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ORIGINAL RESEARCH

Real-World Effectiveness and Safety of Abrocitinib in Patients with Atopic Dermatitis: A 16-Week Single-Center Retrospective Cohort Study Compared with Upadacitinib and Baricitinib

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ABSTRACT

Introduction: Abrocitinib, a selective Janus kinase (JAK)-1 inhibitor, is approved for the treatment of moderate-to-severe atopic dermatitis (AD). Although several real-world studies evaluated the safety and efficacy of abrocitinib, most have been limited by small sample sizes, and there are limited data on South Korean patients with AD. In addition, real-world comparative data across oral JAK inhibitors for AD remain limited.

Methods: We conducted a retrospective, single-center cohort study at the National Medical Center in Seoul, Korea. Patients aged ≥ 12 years with moderate-to-severe AD (baseline EASI ≥ 7) who initiated abrocitinib between September 2022 and April 2024 were included in the primary cohort; additional cohorts treated with upadacitinib or baricitinib during predefined periods were analyzed for between-drug comparisons. Efficacy was assessed at baseline, week 2, and week 16 using the Eczema Area and Severity Index (EASI) and patient-reported outcomes (PROs). Safety was evaluated by adverse events (AEs), physical examinations, and laboratory tests.

Results: Of the 66 patients enrolled, 57 patients completed 16 weeks of abrocitinib treatment in the analysis. The mean EASI score significantly decreased after 16 weeks. At week 16, 94.4%, 72.2%, and 25.9% of patients with AD achieved EASI-50, -75, and -90, respectively. Additionally, of the 21 patients who had previously experienced biologics or other JAK inhibitors, 95.5%, 72.7%, and 22.7% achieved EASI-50, -75, and -90, respectively. Further analysis of the EASI breakdown showed improvements of more than 80% across all body regions, with the lower extremities showing the greatest reduction (87.5%) and lichenification exhibiting the highest symptom improvement (89.7%). In descriptive, unadjusted comparisons, abrocitinib showed numerically higher EASI-50 and EASI-75 response rates at week 16 than upadacitinib and baricitinib. Acne was the most frequent adverse event with abrocitinib (43.9%), followed by urticaria (24.6%) and herpes simplex infection

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(12.3%); no dose reductions or treatment discontinuations due to adverse events occurred.
Conclusion: Abrocitinib demonstrates real-world efficacy and safety in moderate-to-severe AD, including patients with inadequate responses to other dupilumab or JAK inhibitors, including those with prior exposure to biologics or other JAK inhibitors.

PLAIN LANGUAGE SUMMARY

Abrocitinib is an oral medicine approved for adults and adolescents with moderate-to-severe atopic dermatitis (AD). It is taken once daily (100 mg or 200 mg) and works by blocking a signaling pathway in the immune system called Janus kinase (JAK). Real-world evidence in South Korea and comparisons with other oral JAK inhibitors (upadacitinib and baricitinib) are limited. In this retrospective study, we reviewed the medical records of 66 Korean patients with moderate-to-severe AD who started abrocitinib at a single center, along with additional patients treated with upadacitinib or baricitinib during predefined periods at the same center. We assessed changes in disease severity scores and patient-reported symptoms over 16 weeks and recorded side effects and laboratory findings. Most patients receiving abrocitinib showed rapid improvement within 2 weeks, and these benefits were maintained through week 16, including in patients who previously had an inadequate response to dupilumab or another JAK inhibitor. The most common side effect was acne, followed by urticaria and herpes simplex infection, and no patients discontinued abrocitinib because of adverse events. These findings suggest that abrocitinib is an effective and generally well-tolerated treatment option for Korean patients with moderate-to-severe AD in routine clinical practice.

Keywords: Abrocitinib; Atopic dermatitis; JAK inhibitor; Real-world data

Key Summary Points

Why carry out this study?

Abrocitinib is an oral, once-daily, selective Janus kinase (JAK)-1 inhibitor approved for the treatment of adults and adolescents with moderate-to-severe atopic dermatitis (AD) and is available in doses of 100 mg and 200 mg.

Real-world evidence on abrocitinib in Korean patients is limited, and there are few data directly comparing different JAK inhibitors for AD in routine clinical practice.

This study aimed to evaluate the 16-week real-world effectiveness and safety of abrocitinib in Korean patients with moderate-to-severe AD, including those with inadequate responses to previous biologics or JAK inhibitors, and to compare outcomes with upadacitinib and baricitinib.

What was learned from the study?

Abrocitinib produced rapid and substantial improvements in Eczema Area and Severity Index (EASI) scores and patient-reported symptoms, with 72.2% of patients achieving EASI-75 and 25.9% achieving EASI-90 at week 16, including high response rates in patients who had previously failed biologic or JAK inhibitor therapy.

Acne was the most frequent adverse event with abrocitinib, followed by urticaria and herpes simplex infection, but events were generally manageable and did not lead to treatment discontinuation.

In descriptive, unadjusted comparisons across JAK inhibitors treated at our center during different periods, abrocitinib showed numerically higher EASI-50 and EASI-75 response rates at week 16, whereas upadacitinib showed the highest EASI-90 response rate.

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by severe pruritus and eczematous skin lesions resulting from skin barrier dysfunction [1, 2]. Overall, AD exerts health-related, social, economic, academic, and occupational effects on its patients, which leads to substantial health and economic burdens for patients with AD and their families. Indeed, according to the Global Burden of Disease 2019 database, AD had the highest disease burden among skin diseases, accounting for approximately US $3300 per person per year [3, 4]. Understandably, the disease burden of AD increases with disease severity, with the burden of severe AD estimated to be eight times greater than that of mild AD [5, 6]. For this reason, new treatment options for AD have recently been developed with advances in the understanding of disease mechanisms [7, 8].

Abrocitinib is a Janus kinase 1 (JAK1)-selective inhibitor approved for the treatment of moderate-to-severe AD [9]. JADE MONO-1, JADE EXTEND, and JADE COMPARE trials showed significant improvements in disease activity and patient-reported outcomes (PROs) of moderate-to-severe AD during abrocitinib treatment [10–12].

Although multiple real-world studies have evaluated abrocitinib, most have been constrained by small sample sizes, and data on Korean patients with AD remain scarce. Moreover, despite the increasing use of abrocitinib, upadacitinib, and baricitinib for the treatment of AD in Korea, real-world evidence directly comparing the efficacy and AE profiles of these JAK inhibitors is still insufficient.

Accordingly, this study aimed to evaluate the real-world effectiveness and safety of abrocitinib in Korean patients with moderate-to-severe AD, including those who have shown inadequate responses to other biologics or JAK inhibitors, and to compare its efficacy and safety profile with those of upadacitinib and baricitinib using real-world data collected at our center. Here, we report these real-world outcomes and present comparative data on clinical responses and AEs among JAK inhibitors currently approved for the treatment of AD in Korea.

METHODS

Study Design

We conducted a retrospective cohort study using electronic medical records from the Department of Dermatology, Korean National Medical Center (Seoul, Korea), with abrocitinib as the primary analytic cohort and upadacitinib and baricitinib as comparison cohorts. Patients 12 years or older with moderate-to-severe AD who initiated abrocitinib between 1 September 2022 and 30 April 2024 were identified. Sixty-six patients started abrocitinib during this period; 57 completed 16 weeks of treatment and were included in the abrocitinib analysis set; 9 patients discontinued treatment before week 16, including 6 due to economic burden and 3 who were lost to follow-up. For comparisons across JAK inhibitors, we also included cohorts treated at our center during defined periods: an upadacitinib cohort treated between 1 September 2021 and 1 September 2023 that included 49 patients, and a baricitinib cohort treated between 31 May 2021 and 31 December 2022 that included 26 patients.

Ethical Approval

Ethical approval was obtained from the institutional review board of the Korean National Medical Center (IRB number 2025-11-009). The study was conducted in compliance with the ethical principles from the Declaration of Helsinki and Good Clinical Practice guidelines. Patient consent was waived because of the retrospective nature of the study using de-identified data.

Inclusion Criteria

Eligible patients had a clinician-confirmed diagnosis of moderate-to-severe AD, defined

as a baseline EASI score of 7 or higher, and received continuous 16-week treatment with a JAK inhibitor (abrocitinib, upadacitinib, or baricitinib) within the predefined study periods at our center.

Treatment

The daily doses were 200 or 100 mg for abrocitinib, 30 or 15 mg for upadacitinib, and 4 or 2 mg for baricitinib. Doses could be escalated or reduced according to disease activity. Previous systemic treatments for AD were discontinued at JAK inhibitor initiation, whereas topical tacrolimus and topical corticosteroid were permitted for AD symptom management.

Dose Adjustment

Dose adjustments of abrocitinib were allowed during follow-up according to clinical response. Most patients initiated treatment with abrocitinib 200 mg, and dose reductions to 100 mg were primarily performed in patients who showed sufficient disease improvement. Dose re-escalation to 200 mg was considered in cases of disease flare.

Clinical and Laboratory Assessments

Patient data, including age, sex, age at onset of AD, previous treatments, and allergy history, were obtained from electronic medical records. Prior to initiation of abrocitinib therapy and at week 16, patients underwent laboratory assessments, including a complete blood count, serum biochemical analysis, total eosinophil count (TEC), lactate dehydrogenase (LDH), uric acid, and immunoglobulin E (IgE) levels.

Efficacy Measurements

Efficacy was assessed at baseline, week 2, and week 16 using the EASI. In addition to total EASI scores, changes in EASI by body region and symptom from baseline to week 16 were evaluated. The proportions of patients achieving at least 50%, 75%, and 90% improvement from baseline in EASI (EASI-50, EASI-75, and EASI-90, respectively) at weeks 2 and 16 were also calculated. Subjective symptom improvement was assessed at the same time points using patient-reported outcome tools, including the Peak Pruritus Numerical Rating Scale (ppNRS), Patient-Oriented Eczema Measure (POEM), Dermatology Life Quality Index (DLQI), and Atopic Dermatitis Control Tool (ADCT).

Safety Analysis

Safety assessments were performed during routine outpatient visits and were based on physician-confirmed adverse events recorded in the electronic medical record. Acne severity was assessed using clinical photographs; a dermatologist graded acne using an IGA-based scale and categorized cases as mild, moderate, or severe. Urticaria events were recorded when new wheals were identified during treatment and were distinguished from baseline chronic urticaria based on medical record review. Changes in laboratory test results were reviewed at week 16 of abrocitinib treatment compared with baseline.

Subgroup Analysis Within the Abrocitinib Cohort

Patients were categorized according to previous treatment into three groups: (i) naive to both dupilumab and JAK inhibitors, (ii) previously treated with either dupilumab or a JAK inhibitor, and (iii) a subgroup with prior upadacitinib treatment (a subset of group ii). Comparative analyses of treatment effectiveness were conducted among these subgroups.

Comparative Analyses Across JAK Inhibitors

Between-drug comparisons of clinical effectiveness and safety were performed descriptively between the abrocitinib cohort and the cohorts treated with upadacitinib and baricitinib using real-world data from our center. Because these cohorts were accrued during different calendar periods and treatment allocation was not randomized, the comparisons

are subject to potential confounding, including differences in baseline disease severity, prior advanced therapies, dose selection, and follow-up practices. Accordingly, these analyses were conducted as exploratory, unadjusted descriptive comparisons. Effectiveness and safety outcomes were evaluated at baseline and at weeks 2 and 16 using the EASI-50, -75, -90 and AE assessments described above.

RESULTS

Patient Disposition and Baseline Characteristics

A total of 66 patients were enrolled in this study, of which 57 completed 16 weeks of abrocitinib treatment for analysis. Nine patients did not complete week 16 follow-up and were not included in the week 16 analysis because of economic burden (6 patients) or loss to follow-up (3 patients) (Supplementary Fig. 1). Baseline demographics and disease characteristics of the participants are detailed in (Table 1).

Table 1 Baseline demographics and disease characteristics in abrocitinib cohort

Patient characteristics ($n = 57$)	
Age, mean (SD)	28.88 (± 9.95)
Age, *n* (%)	
Adult (age ≥ 18)	53 (93.0)
Adolescent (12 ≤ age < 18)	4 (7.0)
Sex, *n* (%)	
Male	31 (54.4)
Female	26 (45.6)
AD onset, *n* (%)	
Born	13 (22.8)
Childhood	28 (49.1)
Adolescence	8 (14.0)
Adult	8 (14.0)
Baseline AD severity based on EASI, *n* (%)	
Mild (EASI < 7)	0 (0)
Moderate (7 ≤ EASI < 16)	37 (64.9)
Severe (EASI ≥ 16)	20 (35.1)
Previous treatment, *n* (%)	
Topical	
Topical corticosteroids	55 (96.5)
Topical calcineurin inhibitors	49 (86.0)
Systemic	
Systemic corticosteroids	27 (47.4)
Cyclosporine	46 (80.7)
Methotrexate	6 (10.5)
Dupilumab	5 (8.8)
Baricitinib	12 (21.1)
Upadacitinib	11 (19.3)
Others	
Phototherapy	17 (29.8)
Immunotherapy	4 (7.0)

Dose Adjustment

Among 57 patients who initiated abrocitinib, 56 started at a dose of 200 mg and one patient at 100 mg. Among those who initiated 200 mg, 24 maintained this dose through week 16, while 32 underwent dose reduction to 100 mg during treatment. Dose reduction occurred at a mean of 8.7 weeks after treatment initiation, primarily in the context of clinical improvement. Of these patients, 30 maintained 100 mg through week 16, whereas 2 required re-escalation to 200 mg because of disease flare.

Efficacy Outcomes

EASI

After 2 weeks of abrocitinib treatment, the mean EASI score improved by 70.9% from baseline, increasing further to 79.0% at week 16 (Fig. 1). At week 2, 87.5% of patients achieved EASI-50,

Table 1 continued

Home remedy	8 (14.0)
Korean traditional medicine	25 (43.9)
Past allergic history	
Allergic rhinitis	24 (42.1)
Chronic urticaria	8 (14.0)
Allergic conjunctivitis	8 (14.0)
Asthma	7 (12.3)
Allergen sensitization	
Dermatophagoides	14 (24.6)
Fungus	6 (10.5)
Cat	5 (8.8)
Dog	2 (3.5)
Others	10 (17.5)

while 51.8% and 16.1% achieved EASI-75 and EASI-90, respectively; these responses were sustained through week 16, with 94.4%, 72.2%, and 25.9% of patients achieving EASI-50, EASI-75, and EASI-90, respectively (Fig. 2).

When stratified by baseline disease severity, response rates were as follows. In the moderate group, EASI-50, EASI-75, and EASI-90 response rates were 88.9%, 58.3%, and 13.9% at week 2 and 91.7%, 75.0%, and 30.6% at week 16, respectively (Supplementary Fig. 2A). In the severe group, EASI-50, EASI-75, and EASI-90 response rates were 85.0%, 40.0%, and 20.0% at week 2 and 100.0%, 66.7%, and 16.7% at week 16, respectively (Supplementary Fig. 2B).

Regional analysis showed that all body areas exhibited more than 80% improvement in EASI scores at week 16, with the lower extremities demonstrating the greatest reduction (87.5% improvement). Improvements were also observed across all clinical signs, with mean reductions of 71.2% in erythema, 77.1% in induration, 86.6% in excoriation, and 89.7% in lichenification (Fig. 3a, b).

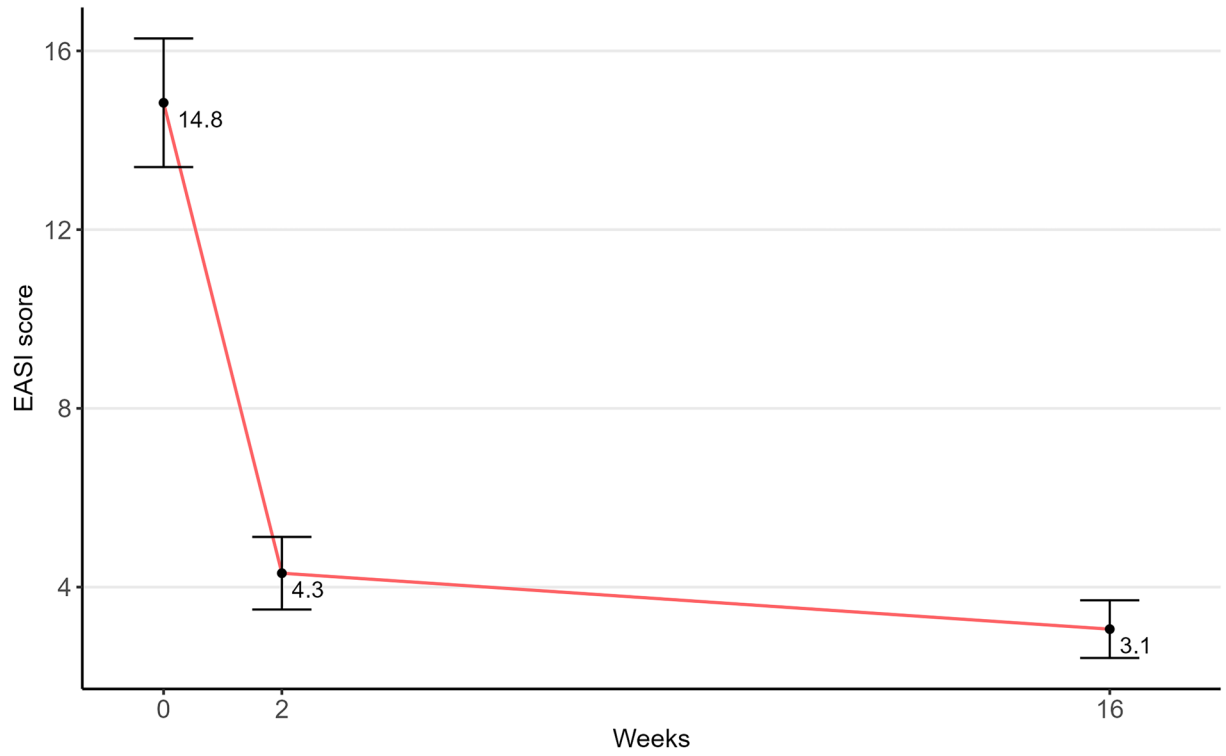

Fig. 1 Mean EASI scores after abrocitinib treatment at baseline, week 2, and week 16

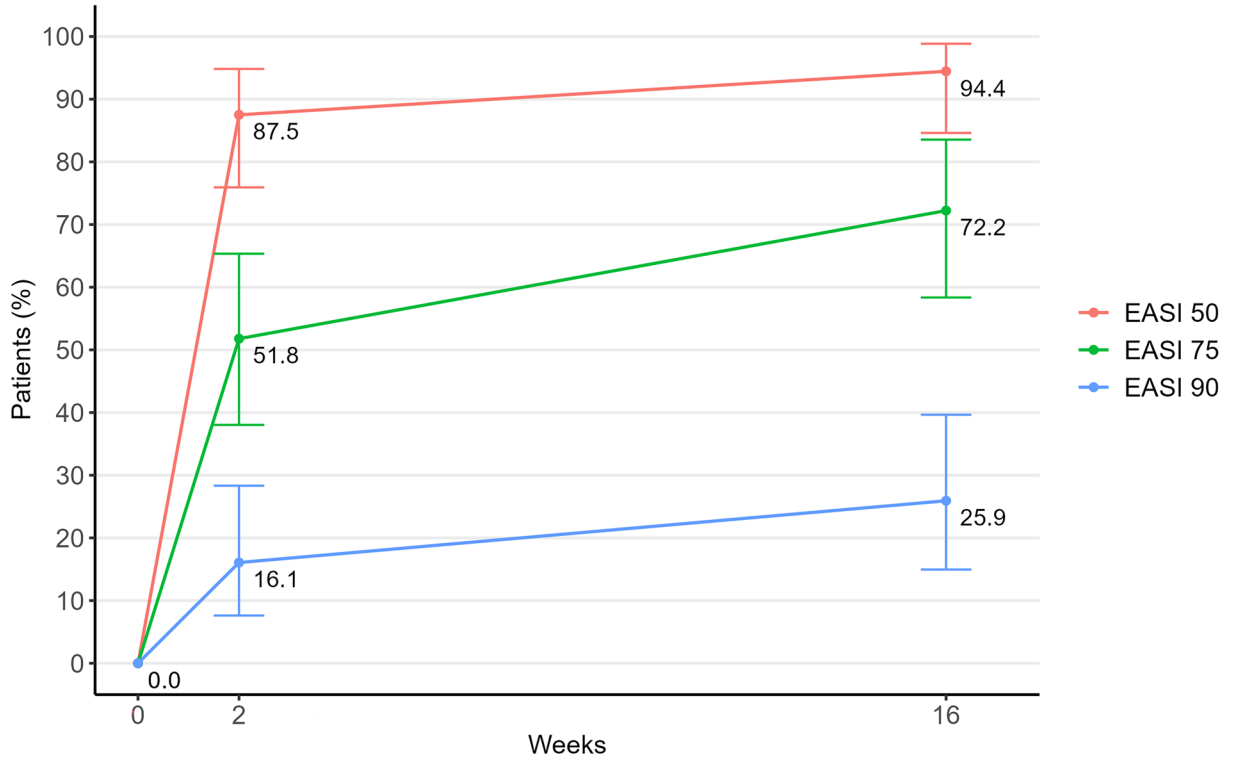

Fig. 2 Proportions of patients achieving EASI-50, EASI-75, and EASI-90 after abrocitinib treatment at weeks 2 and 16

Supplementary Fig. 3 illustrates the clinical course of a 31-year-old patient with involvement of the popliteal fossae. The baseline EASI and ppNRS were 18.7 and 8, respectively, which decreased to 2.7 and 3 after 16 weeks of abrocitinib therapy, accompanied by substantial improvements in patient-reported outcomes (Supplementary Fig. 3).

Patient-Reported Outcomes

Patients' perceptions of symptoms, disease control, and quality of life related to AD showed substantial improvements, as assessed by the ppNRS, POEM, DLQI, and ADCT.

Mean ppNRS scores decreased from baseline to week 2 and continued to decline by week 16 (Fig. 4).

POEM, DLQI, and ADCT scores also showed marked reductions from baseline at week 2, with further decreases observed at week 16 (Supplementary Fig. 4).

Laboratory Findings

Laboratory markers of AD control, including IgE, TEC, and LDH were assessed at baseline and week 16 of abrocitinib treatment. Although IgE and LDH levels increased slightly from baseline, TEC levels declined by 26.11%. However, none of the changes in laboratory parameters reached statistical significance (Table 2).

Safety Outcomes

The most common adverse event (AE) associated with abrocitinib treatment was acne ($n = 25$, 43.9%), followed by urticaria ($n = 14$, 24.6%) (Table 3). There were no dose reductions or treatment discontinuations due to AEs.

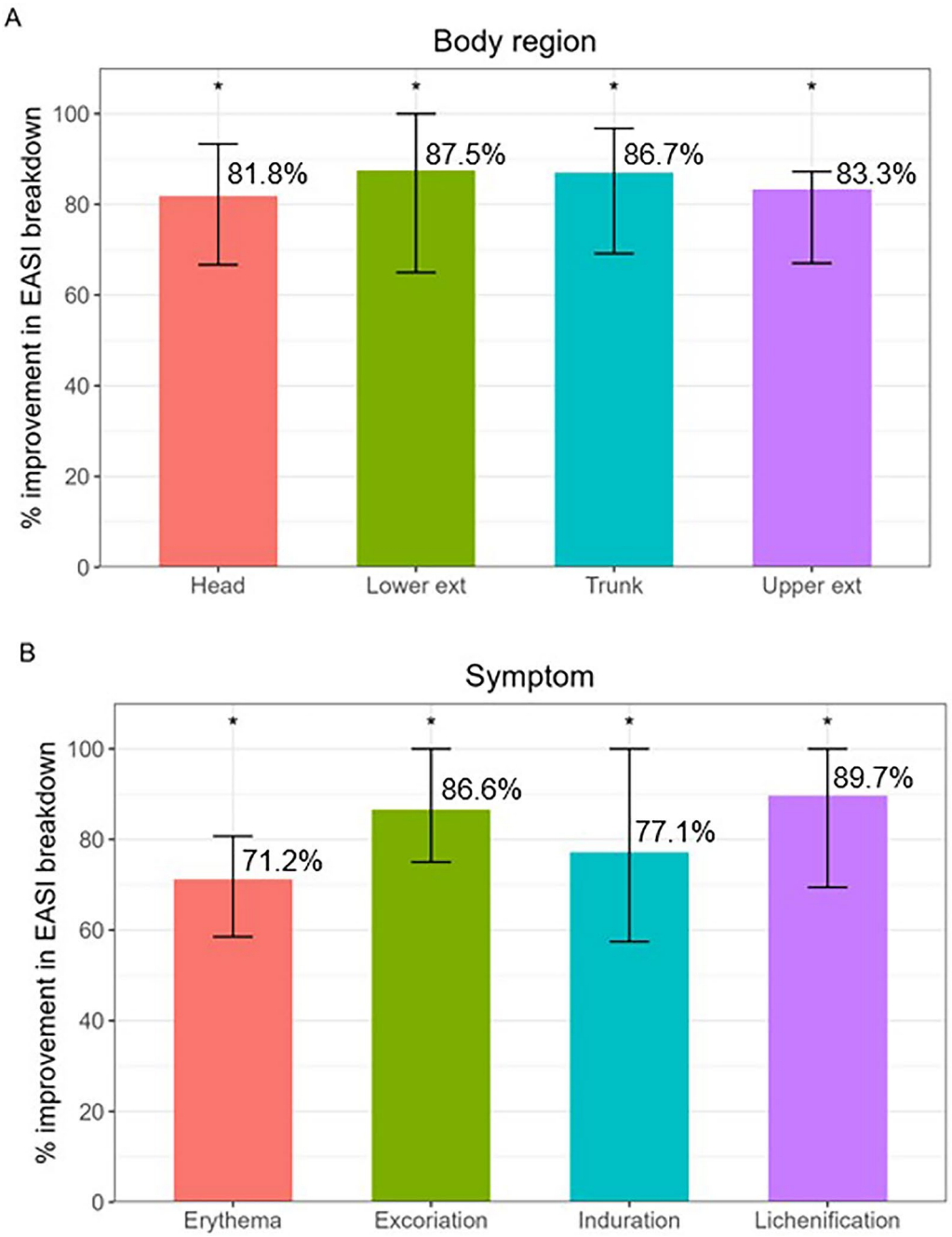

Fig. 3 Percentage improvement from baseline in EASI subscores by body region (**a**) and symptom (**b**) after abrocitinib treatment at week 16 (*$p < 0.05$ by Wilcoxon signed rank test)

Acne

Among the 25 patients who experienced acne during abrocitinib treatment, 14 developed new-onset acne, whereas 11 experienced aggravation of pre-existing acne. The mean time to onset of new acne was approximately 14 weeks after initiating abrocitinib. Based on clinical photographs, acne severity was graded by a dermatologist using an IGA-based ordinal

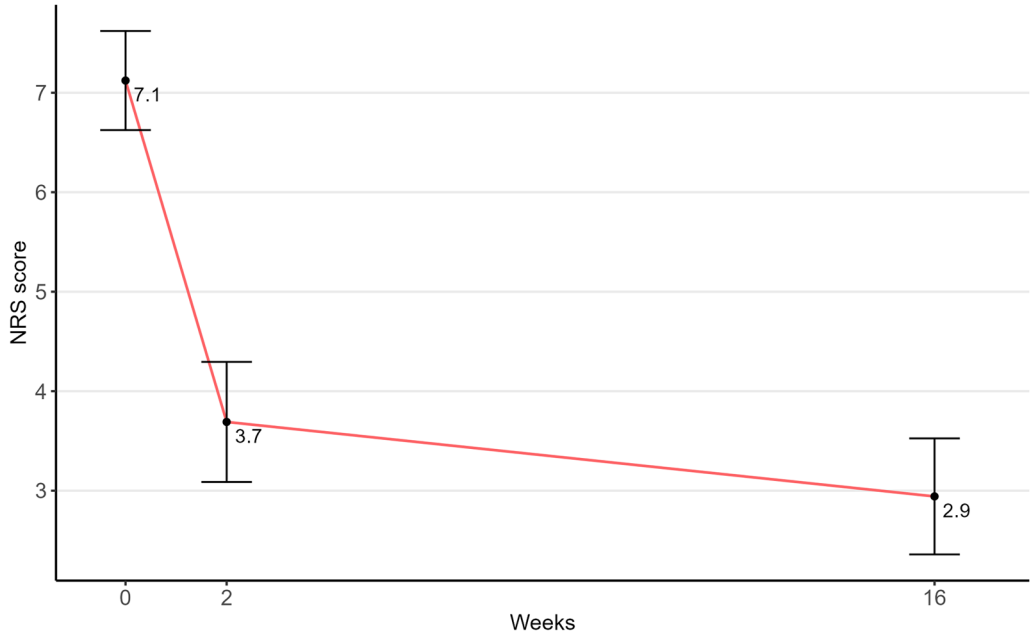

Fig. 4 Mean ppNRS scores after abrocitinib treatment at baseline, week 2, and week 16

Table 2 Percentage change in laboratory markers from baseline to week 16

Outcome	Baseline[a]	After 16 weeks[a]	Percentage change	
			After 16 weeks	*p* value*
IgE	1103.00 (231.00, 4389.00)	1581.00 (148.50, 9671.50)	4.13 (− 34.92, 46.50)	0.337
TEC	298.50 (138.00, 484.50)	198.00 (108.50, 307.00)	− 26.11 (− 47.42, 17.42)	0.065
LDH	226.00 (201.00, 253.50)	223.00 (201.50, 258.00)	1.92 (− 6.49, 17.15)	0.161

[a]Median (Q1, Q3)

**p* values were calculated the Wilcoxon signed rank test

scale: 21 cases were mild and 4 were moderate; no severe cases were observed. The acne manifestations were effectively managed with standard therapeutic interventions: 10 patients received isotretinoin, 4 were treated with doxycycline, and the remaining 11 were managed with topical treatments.

Urticaria

Urticaria was reported by 14 patients during abrocitinib treatment. None of the eight patients with a baseline history of chronic urticaria reported a flare during the 16-week treatment period. All urticaria events were considered new onset during treatment and improved with concomitant antihistamines, without requiring discontinuation of abrocitinib.

Infection

Herpes simplex infections were reported during abrocitinib treatment, with a median onset of 13 weeks. In addition, one patient experienced two episodes of recurrent herpes simplex infection. Other notable herpesvirus-related events

Table 3 AE profile during abrocitinib treatment

AE	*N* (%)
Acne	25 (43.9)
Urticaria	14 (24.6)
Herpes simplex infection	7 (12.3)
Herpes zoster infection	2 (3.5)
Eczema herpeticum	1 (1.8)
Nausea	10 (17.5)
Heartburn	5 (8.8)
Diarrhea	2 (3.5)
Constipation	1 (1.8)
Proteinuria	1 (1.8)
Menstrual irregularity	1 (1.8)
Headache	1 (1.8)

included herpes zoster infection and eczema herpeticum, each occurring in one patient. No patients had received herpes zoster vaccination prior to initiating abrocitinib. During treatment, two patients received the recombinant zoster vaccine as a two-dose series administered approximately 2 months apart: doses were given at weeks 4 and 12 in one patient and at weeks 6 and 14 in the other.

Comparative Efficacy of Abrocitinib Based on Prior Biologic or JAK Inhibitor Treatment

Patients were stratified according to prior advanced therapy before abrocitinib initiation into three subgroups: naive to both dupilumab and JAK inhibitors ($n=36$), previously treated with dupilumab or a JAK inhibitor ($n=21$), and a subgroup with prior upadacitinib treatment ($n=10$). The proportions of patients achieving EASI-50, -75, and -90 at weeks 2 and 16 were compared across these subgroups. In subgroup analyses, the previously treated dupilumab or JAK inhibitor group showed the highest rates of EASI-50 and EASI-75 at week 16, whereas the naive group demonstrated the highest EASI-90 at week 16; response rates were lowest in the upadacitinib subgroup (Fig. 5a–c).

Comparative Analyses Across JAK Inhibitors

For between-drug comparisons, clinical effectiveness and safety were evaluated descriptively in unadjusted analyses between the abrocitinib cohort and the cohorts treated with upadacitinib and baricitinib using real-world data from our center in patients with moderate-to-severe AD. Baseline demographics and disease characteristics of the participants are presented in Supplementary Tables 1 and 2. Regarding efficacy, EASI-50 response rates at week 2 were 87.5% with abrocitinib, 66.7% with upadacitinib, and 47.5% with baricitinib. By week 16, these rates increased to 94.4%, 87.8%, and 50.7% with abrocitinib, upadacitinib, and baricitinib, respectively. Similarly, EASI-75 response rates at week 2 were 51.8% with abrocitinib, 35.4% with upadacitinib, and 18.0% with baricitinib, while at week 16 these rates were 72.2%, 71.4%, and 30.4%, respectively. For EASI-90, response rates at week 2 were 16.1% with abrocitinib, 6.2% with upadacitinib, and 14.8% with baricitinib; by week 16, these rates were 25.9%, 30.6%, and 17.4% with abrocitinib, upadacitinib, and baricitinib, respectively (Fig. 6a–c). After 16 weeks of JAK inhibitor treatment, the incidence of acne was 43.9% with abrocitinib, 50.0% baricitinib, 44.9% upadacitinib (Fig. 7). Additionally, the incidence of herpes simplex was 12.3% with abrocitinib, compared with 19.2% with baricitinib and 18.4% with upadacitinib (Fig. 8).

DISCUSSION

This retrospective real-world study evaluated the efficacy and safety of abrocitinib in patients with moderate-to-severe AD treated at the National Medical Center in South Korea. It represents the largest single-center Korean cohort investigated to date.

Our study demonstrated marked improvements in both EASI and PROs throughout

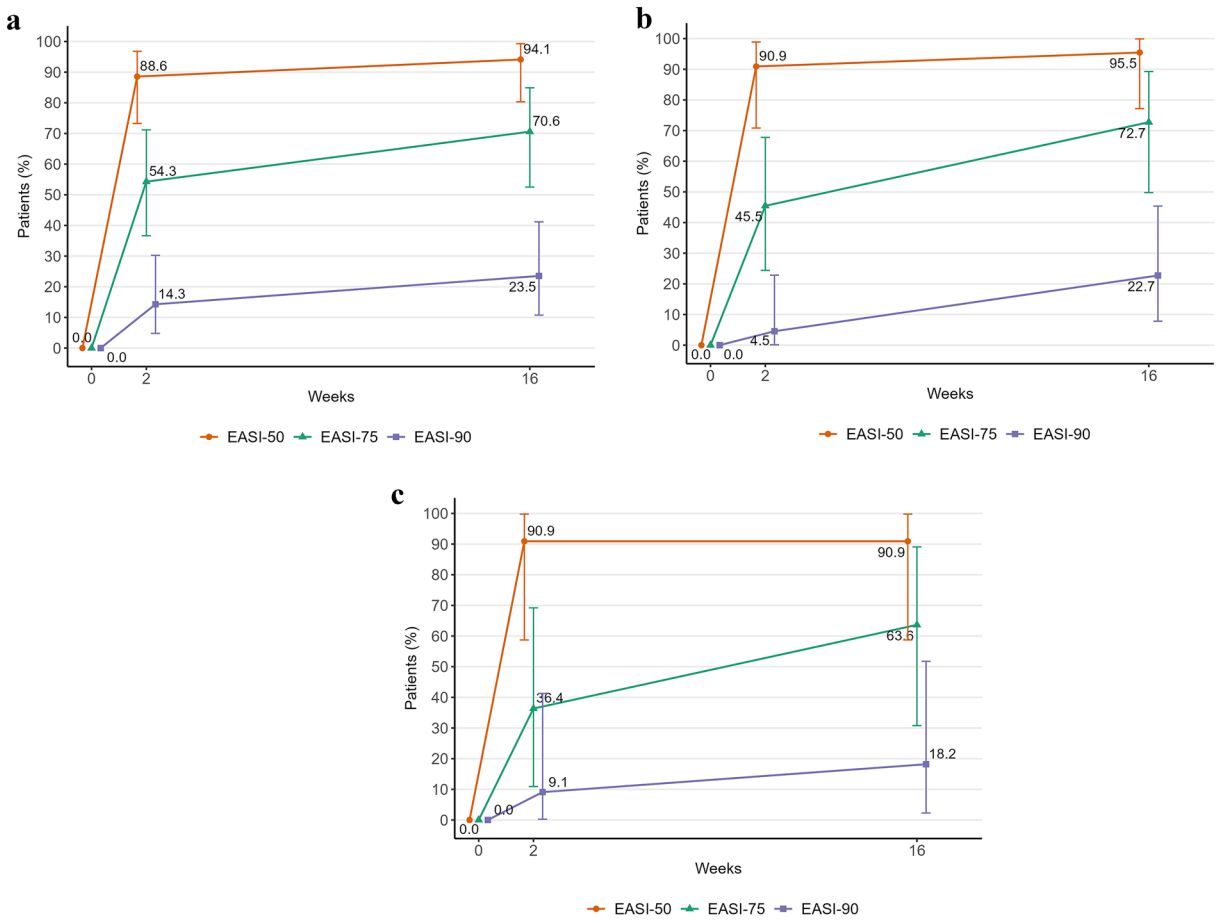

Fig. 5 Comparison of abrocitinib efficacy among patients naive to dupilumab and JAK inhibitors (**a**), patients previously treated with dupilumab or a JAK inhibitor (**b**), and patients with prior upadacitinib treatment (**c**)

the treatment. A total of 72.2% of patients achieved EASI-75 at week 16, which is higher than the response rates reported in clinical trials, including JADE MONO-1, and in other real-world studies, although still slightly lower than those in the cohort reported by Armario et al. [10, 13–16]. Stratified analyses by baseline severity suggested that clinical responses were observed in both moderate and severe AD subgroups over 16 weeks, supporting effective disease control across severity strata in routine practice. All patient-reported outcome measures also demonstrated a rapid improvement from week 2, which was sustained throughout the 16-week treatment period.

EASI subscore analysis showed greater than 80% improvement across all body regions, with the greatest improvement observed in the lower extremities (87.5%). In a secondary analysis of JADE COMPARE, Alexis et al. evaluated body-region responses. They reported greater week-16 improvement with abrocitinib 200 mg once daily than with dupilumab 300 mg every 2 weeks, particularly in the lower extremities (84.5%) [17]. Taken together, these observations suggest that abrocitinib may represent an effective therapeutic option, particularly for patients whose AD predominantly involves the lower extremities.

Among individual symptoms, lichenification showed the greatest reduction, with an 89.7% improvement from baseline. These findings support the hypothesis that abrocitinib, through selective inhibition of JAK1, may effectively

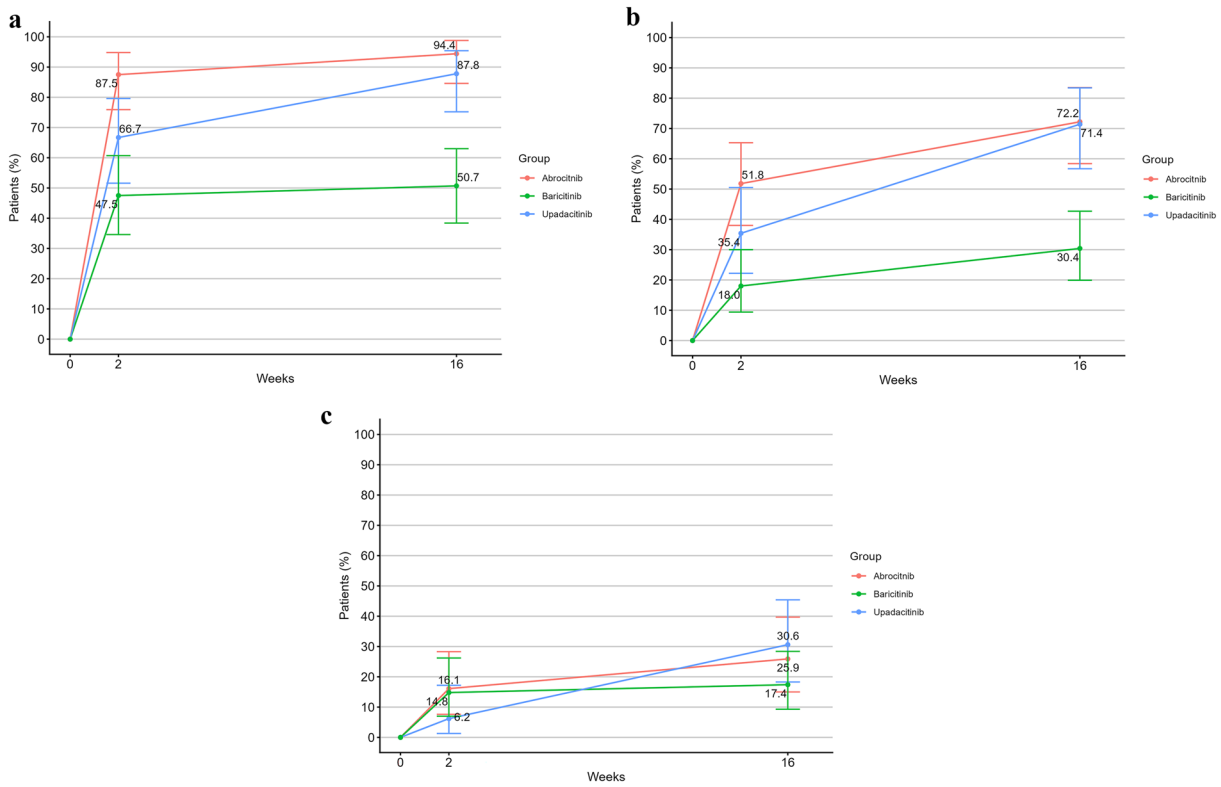

Fig. 6 Proportion of patients achieving EASI-50 (**a**), EASI-75 (**b**), and EASI-90 (**c**) at weeks 2 and 16 with abrocitinib, baricitinib, and upadacitinib treatment

improve lichenification by modulating interleukin (IL)-22, a key mediator of epidermal hyperplasia and barrier dysfunction. Considering that elevated levels of IL-22 have been observed in both the serum and skin lesions of Asian patients with AD, abrocitinib may play a significant role in improving symptoms in this population [18, 19].

Comparison of efficacy among the three subgroups—patients naive to both dupilumab and JAK inhibitors, patients previously treated with dupilumab or JAK inhibitor, and patients with prior upadacitinib treatment—showed that the previously treated dupilumab or JAK inhibitor subgroup achieved the highest EASI-50, EASI-75, and EASI-90 response rates, whereas response rates tended to be lower in the subgroup with prior upadacitinib treatment. In a previous study of JADE DARE participants who switched from dupilumab to abrocitinib, 95.0% (264/278) of patients who were EASI-50 responders at week 26 on dupilumab and 56.0% (9/16) of those who were EASI-50 nonresponders achieved EASI-75 after 12 weeks of abrocitinib treatment [20]. Thus, prior inadequate response to dupilumab has been associated with attenuated responses to subsequent abrocitinib compared with biologic-naive patients. In our cohort, however, patients previously treated with dupilumab or a JAK inhibitor did not appear less responsive and instead showed higher EASI response rates than dupilumab and JAK inhibitor-naive patients. To our knowledge, no studies have specifically evaluated patients with inadequate response to another JAK inhibitor who subsequently switched to abrocitinib, underscoring the need for further investigation in this subgroup.

Laboratory data showed a 26.1% reduction in TEC from baseline to week 16, although this change did not reach statistical significance. By contrast, Uchiyama et al. reported a significant reduction in TEC at 12 weeks [14]. We also evaluated potential prognostic factors for

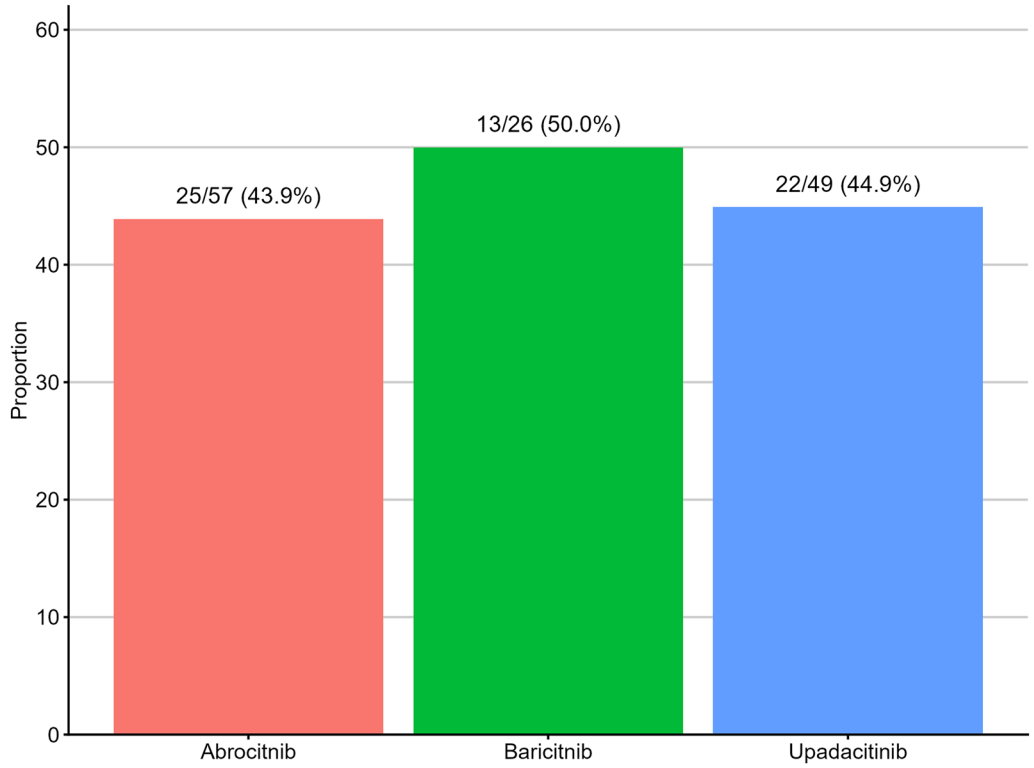

Fig. 7 Incidence of acne after 16 weeks of abrocitinib, baricitinib, and upadacitinib treatment

achieving EASI-75 at week 16, including sex, baseline EASI severity, age at AD onset, baseline IgE, TEC, LDH, BMI, and previous systemic treatments. Among these, adult-onset AD was the only factor significantly associated with achieving EASI-75.

The comparisons between the JAK inhibitors in this study are exploratory and unadjusted. The cohorts for upadacitinib and baricitinib were accrued during different time periods, with nonrandom treatment allocation, and potential differences in baseline disease severity, prior treatments, dosing, and follow-up practices. Therefore, these findings should be interpreted with caution, as unmeasured confounding factors may have influenced the observed differences in effectiveness and safety. In unadjusted comparisons, abrocitinib showed higher EASI-50 and EASI-75 response rates at week 16 compared to upadacitinib and baricitinib, while upadacitinib showed a higher EASI-90 response rate. In a real-world study by Ibba et al., after 16 weeks of treatment, 76.0% and 62.5% of patients receiving upadacitinib achieved EASI-75 and EASI-90, respectively, whereas 88.0% and 52.0% of those receiving abrocitinib reached these endpoints [21]. Given that many key cytokines involved in AD pathogenesis signal through JAK1, selective JAK1 inhibition, rather than pan-JAK or JAK1/2 inhibition, may contribute to differences in clinical responses observed in real-world settings; however, further studies with adjusted or randomized designs are needed to clarify the comparative clinical relevance of these findings.

Because many key cytokines involved in AD pathogenesis signal via JAK1, selective JAK1 inhibition, rather than JAK1/2 inhibition, may provide greater efficacy while reducing interference with other signaling pathways [22–24]. Although both abrocitinib and upadacitinib are classified as selective JAK1 inhibitors, subtle pharmacologic differences between them may contribute to the distinct clinical outcomes observed. Additional studies, particularly

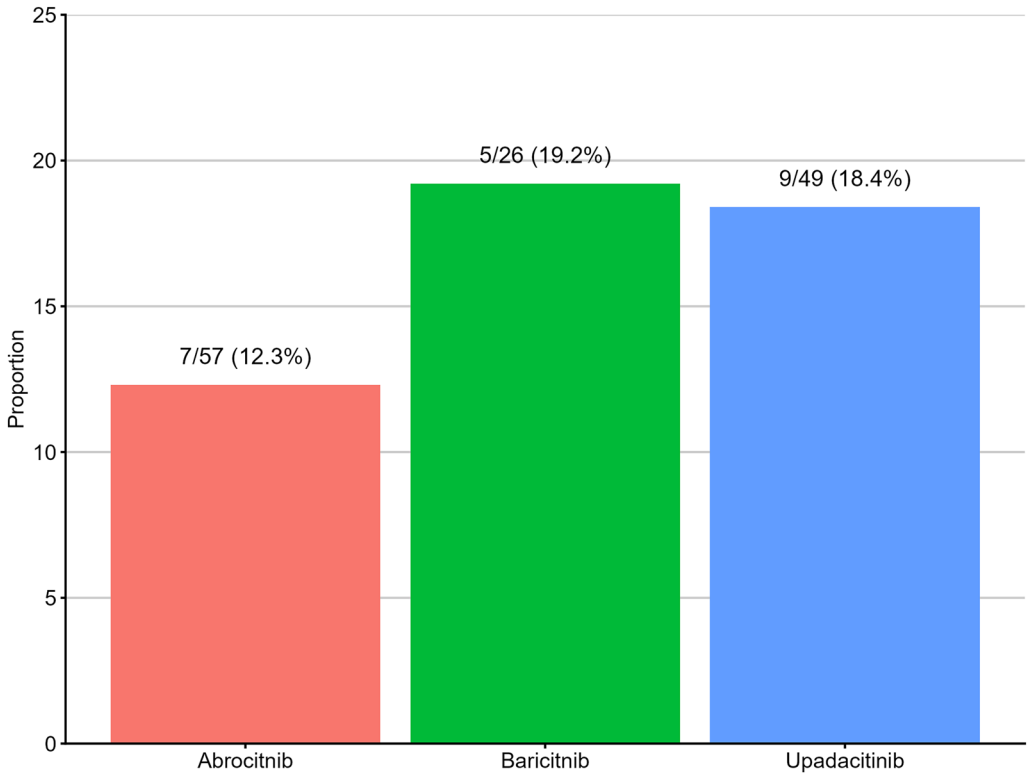

Fig. 8 Incidence of herpes simplex after 16 weeks of abrocitinib, baricitinib, and upadacitinib treatment

head-to-head comparisons, are required to clarify the clinical relevance of these differences.

Concomitant topical therapy may have contributed to real-world outcomes. In our routine practice, topical corticosteroids were used in a limited manner, mainly for severe scalp lesions when clinically needed, while topical tacrolimus was recommended twice daily during AD flares and then continued as maintenance therapy (at least twice weekly). However, because topical treatment intensity (e.g., frequency and amount) was not captured in a standardized manner in this retrospective dataset, we could not formally assess whether topical use remained stable or changed over follow-up; this should be considered when interpreting effectiveness outcomes.

Several clinical trials and meta-analyses have demonstrated an increased risk of incident acne with systemic JAK inhibitors, particularly JAK1-selective agents such as abrocitinib [25–28]. Acne events with abrocitinib are typically mild to moderate and dose dependent, with higher rates observed at 200 mg than at 100 mg, and they rarely lead to treatment discontinuation [27, 29, 30]. Although the exact pathogenesis of JAK inhibitor-associated acne remains unclear, emerging data suggest that JAK1 inhibition may modulate sebaceous gland activity and retinoid–mTORC1 signaling, thereby promoting follicular hyperkeratinization and increased sebum production [28, 31, 32].

In our cohort, acne was the most common AE, occurring in 43.9% of patients, and its incidence was higher than that reported in other abrocitinib studies, where acne occurred in 6.6% of patients over 12 weeks in the JADE-MONO1 trial, 20.0% during the observation period in the cohort described by Olydam et al., and 22.37% over 24 weeks in the study by Armario-Hita et al. [10, 13, 16]. This discrepancy may partly reflect the younger mean age of our cohort (28.8 years) and the high proportion of patients with a prior history of acne (44%), compared with the mean ages reported in JADE-MONO1 (33.0 years for 200 mg and 32.6 years for 100 mg), Olydam

et al. (32.0 years), and Armario-Hita et al. (34.0 years). In addition, routine counseling at treatment initiation regarding potential acne during JAK inhibitor therapy may have increased patient awareness and facilitated earlier reporting and documentation. Previous studies of JAK inhibitors have also suggested that JAK inhibitor-associated acne occurs more often in younger patients and is more frequent in individuals with a personal history of acne, which is consistent with the age distribution and baseline characteristics of our cohort [26, 33]. In descriptive, unadjusted comparisons across JAK inhibitors in our real-world cohort, acne was observed numerically more frequently with upadacitinib and baricitinib than with abrocitinib. Whereas a recent real-world comparative study of upadacitinib and abrocitinib reported acne incidences of 6.7% and 8.0%, respectively, after 52 weeks of treatment, suggesting a slightly higher risk with abrocitinib [21].

Regarding urticaria, symptoms appeared as new-onset rather than flares of pre-existing chronic urticaria. Although urticaria is not commonly reported in prior abrocitinib studies, the relationship to abrocitinib remains unclear in this retrospective review and warrants further evaluation.

An increased risk of infection during abrocitinib therapy has been reported in prior studies, with herpes simplex consistently being the most frequent infection. Suppression of the JAK–STAT pathway may impair antiviral host defenses and facilitate reactivation of latent herpes viruses [7, 34–36]. In our study, the incidence of herpes simplex infection was 12.3%, which was at the higher end of the range reported in real-world cohorts and clinical trials. In real-world data, Kamphuis et al. reported an incidence of 12.6% during the first 28 weeks of treatment, Olydam et al. reported 3.3% over the entire follow-up period, and Simpson et al. reported 7.3% with abrocitinib 100 mg and 8.1% with 200 mg [13, 37, 38]. In the JADE-MONO1 trial, the incidence of herpes simplex was 5% during the 12-week treatment period [10]. In descriptive, unadjusted comparisons, herpes simplex infections were numerically more frequently observed with upadacitinib and baricitinib than with abrocitinib. In a comparative study of JAK inhibitors, Ibba et al. observed prevalences of 4.0% in the upadacitinib group and 1.9% in the abrocitinib group (baseline to 52 weeks) [21]. In addition, the 52-week BioDay analysis identified herpes simplex as the most common infection, with incidence rates per 1000 patient-years of 172.3 in patients treated with abrocitinib, 113.4 in those treated with baricitinib, and 136.2 in those treated with upadacitinib [39]. The comparatively high incidence of herpes simplex in our cohort may reflect both baseline AD severity and the way herpes simplex events were detected and recorded. Our cohort consisted exclusively of patients with moderate-to-severe AD, and higher baseline disease severity has been associated with an increased risk of herpesvirus infections, including herpes simplex [40]. In addition, we screened even mild episodes of labial herpes as AEs, which likely led to higher detection rates than in studies.

Limitation

This study has several limitations. First, the retrospective single-center design may limit external validity and is subject to residual confounding. Second, comparisons across JAK inhibitors were exploratory and unadjusted, as cohorts were assembled in different calendar periods and may have differed in baseline characteristics, dosing, and follow-up practices. Third, concomitant topical treatment intensity was not captured in a standardized manner, and adverse events were identified during routine outpatient visits, which may have influenced event detection and severity characterization. Finally, follow-up was limited to 16 weeks; longer-term effectiveness and safety should be evaluated in future analyses with extended follow-up.

CONCLUSION

The primary strength of this study is its real-world, single-center design, including the largest cohort of abrocitinib-treated patients

with AD in Korea. To our knowledge, it is also the first study to directly compare JAK inhibitors for AD using real-world data from a single center. Over 16 weeks of treatment, abrocitinib demonstrated high efficacy and a favorable safety profile, in line with findings from clinical trials and other real-world studies. Significant improvements were observed across all body regions and symptoms, particularly in the lower extremities and lichenification, with strong efficacy even among patients resistant to dupilumab or other JAK inhibitors. We additionally report descriptive, exploratory comparisons with upadacitinib and baricitinib, which should be interpreted with caution. Overall, these findings support abrocitinib as an effective and well-tolerated treatment option for Asian patients with AD, including those refractory to prior dupilumab or JAK inhibitor therapies.

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Declarations

Conflicts of Interest. Ho Eun Gwag, Minjung Park, So Yun Park, Narang Hong, Seok Jae Heo, Hye Jung Jung, Mi Youn Park, Yu Sung Choi have no financial or other conflicts of interest to disclose. Jiyoung Ahn reported receiving grants from Sanofi Genzyme, Eli Lilly, Amgen and Leo Pharmaceuticals and consulting fees from AbbVie and Pfizer.

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