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Clinical course after switching from upadacitinib to biologic therapy in patients with atopic dermatitis

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Background: Upadacitinib provides rapid and substantial improvement in patients with moderate-to-severe atopic dermatitis (AD); however, the clinical course after switching from upadacitinib to biologic therapy remains poorly characterized.
Methods: This retrospective descriptive case series included 12 Japanese patients with moderate-to-severe AD who transitioned from upadacitinib to biologic therapy between April 2021 and March 2026. Seven patients received upadacitinib 15 mg and five received 30 mg before switching to dupilumab (n = 6), tralokinumab (n = 2), or lebrikizumab (n = 4). Clinical severity, patient-reported outcomes, laboratory biomarkers, and adverse events were evaluated before and after switching.
Results: The EASI-75 achievement rate decreased from 67% (8/12) during upadacitinib treatment to 50% (6/12) at 16 weeks after switching. Similarly, the proportion of patients achieving a ≥4-point improvement in pruritus numerical rating scale decreased from 90% (9/10) to 80% (8/10), although this change was not statistically significant. Transient disease flare occurred shortly after switching in three patients (25%), requiring temporary oral cyclosporine treatment. Among seven patients who switched after a uniform 12-week course of upadacitinib, worsening was most evident 4 weeks after switching, although clinical outcomes remained significantly improved compared with baseline at 16 weeks after switching.
Conclusion: Overall disease control was generally maintained after switching from upadacitinib to biologic therapy, although transient early worsening occurred in a subset of patients. These findings highlight the importance of careful clinical monitoring during the early transition period.

KEYWORDS
atopic dermatitis, biologics, case series, Japanese, upadacitinib

Introduction

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by recurrent eczematous lesions and pruritus that substantially impair patients' quality of life. In recent years, the treatment landscape for moderate-to-severe AD has expanded considerably with the introduction of biologic agents targeting type 2 inflammatory pathways and oral Janus kinase (JAK) inhibitors. Among these agents, upadacitinib, a selective JAK1 inhibitor, provides rapid improvement in both skin inflammation and pruritus and has demonstrated high efficacy in clinical trials and real-world practice [1, 2].

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Long-term studies have provided accumulating evidence regarding the efficacy and safety of both JAK inhibitors and biologic therapies in patients with AD [3]. Nevertheless, JAK inhibitors require regular safety monitoring and consideration of potential long-term adverse events. In routine clinical practice, patients receiving upadacitinib may therefore transition to biologic therapy for various reasons, including long-term treatment planning, safety considerations, patient preference, or inadequate treatment response. However, the clinical course during such treatment transitions has not been fully characterized.

Previous reports on switching from upadacitinib to biologic therapy have mainly involved patients with inadequate treatment response or adverse events. Hagino et al. evaluated 20 patients who switched from upadacitinib to tralokinumab because of inadequate response or adverse events, whereas Ito et al. reported two patients who experienced immediate disease exacerbation after switching from upadacitinib to dupilumab [4, 5]. Although these reports provide important information regarding treatment transitions from upadacitinib to biologic therapy, limited evidence is available regarding transitions in patients switching for reasons other than treatment failure.

More recently, Wang et al. reported that four of nine patients who immediately switched from JAK inhibitors to dupilumab experienced disease recurrence within the first 4 weeks after switching [6]. However, the clinical course and management of disease worsening following treatment switching remain insufficiently characterized, particularly in patients switching for reasons other than inadequate treatment response.

Therefore, we retrospectively characterized the clinical course of patients with moderate-to-severe AD who switched from upadacitinib to biologic therapy in routine clinical practice. We particularly focused on changes in physician-assessed disease severity and patient-reported outcomes during the transition period, as well as the occurrence and management of transient disease flare after switching.

Materials and methods

Study design and patients

This retrospective descriptive case series included patients with moderate-to-severe AD who transitioned from upadacitinib to biologic therapy at Gunma University Hospital between April 2021 and March 2026. All patients fulfilled the diagnostic criteria for AD [7]. Patients were included if they had received upadacitinib for the treatment of AD and subsequently transitioned directly to dupilumab, tralokinumab, or lebrikizumab, with clinical data available before and after treatment switching.

All patients continued upadacitinib until the day before initiation of biologic therapy, with no washout period between treatments. Throughout the treatment course, patients concomitantly used topical corticosteroids and moisturizers.

The reasons for switching were retrospectively reviewed from the medical records. Long-term treatment planning was defined as switching to biologic therapy for considerations other than inadequate treatment response, including long-term safety concerns, the need for regular laboratory monitoring, and patient preferences, as documented in the medical records.

TABLE 1 Patient demographics and baseline disease characteristics.

Characteristic	Total patients (n = 12)
Age, years	
Median (SD)	35.5 (15.6)
Range	15–68
Gender	
Male	8 (66.7%)
Female	4 (33.3%)
EASI (total)	
Median (SD)	34.3 (9.3)
Range	16.2–47.5
EASI (head and neck)	
Median (SD)	2.6 (1.2)
Range	0–4.5
IGA	
Median (SD)	3.5 (0.6)
Range	2–4
BSA, %	
Median (SD)	63.5 (18)
Range	40–90
NRS	
Median (SD)	6.4 (2.5)
Range	2–9
POEM	
Median (SD)	15 (7.5)
Range	3–28
Duration of upadacitinib treatment	
12 weeks	7
>12 weeks	5
Biologics	
Dupilumab	6
Tralokinumab	2
Lebrikizumab	4

Clinical assessments

Clinical assessments included Investigator's Global Assessment (IGA), affected body surface area (BSA), Eczema Area and Severity Index (EASI; total and head/neck), pruritus numerical rating scale (NRS), and Patient-Oriented Eczema Measure (POEM). Laboratory assessments included peripheral blood eosinophil counts and serum

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TABLE 2 Details of 12 patients with atopic dermatitis treated with upadacitinib and biologics.

Case	Age	Sex	EASI (total)			NRS		
			Baseline	(Switch point JAK→Bio)	(16 weeks post-bio)	Baseline	(Switch point JAK→Bio)	(16 weeks post-bio)
#1	15	M	16.2	12.9	4.8	2	4	2
#2	38	M	25.1	1.2	0.8	2	1	1
#3	48	F	30.4	15.5	1.7	8	1	2
#4	55	F	37.5	0.6	7.1	8	0	4
#5	26	M	38.4	25.5	14.8	8	1	6
#6	33	M	45	1.6	2.5	5	0	0
#7	41	M	47.5	5	2.7	8	3	2
#8	26	F	35.8	5.4	11.9	8	1	4
#9	24	M	25.8	20.4	24	4	5	4
#10	23	F	45.8	2	13.8	7	2	3
#11	49	M	31.3	1.8	0	8	0	0
#12	68	M	33.5	0.5	12	9	0	5

Case	Dose of upadacitinib	Duration of upadacitinib treatment (weeks)	Additional treatment for flare	Biologics	Reason for switching
#1	15 mg	12	-	Dupilumab	Inadequate response
#2	30 mg	12	-	Dupilumab	Long-term treatment planning (Concerns regarding long-term safety)
#3	30 mg	12	CyA 100 mg for 35 days	Dupilumab	Inadequate response
#4	15 mg	12	-	Tralokinumab	Long-term treatment planning (Concerns regarding long-term safety)
#5	15 mg	12	CyA 100 mg for 56 days	Dupilumab	Inadequate response
#6	30 mg	12	-	Dupilumab	Long-term treatment planning (Concerns regarding long-term safety)
#7	30 mg	12	-	Lebrikizumab	Long-term treatment planning (The need for laboratory monitoring)
#8	30 mg	83	-	Lebrikizumab	Long-term treatment planning (Concerns regarding long-term safety)
#9	30 mg	23	-	Lebrikizumab	Inadequate response
#10	15 mg	64	-	Dupilumab	Long-term treatment planning (The need for laboratory monitoring)
#11	15 mg	75	-	Lebrikizumab	Long-term treatment planning (The need for laboratory monitoring)
#12	15 mg	115	CyA 200 mg for 30 days	Tralokinumab	Long-term treatment planning (The need for laboratory monitoring)

levels of immunoglobulin E (IgE), lactate dehydrogenase (LDH), and thymus and activation-regulated chemokine (TARC). Adverse events and the use of additional systemic treatment for disease flare were also retrospectively assessed. Clinical and laboratory assessments were evaluated at baseline before upadacitinib treatment, at the time of switching from upadacitinib to biologic therapy, and 16 weeks after switching. In addition, to characterize the temporal course during treatment transition under a uniform

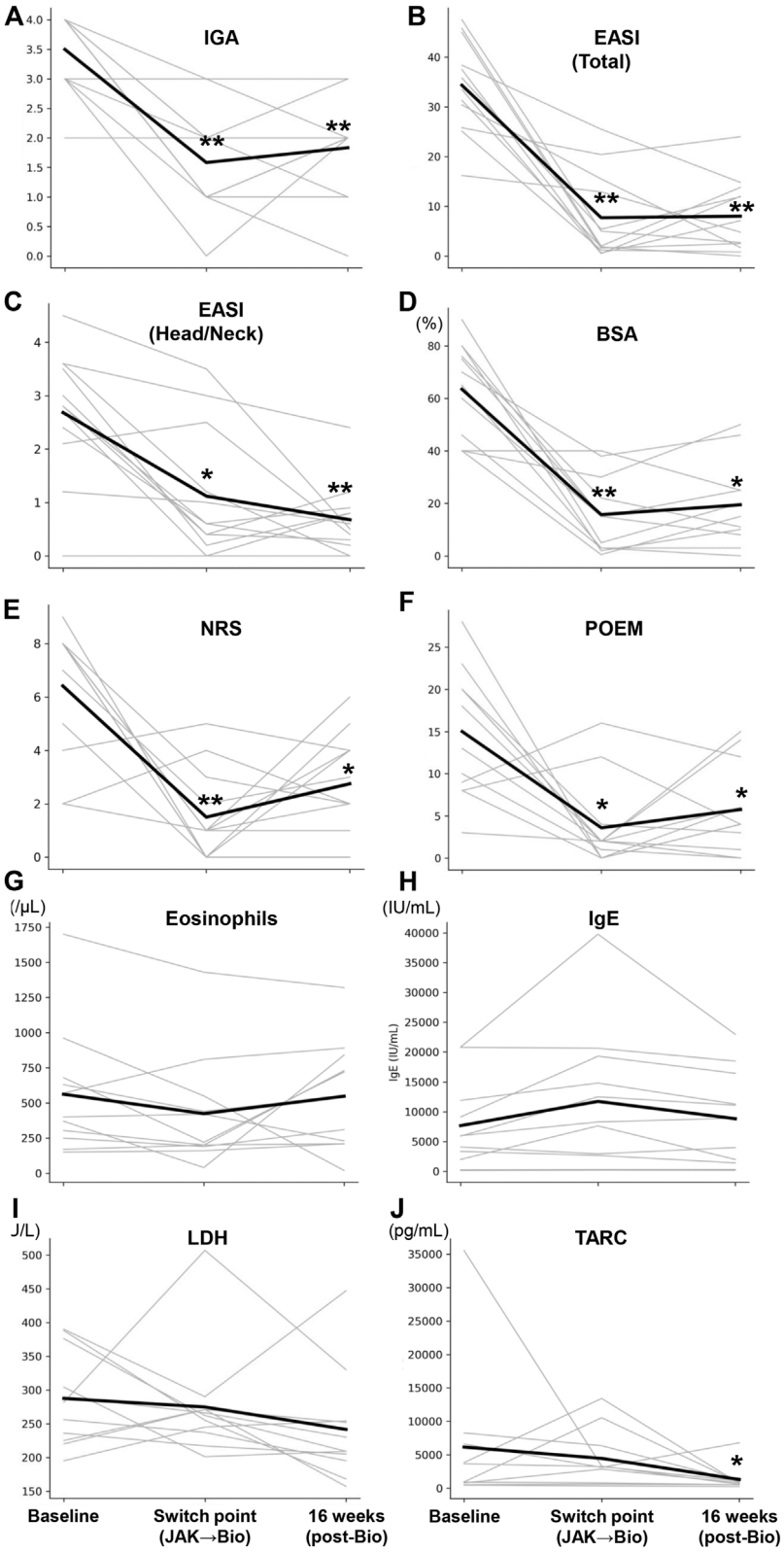


FIGURE 1
Changes in efficacy outcomes and serum biomarkers at the time of switching from upadacitinib to biologic therapy (JAK→Bio) and at 16 weeks after biologic treatment. The line chart shows individual data (gray line) and average (black) changes in each score (n = 10–12). **(A)** Investigator's Global Assessment (IGA). **(B,C)** Eczema Area and Severity Index (EASI) **(B)** total and **(C)** head/neck. **(D)** Affected body surface area (BSA). **(E)** Pruritus numerical rating scale (NRS). **(F)** Patient-oriented eczema measure (POEM). **(G)** Numbers of eosinophils in peripheral blood. **(H)** Serum immunoglobulin (Ig) E. **(I)** Serum lactate dehydrogenase (LDH). **(J)** Serum thymus and activation-regulated chemokine (TARC). Statistical analyses were performed using Friedman's test followed by Dunn's multiple comparisons test using GraphPad Prism nine software. *$p < 0.05$, **$p < 0.01$.

upadacitinib treatment period, the seven patients who transitioned to biologic therapy after 12 weeks of upadacitinib treatment were separately analyzed at weeks 0, 4, 12 (time of switching), 16 (4 weeks after switching), and 28 (16 weeks after switching).

Statistical analysis

Continuous variables are presented as mean ± standard deviation, unless otherwise specified. Changes in clinical scores and laboratory parameters at baseline, at switching, and 16 weeks after switching were analyzed using Friedman's test followed by Dunn's multiple-comparisons test. In the subgroup of seven patients who transitioned after a uniform 12-week course of upadacitinib, clinical scores at each post-treatment time point were compared with baseline values using the Wilcoxon matched-pairs signed-rank test. A two-sided P value < 0.05 was considered statistically significant. Statistical analyses were performed using GraphPad Prism 9 (GraphPad Software, San Diego, CA, USA).

Ethics

This study was approved by the Institutional Review Board of Gunma University (HS2022-169) and was conducted in accordance with the principles of the Declaration of Helsinki.

Results

Patient characteristics and treatment transition

Twelve Japanese patients with moderate-to-severe AD (eight males and four females; mean age, 37.1 ± 15.6 years) were included in the analysis. Baseline demographic and clinical characteristics are summarized in Table 1, and individual treatment details are provided in Table 2. Seven patients received upadacitinib 15 mg and five received 30 mg before switching to dupilumab (n = 6), tralokinumab (n = 2), or lebrikizumab (n = 4). Seven patients received upadacitinib for 12 weeks, whereas five received treatment for more than 12 weeks (mean duration, 10.4 months). The reasons for switching were long-term treatment planning in eight patients (67%) and inadequate response in four patients (33%). Among the four patients who switched because of inadequate response, two were receiving upadacitinib 15 mg and switched to biologic therapy without dose escalation because of concerns about increasing the dose to 30 mg. All patients continued upadacitinib until the day before initiation of biologic therapy, with no washout period between treatments.

Clinical outcomes after switching to biologic therapy

Changes in clinical outcomes and laboratory biomarkers at baseline, at the time of switching, and 16 weeks after switching are shown in Figure 1. Physician-assessed clinical scores, including IGA, total EASI, head/neck EASI, and BSA, as well as patient-reported outcomes, including pruritus NRS and POEM, significantly improved during upadacitinib treatment compared with baseline. Overall clinical improvement was generally maintained at 16 weeks after switching to biologic therapy.

However, mild worsening of skin lesions and patient-reported outcomes was observed after switching. The EASI-75 achievement rate decreased from 67% (8/12) during upadacitinib treatment to 50% (6/12) at 16 weeks after switching. Three patients who had previously achieved EASI-75 no longer met this criterion after switching to dupilumab, tralokinumab, and lebrikizumab (one patient each), whereas one patient who switched to dupilumab newly achieved EASI-75 (Table 2). Among the six patients who switched to dupilumab, one lost EASI-75 response, one newly achieved EASI-75, and two experienced disease flare requiring temporary oral cyclosporine. Among the two patients who switched to tralokinumab, one lost EASI-75 response and one experienced disease flare. Among the four patients who switched to lebrikizumab, one lost EASI-75 response, while no disease flare was observed (Table 2).

Similarly, the proportion of patients achieving a ≥4-point improvement in pruritus NRS decreased from 90% (9/10) during upadacitinib treatment to 80% (8/10) at 16 weeks after switching, although this change was not statistically significant.

Disease flare and additional systemic treatment

Transient disease flare occurred shortly after switching in three of the 12 patients (25%), requiring temporary treatment with oral cyclosporine. Cyclosporine was administered at 100 mg/day for 35 days, 100 mg/day for 56 days, and 200 mg/day for 30 days in these three patients, respectively (Table 2). Of the three patients who experienced disease flare, two had switched to dupilumab and one to tralokinumab. Given the small number of patients receiving each biologic agent, no formal comparison of flare risk among biologic therapies was performed.

Temporal course after switching in patients treated with upadacitinib for 12 weeks

To further characterize the temporal course during treatment transition, we separately analyzed the seven patients who switched to biologic therapy after a uniform 12-week course of upadacitinib (Figure 2). Physician-assessed disease severity and patient-reported outcomes improved significantly during the initial 12 weeks of upadacitinib treatment. Four weeks after switching to biologic therapy (week 16), mild worsening of skin lesions was observed, accompanied by deterioration in patient-reported outcomes. At 16 weeks after switching (week 28), both physician-assessed clinical scores and patient-reported outcomes remained significantly improved compared with baseline.

Laboratory biomarkers and adverse events

Peripheral blood eosinophil counts remained largely unchanged throughout the treatment course. Serum IgE levels showed a mild increasing trend during upadacitinib treatment followed by a

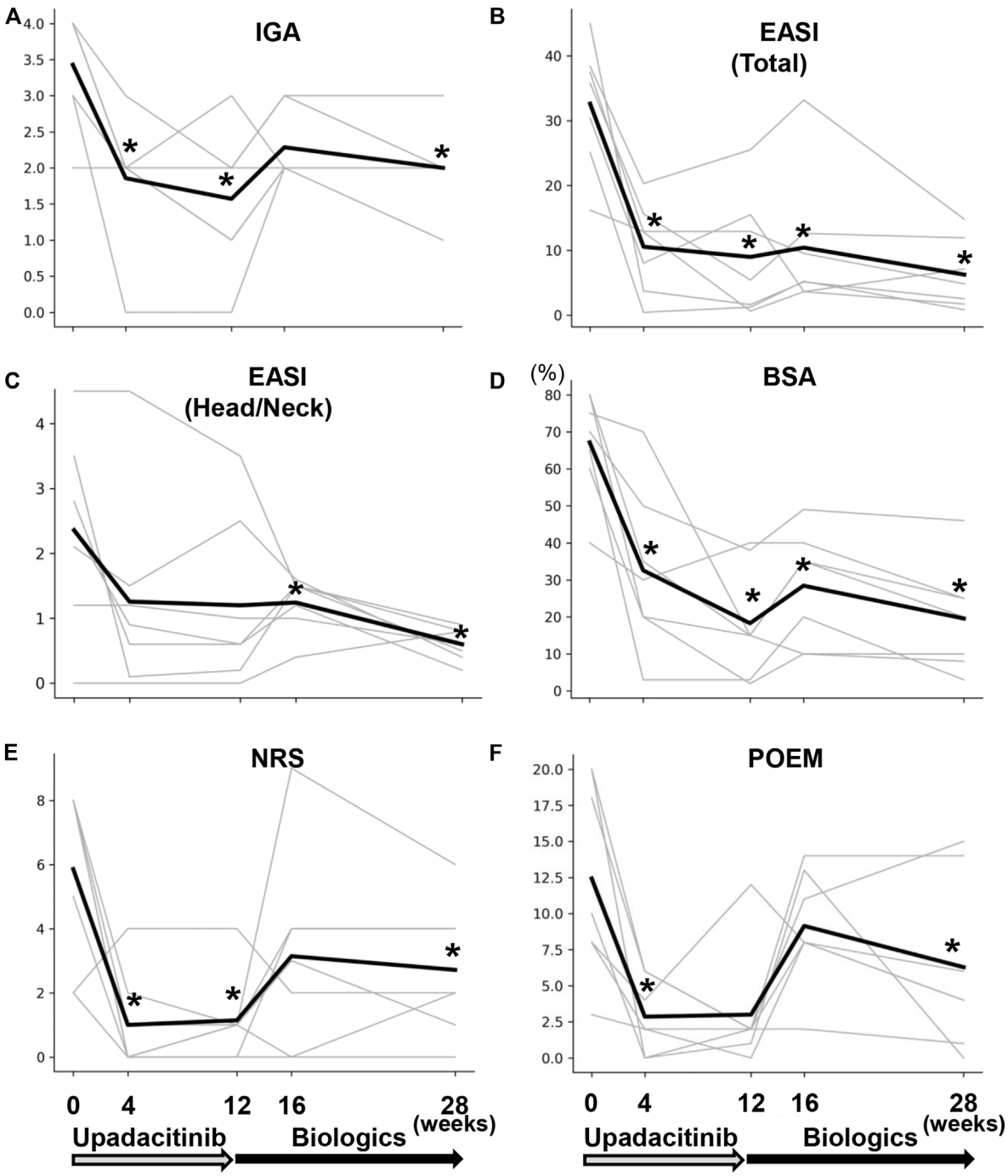


FIGURE 2
Changes in clinical outcomes at weeks 0, 4, 12, 16, and 28. Patients received upadacitinib from weeks 0–12 and biologic therapy from weeks 12–28. The line chart shows individual data (gray line) and average (black) changes in each score (n = 7). **(A)** IGA. **(B)** EASI total and **(C)** EASI head/neck. **(D)** BSA. **(E)** Pruritus NRS. **(F)** POEM. Statistical analyses were performed using the Wilcoxon matched-pairs signed-rank test to compare clinical scores at each post-treatment time point with baseline values. *p < 0.05, **p < 0.01.

decreasing trend after switching, whereas serum LDH and TARC levels showed decreasing trends during both upadacitinib and biologic treatment (Figure 1). During upadacitinib treatment, herpes simplex infection (n = 2), acne (n = 2), herpes zoster (n = 1), and liver dysfunction (n = 1) were observed. No new adverse events occurred during the 16-week observation period after switching to biologic therapy.

Discussion

In this retrospective descriptive case series, we characterized the clinical course after switching from upadacitinib to biologic therapy in patients with moderate-to-severe AD. The principal finding was that overall clinical improvement achieved with upadacitinib was generally maintained after switching, although transient worsening

of disease activity occurred during the early transition period. Three of 12 patients (25%) experienced disease flare requiring temporary oral cyclosporine. In the subgroup of patients who underwent switching after a uniform 12-week course of upadacitinib, worsening was most evident 4 weeks after switching, although clinical outcomes remained significantly improved compared with baseline at 16 weeks after switching. These findings highlight the importance of recognizing the early transition period as a potential window of temporary loss of disease control.

Previous real-world studies have primarily described patients who switched from upadacitinib to biologic therapy because of inadequate treatment response. Hagino et al. reported favorable outcomes after switching from upadacitinib to tralokinumab or lebrikizumab in patients with AD who had responded inadequately to upadacitinib [4, 8]. In contrast, two-thirds of patients in our study switched for long-term treatment planning rather than inadequate response. Importantly, the reasons for switching may have influenced the observed clinical outcomes. Patients who switched because of inadequate response may have had less favorable disease control at the time of switching than those who switched for long-term treatment planning. Therefore, the pooled efficacy outcomes at 16 weeks after switching should be interpreted cautiously, as they may reflect differences in disease status and treatment response before switching rather than the effects of biologic therapy alone. Thus, our study provides additional information on the clinical course after treatment transition in patients who were not necessarily experiencing treatment failure, reflecting the broader circumstances in which treatment switching may be considered in routine clinical practice.

The most clinically relevant observation was the transient worsening shortly after switching. All patients continued upadacitinib until the day before biologic initiation, with no washout period; nevertheless, three patients required temporary oral cyclosporine for disease flare. Upadacitinib has a relatively short terminal elimination half-life of approximately 8–14 h [9], whereas biologic therapies generally require several weeks to achieve their full clinical effect. A similar early exacerbation after switching from upadacitinib to dupilumab has previously been reported [5]. Similarly, Wang et al. reported that four of nine patients who immediately switched from JAK inhibitors to dupilumab experienced disease recurrence within the first four weeks after switching [6]. These findings are consistent with our observations and suggest that disease worsening may occur during the early transition period. This difference in pharmacologic time course may create a temporary therapeutic gap after switching, even when no washout period is used. This interpretation is also consistent with the temporal pattern observed in the seven patients who switched after 12 weeks of upadacitinib treatment, in whom worsening was most apparent four weeks after switching, while clinical outcomes remained significantly improved compared with baseline at 16 weeks after switching. However, this proposed therapeutic gap remains hypothetical. Given the retrospective study design and the absence of a comparator group, we cannot exclude the possibility that the observed worsening reflected natural fluctuations in disease activity or the effects of discontinuing upadacitinib itself. Therefore, a causal relationship between the pharmacological transition and disease worsening cannot be established.

A modest reduction in treatment response was also observed after switching. The EASI-75 achievement rate decreased from 67% during upadacitinib treatment to 50% at 16 weeks after switching, while the proportion of patients achieving a ≥4-point improvement in pruritus NRS decreased from 90% to 80%. These findings are generally consistent with previous network meta-analyses suggesting that upadacitinib may provide somewhat greater clinical efficacy than biologic therapies [10]. Therefore, the potential consequences of treatment transition should be considered on an individual basis. For patients with well-controlled disease on upadacitinib, switching to biologic therapy may be accompanied by a temporary or modest loss of disease control, and patients should be informed of this possibility before treatment transition.

The occurrence of flare also has practical implications for clinical management. In our case series, three patients required oral cyclosporine at 100–200 mg/day for approximately 1–2 months, after which additional systemic treatment could be discontinued. Although the small number of patients precludes identification of predictors of flare or meaningful comparisons among dupilumab, tralokinumab, and lebrikizumab, these observations suggest that close monitoring during the first several weeks after switching is warranted. Appropriate use of topical therapy and, when necessary, temporary additional systemic treatment may be considered to manage disease worsening during the transition period.

Long-term studies have provided accumulating evidence regarding the sustained efficacy and safety of both JAK inhibitors and biologic therapies in patients with AD [3]. In our study, no new adverse events occurred during the 16-week observation period after switching. However, none of the patients discontinued upadacitinib specifically because of adverse events. Therefore, our findings should not be interpreted as demonstrating a safety advantage of switching to biologic therapy or a clinical benefit of avoiding long-term JAK inhibitor treatment. Treatment selection should take into account disease control, treatment goals, individual safety considerations, and patient preferences.

This study has several limitations. First, the small sample size limited statistical power and precluded meaningful comparisons among individual biologic agents. The small sample size, particularly the limited number of patients with available pruritus NRS data, also reduced the statistical power to detect clinically meaningful changes. Therefore, the absence of statistically significant differences in pruritus NRS should not be interpreted as evidence of symptom stability or equivalence between treatments. Second, this was a single-center retrospective descriptive case series without a comparator group; therefore, the transient worsening observed after switching cannot be definitively attributed to the treatment transition itself. Third, the study population was clinically heterogeneous in terms of upadacitinib dose and treatment duration, subsequent biologic agents, and reasons for switching. Although we separately analyzed patients who received upadacitinib for a uniform duration of 12 weeks, other sources of heterogeneity remained. Therefore, the pooled findings should be interpreted as descriptive observations of treatment transitions in routine clinical practice rather than as evidence of consistent outcomes across different clinical subgroups. Fourth, the reasons for switching

varied among patients, including long-term treatment planning and inadequate response, which may have influenced subsequent clinical outcomes. Finally, the 16-week follow-up period after switching was relatively short, and longer-term studies are needed to determine whether disease control remains stable over time.

In conclusion, this small retrospective case series suggests that clinical improvement may persist after switching from upadacitinib to biologic therapy in patients with moderate-to-severe AD, although transient worsening requiring additional systemic treatment was observed. Careful monitoring, particularly during the first several weeks after switching, is warranted. Larger prospective studies are needed to identify patients at increased risk of flare and to establish optimal strategies for transitioning from JAK inhibitors to biologic therapy.

Data availability statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Ethics statement

The studies involving humans were approved by the Institutional Review Board of Gunma University (HS2022-169). The studies were conducted in accordance with local legislation and institutional requirements. The participants provided their written informed consent to participate in this study.

Author contributions

AU and S-iM contributed to the study design. AU, KK, TA, MI, and AK were involved in patient care and contributed to data collection and analysis. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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