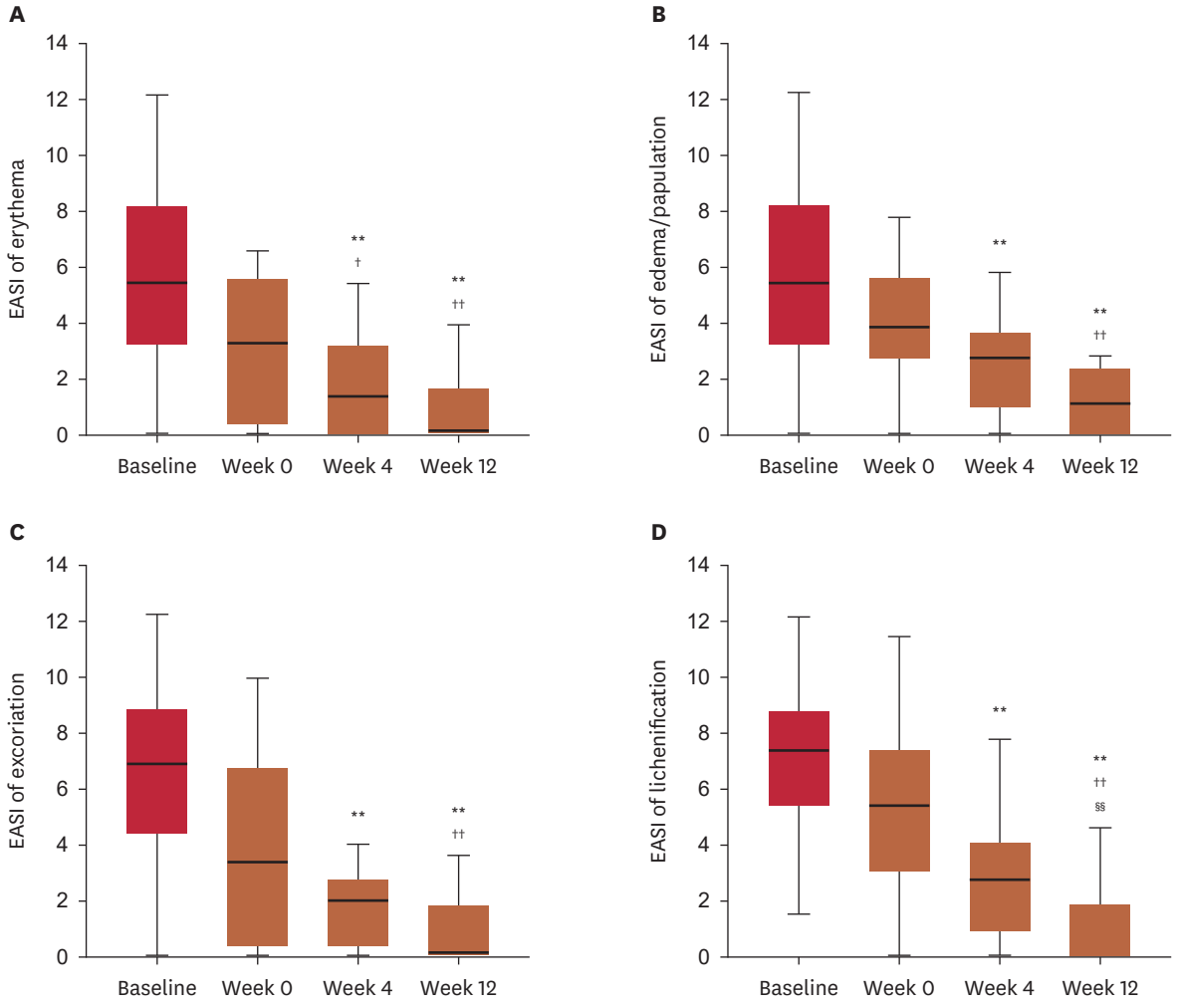

Fig. 3. Improvement of eczema area and severity index (EASI) scores of erythema (A), edema/papulation (B), excoriation (C), or lichenification (D) after switching from 15 mg of upadacitinib to tralokinumab in patients with atopic dermatitis (n=20). Data shown as box plots are median [interquartile range]; upper and lower points are maximal and minimal values, respectively.
$^{**}p<0.01$ vs. values at baseline; $^{\dagger}p<0.05$, $^{\dagger\dagger}p<0.01$ vs. values at week 0; $^{\S\S}p<0.01$ vs. values at week four, according to the Friedman's test with Bonferroni post-hoc test.

effective in patients who do not respond well to other treatments.

In the present study, we observed that the anti-pruritic effect of tralokinumab was less pronounced than its effect on rashes. Previous studies have shown that cytokines, such as thymic stromal lymphopoietin, IL-4, IL-13, and IL-31, are key mediators of pruritus in AD, and JAK inhibitors that block the effects of multiple cytokines can effectively reduce pruritus[22], while tralokinumab only inhibits the effect of IL-13. The lower improvement of pruritus by tralokinumab compared to that of rash may be because IL-13 may only partially contribute to pruritus, while the rash may be highly dependent on IL-13 in AD. In addition, our study demonstrated an increase in PP-NRS 4 achievement rates from baseline to week 4, rising from 42.1% to 52.9%, followed by a slight decline to 41.7% at week 12. Achieving PP-NRS 4 represents a reduction in pruritus by 4 or more points from baseline, a threshold considered the Minimal Clinically Important Difference. This improvement suggests that tralokinumab may contribute to enhancing patients' quality of life by significantly alleviating pruritus.

We observed an improvement in the head and neck EASI scores after transitioning from 15 mg of upadacitinib to tralokinumab. It is well known that AD rashes in these areas, particularly on the face, tend to be resistant to treatment[23], especially to therapies, such as dupilumab or JAK inhibitors[10,11,24,25]. Factors, such as exposure to pathogens like *Demodex and Malassezia*[26,27], along with constant exposure to environmental factors, such as airborne allergens, UV rays, and cosmetic products, contribute to their persistent nature. Previous studies have demonstrated that tralokinumab significantly reduced head and neck EASI scores in patients with AD at weeks 4 and 16, with no significant differences in the response between biologic-naïve and biologic-pretreated

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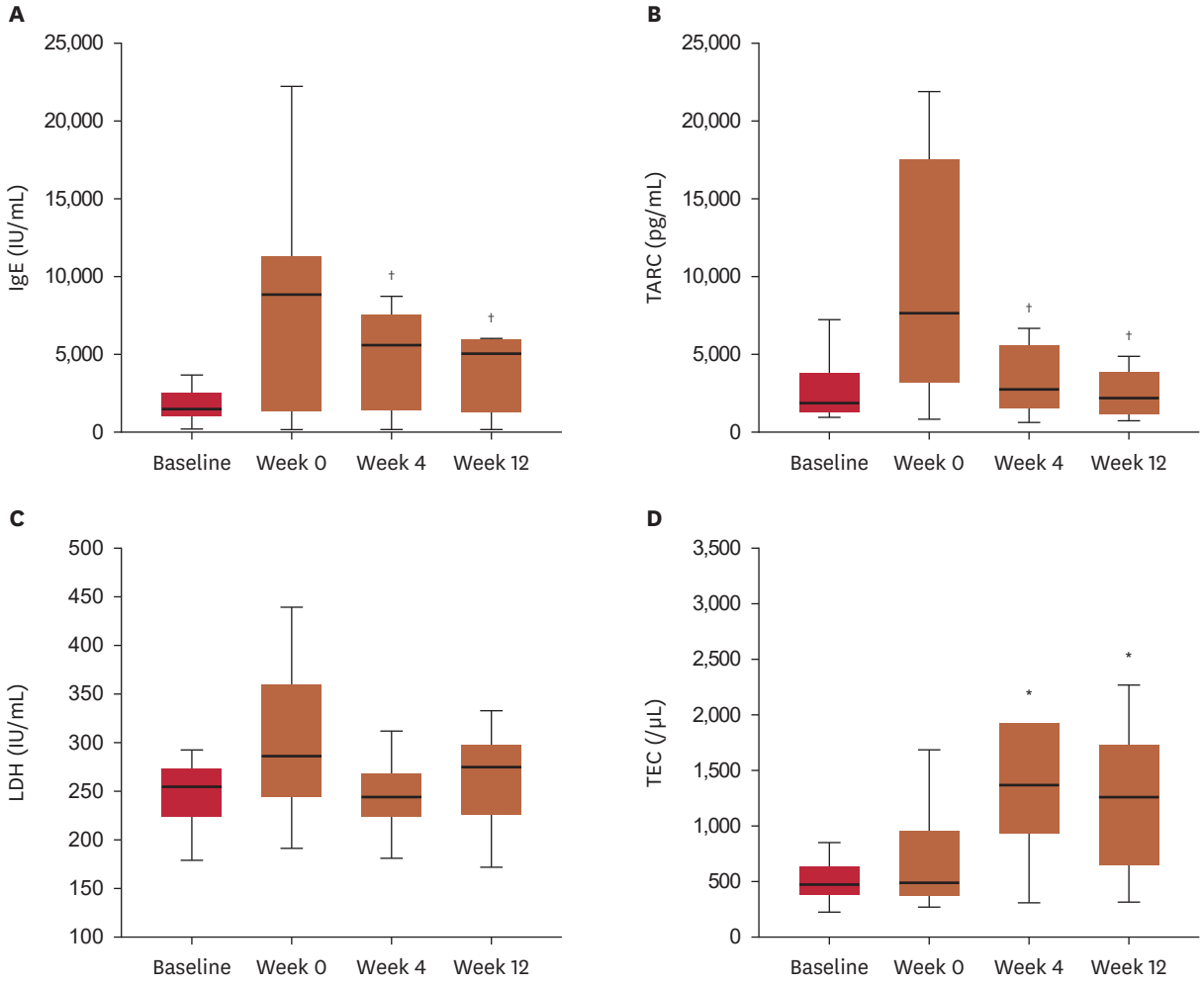

Fig. 4. Transition of IgE (A), TARC (B), LDH (C) and TEC (D) after switching from upadacitinib 15 mg to tralokinumab in patients with atopic dermatitis (n=20). The data shown as box plots are median [interquartile range]; upper and lower points are maximal and minimal values, respectively.
IgE: immunoglobulin E, TARC: thymus and activation-regulated chemokine, LDH: lactate dehydrogenase, TEC: total eosinophil count.
*p<0.05 versus values at baseline; †p<0.05 versus values at week 0, by Friedman's test with Bonferroni post-hoc test.

patients[28]. These findings suggest that tralokinumab may offer substantial therapeutic benefits for the treatment of head and neck rashes in patients with AD. However, the exact mechanism underlying the effectiveness of tralokinumab in the treatment of head and neck rashes remains unclear. In contrast, treatment with dupilumab, which blocks IL-4/13, has been associated with the development of erythema on the face and neck, which is potentially linked to *Demodex* in patients with AD[26]. Since tralokinumab specifically inhibits IL-13 but not IL-4, it may be a more favorable option for treating head and neck rashes. Further studies are required to better understand how these cytokines influence the regulation of rashes in patients with AD.

We found that IgE (**Fig. 4A**) and TARC (**Fig. 4B**) levels significantly declined at week 12 compared with those at weeks 0 and 4 of tralokinumab treatment. Previous clinical trials have also shown that tralokinumab treatment reduced IgE and TARC levels at weeks 12 or 16 compared to placebo[29,30]. The results are similar to those of dupilumab treatment in real-world studies[31].

Type 2 cytokines, such as IL-4 and IL-13, are involved in activating B cells and promoting immunoglobulin class switching to IgE[32]. By inhibiting the above effect of IL-13, tralokinumab may reduce serum IgE levels. Thymus and activation-regulated chemokine is a chemokine that attracts type 2 helper T cells to inflammation sites[33,34]. IL-13 acts on keratinocytes or dendritic cells to induce TARC expression[35]. The inhibition of the above effect of IL-13 by tralokinumab may lead to a reduction in TARC levels.

In this study, tralokinumab treatment increased TEC at weeks 4 and 12. Similarly, previous studies showed that treatment with anti-IL-13 antibodies, such as lebrikizumab and tralokinumab, or the IL-4Rα antibody dupilumab, prompted a transient rise in TEC among patients with AD[36-40]. IL-4 and IL-13 are known to stimulate the secretion of eosinophil-attracting chemokines, such as

CCL11 and CCL24 within fibroblasts, monocytes, or smooth muscle cells[41,42]. Blocking these cytokines with anti-IL-13 or anti-IL-4Rα antibodies can inhibit the production of these chemokines, leading to reduced tissue eosinophilia and retention of eosinophils in the blood, resulting in blood eosinophilia as revealed in asthma models using mice deficient in IL-4 and IL-13[43].

After switching from 15 mg of upadacitinib to tralokinumab, no serious AEs or AEs leading to discontinuation were observed. The safety profile observed in the current study aligned with that reported in the ECZTRA clinical trials, which revealed no new AEs[13,17]. The AEs in the present study were mild and manageable. Furthermore, a detailed analysis of AEs leading to upadacitinib discontinuation revealed that all reported AEs (29 events in 15 patients) were resolved or improved after switching to tralokinumab. Notably, three mild recurrences of AEs were observed post-switch, comprising one case of acne and two cases of herpes labialis, all of which were effectively managed with appropriate treatment. Importantly, no patients experienced worsening of AEs after the transition. These findings emphasize the potential of tralokinumab to mitigate AEs associated with upadacitinib, further supporting its safety and tolerability as a treatment option for patients with moderate-to-severe AD.

This study has a few limitations. First, this was conducted at a single facility and involved only a limited number of participants. Second, the duration of upadacitinib treatment varied across patients. Third, the study focused specifically on upadacitinib non-responders, which may limit the generalizability of the findings to a broader AD population. Patient baseline disease severity, comorbidities, and treatment history may influence responses to tralokinumab and should be considered when interpreting the results. Fourth, the abrupt discontinuation of upadacitinib can lead to early exacerbation of pruritus and, in some cases, worsening of the rash, particularly around the second week post-transition. To manage this transition period, we advised the patients to use potent topical corticosteroids, and most patients who experienced initial worsening showed improvement by week four. However, because of the high rate of missing clinical assessment data at week two, we were unable to include this information in our main analysis. This limitation highlights the importance of using early post-transition data in future studies. Finally, the tralokinumab treatment period was limited to 12 weeks, which may be relatively short for evaluating long-term outcomes.

In conclusion, tralokinumab treatment improved rash in patients with AD who did not adequately respond to or tolerate 15 mg of upadacitinib. These results indicate that switching to tralokinumab could be a viable treatment option for patients with AD with inadequate response or AEs related to 15 mg of upadacitinib.

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CONFLICTS OF INTEREST

H.S., T.H., and N. K. received lecture fees from LEO Pharmaceutical. The authors report no conflicts of interest.

DATA SHARING STATEMENT

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

SUPPLEMENTARY MATERIALS

Supplementary Table 1

Characteristics of atopic dermatitis patients transitioned from upadacitinib to tralokinumab

Supplementary Table 2

TEAEs during treatment with upadacitinib15 mg or after switching to tralokinumab in patients with atopic dermatitis (n=20)

Supplementary Fig. 1

The improvement of total eczema area and severity index (EASI) (A) and peak pruritus-numerical rating scale (PP-NRS) (B) after switching from upadacitinib 15 mg to tralokinumab in patients with atopic dermatitis (n=20). The data shown as box plots are median [interquartile range]; upper and lower points are maximal and minimal values, respectively.

Supplementary Fig. 2

Median percent reductions of total EASI and PP-NRS from baseline. The data are shown as median [interquartile].

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