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Original Article

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Consensus-Based Guidelines for the Treatment of Atopic Dermatitis in Korea (Part II): Biologics and JAK inhibitors

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ABSTRACT

Background: Atopic dermatitis (AD) is a common skin disease with a wide range of symptoms. Due to the rapidly changing treatment landscape, regular updates to clinical guidelines are needed.
Objective: This study aimed to update the guidelines for the treatment of AD to reflect recent therapeutic advances and evidence-based recommendations.
Methods: The Patient characteristics, type of Intervention, Control, and Outcome framework was used to determine 48 questions related to AD management. Evidence was graded, recommendations were determined, and, after 2 voting rounds among the Korean Atopic Dermatitis Association (KADA) council members, consensus was achieved.

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Results: This guideline provides treatment guidance on advanced systemic treatment modalities for AD. In particular, the guideline offers up-to-date treatment recommendations for biologics and Janus-kinase inhibitors used in the treatment of patients with moderate to severe AD. It also provides guidance on other therapies for AD, along with tailored recommendations for children, adolescents, the elderly, and pregnant or breastfeeding women.
Conclusion: KADA's updated AD treatment guidelines incorporate the latest evidence and expert opinion to provide a comprehensive approach to AD treatment. The guidelines will help clinicians optimize patient-specific therapies.

Keywords: Atopic dermatitis; Biologics; Consensus; Guideline; Janus Kinase inhibitors; Republic of Korea

INTRODUCTION

The Korean Atopic Dermatitis Association (KADA) developed a clinical severity classification specific to the Korean population in 2019[1] and updated systemic treatment guidelines for moderate to severe atopic dermatitis (AD) in 2021[2]. Recent approvals of biologics and Janus-kinase (JAK) inhibitors in Korea have necessitated updates to incorporate these advanced therapies, offering targeted and personalized treatment options.

Part II of these guidelines is designed to provide evidence-based recommendations on advanced systemic therapies including biologics and JAK inhibitors, to support dermatologists in managing moderate to severe AD with current and tailored strategies.

MATERIALS AND METHODS

The KADA task force team consisting of 10 dermatologists, representing AD experts performed extensive, up-to-date literature reviews on management for AD.

Based on the comprehensive literature review, the task force team established a total of 48 questions using the Patient characteristics, type of Intervention, Control, and Outcome (PICO) framework regarding AD management and requested expert opinions on each of these questions (**Supplementary Table 1**).

Database and literature searches

The task force team conducted a comprehensive search of various databases, including PubMed, Scopus, the Cochrane Library, and KoreaMed. This search encompassed articles published until December 31, 2023. The search queries employed a combination of keywords: "atopic eczema," "atopic dermatitis," "antihistamine," "antimicrobial," "antifungal," "antiviral," "biologics," "dupilumab," "lebrikizumab," "tralokinumab," "nemolizumab", "omalizumab," "JAK inhibitors," "abrocitinib," "upadacitinib," "baricitinib," "phototherapy," "allergen-specific immunotherapy," "probiotics," "prebiotics," "vitamin D," "essential fatty acid," "small molecule inhibitors," "children," "adolescent," "adult," "elderly," "pregnancy," and "breastfeeding." In addition to database searches, the team also conducted manual searches by reviewing the reference lists of relevant systematic reviews and guidelines issued by other research groups. Through these efforts, the team compiled all pertinent statements related to the management of AD.

Evaluation of the literature

The quality of evidence was assessed, and the strength of each PICO-based recommendation was then determined. Evidence for each statement was graded using the following system: level 1a, systematic review (with homogeneity) of randomized controlled trials (RCTs); level 1b, individual RCT (with narrow confidence interval); level 1c, all or none; level 2a, systematic review (with homogeneity) of cohort studies; level 2b, individual cohort study (including low-quality RCTs); level 2c, "outcome" research; level 3a, systematic review (with homogeneity) of case–control studies; level 3b, individual case–control study; level 4, case series (and poor-quality cohort and case-control studies); and level 5, expert opinion.

The modified Grading of Recommendations Assessment, Development and Evaluation system was applied to determine recommendation strength. Beyond the level of evidence and balance of benefits and harms, factors such as feasibility in primary care, acceptability, and degree of utilization were carefully considered. Recommendations were categorized as follows: A, strong recommendation for using an intervention. The benefits of this intervention significantly outweigh potential harms (generally recommended); B, weak recommendation for using an intervention. The benefits of this intervention outweigh potential harms, but there is some uncertainty (recommended selectively); C, weak recommendation against using an intervention. The harms of this intervention outweigh benefits, but there is some uncertainty (not recommended unless there are specific considerations); D, strong

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recommendation against using an intervention. The harms of this intervention significantly outweigh benefits (generally not recommended).

Consensus process

Fifty-six KADA council members were invited to express their level of agreement with each draft statement, using a voting scale from 1 to 10 (where 1 indicated strong disagreement and 10 indicated strong agreement). Voting scores were categorized into 3 groups: 1 to 3 (disagreement), 4 to 6 (neutrality), and 7 to 10 (agreement). Consensus was defined as achieving at least 70% of votes in the 7 to 10 range, indicating agreement. The consensus recommendation process included 2 rounds of voting to finalize the guidelines.

RESULTS

Biologics

1) Dupilumab

> We recommend the use of dupilumab in adult, adolescent, and pediatric patients over 6 months of age with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 98%).

Dupilumab, a fully human immunoglobulin G4 (IgG4) monoclonal antibody targeting the interleukin (IL)-4 receptor α, inhibits IL-4 and IL-13 signaling, thereby reducing T helper cell–mediated inflammation, decreasing immunoglobulin E (IgE) production, and improving skin barrier function.[3,4] In Korea, dupilumab is approved for the treatment of moderate to severe AD in adults (aged 18 and above), adolescents (12–17 years), and children (6 months–11 years) for whom topical therapies are not adequately controlled or are not advisable[1]. It is also approved for severe eosinophilic asthma and chronic rhinosinusitis with nasal polyps, which are type 2 inflammatory diseases, and in the United States, it has received approval for eosinophilic esophagitis and nodular prurigo.

Phase 3 trials (LIBERTY AD SOLO1, SOLO2, CHRONOS, CAFÉ) showed significant improvements in severity scores (Eczema Area and Severity Index [EASI], Investigator's Global Assessment [IGA], SCORing Atopic Dermatitis), pruritus, and quality of life[5-7]. The LIBERTY AD SOLO1 and SOLO2 studies reported 48% achieving EASI-75 at week 16 (vs. 13% placebo)[7]. In an open-label extension study, long-term data showed that 90.9% of patients achieved EASI-75 after 4 years[8].

A meta-analysis of 22 real-world studies involving 3,303 patients with AD reported a 59.8% achievement rate of EASI-75 at week 16[9]. Real-world efficacy in Korea aligns with that of other countries[10,11], with 56.1% achieving EASI-75 at 16 weeks of dupilumab administration, and 90.2% at 52 weeks[12]. Predictive factors for reduced efficacy include male sex, high eosinophil levels, and elevated lactate dehydrogenase[13]. Differential diagnoses, such as contact dermatitis, scabies, or cutaneous T-cell lymphoma, should be considered if dupilumab is ineffective[14].

For adults, dupilumab is administered as a 600 mg loading dose followed by 300 mg every 2 weeks. It can be combined with topical corticosteroids (TCSs), topical calcineurin inhibitors (TCIs), or phototherapy, etc. In refractory cases, adding oral immunomodulators or JAK inhibitors can be helpful[15,16].

Dupilumab is well-tolerated[8], with common side effects including conjunctivitis, upper respiratory infection, herpes simplex, injection site reaction, and headache. Some patients may experience eye-related side effects (keratitis, conjunctivitis, dry eye, ocular pruritus, blepharitis), which could be managed based on severity. Mild cases can be treated with warm compresses, artificial tears, sodium hyaluronate, or antihistamine eye drops, whereas moderate to severe cases may require tacrolimus, cyclosporine, corticosteroid, lifitegrast eye drops[17]. Paradoxical head and neck erythema can typically appear within 6 months, potentially caused by contact dermatitis, seborrheic dermatitis, rosacea, and withdrawal from topical steroids[18-20].

2) Tralokinumab

> We recommend the use of tralokinumab in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 97%).

Tralokinumab, a fully human, high-affinity IgG4 monoclonal antibody targeting IL-13, reduces the inflammatory response in AD[21]. It is approved for moderate to severe AD in adults and adolescents whose disease is inadequately controlled by topical therapies or when such therapies are not advisable. Tralokinumab was approved by the European Medicines Agency (EMA) and the U.S. Food and Drug Administration in 2021, and by Korea's Ministry of Food and Drug Safety (MFDS) in 2023.

Phase 3 trials confirmed tralokinumab's efficacy in improving skin clearance, reducing itch, and enhancing quality of life in AD[22,23]. ECZTRA 1 and 2 monotherapy achieved EASI-75 in 25.0% and 33.2% of patients (vs. 12.7% and 11.4% placebo)[22], while ECZTRA 3, with TCS as needed, showed higher rates of EASI-75 (56.0% vs. 35.7%) at week 16[23]. A 2-year open-label extension study reported sustained improvements, with 82.5% achieving EASI-75[24]. Real-world evidence also supports the efficacy and safety of tralokinumab treatment[25].

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Tralokinumab is administered with a 600 mg loading dose, followed by 300 mg every 2 weeks (Q2W) via subcutaneous injection[23]. It has a favorable safety profile, with common side effects including viral infections, upper respiratory infections, conjunctivitis, and injection site reactions[26]. Ocular complications are less frequent than with dupilumab. Long-term safety data (up to 2 years) show no new concerns, and no specific monitoring is required.

3) Lebrikizumab

> We recommend the use of lebrikizumab in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: A, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 97%).

Lebrikizumab, a high-affinity IgG4 monoclonal antibody targeting soluble IL-13, inhibits a key pathway in AD by preventing IL-4Rα/IL-13Rα1 heterodimer receptor signaling complex formation[27]. It was approved by EMA in 2023, and MFDS in Korea in 2024.

Phase 3 trials (ADvocate1, ADvocate2[27], and ADhere[28]) demonstrated significant improvements in moderate to severe AD. EASI-75 was achieved at week 16 in 52.1% and 58.8% of monotherapy participants (vs. 18.1% and 16.2% placebo)[27]. When combined with TCS, lebrikizumab showed potential for a higher level of efficacy[28].

Lebrikizumab is administered with a 500 mg loading dose at baseline and week 2, followed by 250 mg Q2W. Common adverse events include conjunctivitis, headache, hypertension, injection site reactions, and herpes infections, mostly mild or moderate[29]. Lebrikizumab effectively reduces AD severity and improves quality of life.

4) Nemolizumab

> We suggest considering the use of nemolizumab in patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: B, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 86%).

Nemolizumab, a humanized monoclonal antibody targeting the IL-31 receptor alpha, treats pruritus in AD[3]. Approved in Japan in 2022[30], it demonstrated significant efficacy in phase III trials, reducing pruritus VAS scores by 42.8% (vs. 21.4% placebo) and EASI scores by 45.9% (vs. 33.2% placebo)[31]. Long-term benefits were sustained up to 68 weeks, even after treatment cessation[32].

The approved dose in Japan is 60 mg subcutaneously every 4 weeks (Q4W). Reported adverse events include AD exacerbation, injection site reactions, cytokine abnormalities, headache, edema, and elevated creatine phosphokinase (CPK)[31,32]. Nemolizumab shows potential for AD and pruritus, but further real-world data are needed.

5) Omalizumab

> We propose limited use of omalizumab for patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: C, Grade of evidence: 3b, % of respondents [agreement score ≥7]: 34%).

Omalizumab, a humanized monoclonal anti-IgE antibody, targets allergic asthma and chronic spontaneous urticaria by neutralizing IgE. While elevated IgE is common in AD, its role in pathogenesis remains unclear[33].

Several controlled and uncontrolled studies, as well as case series and reports, have indicated varying degrees of therapeutic success with better efficacy in patients with lower IgE levels than those with very high levels[33-35]. The ADAPT study found modest improvements in disease severity and quality of life in pediatric AD patients with high IgE levels[36], but small sample sizes and strong placebo effects limit conclusions.

Doses range from 150–600 mg every 2–4 weeks. Omalizumab is generally well-tolerated, with common side effects including injection site reactions, headaches, and sinusitis. Due to variable responses, we suggest limited use of omalizumab for patients with moderate to severe AD.

6) Selective addition of systemic immunosuppressants or oral JAK inhibitors

> We propose considering the selective addition of systemic immunosuppressants or oral JAK inhibitors for patients with moderate to severe AD who are not adequately controlled by biologic agents (Recommendation strength: C, Grade of evidence: 4, % of respondents [agreement score ≥7]: 74%).

AD is a type 2 inflammatory disease with diverse phenotypes and endotypes, leading to variable responses to biologic agents targeting type 2 inflammation. Some patients may experience insufficient improvement, such as persistent head and neck erythema, despite dupilumab treatment. Case reports suggest improved outcomes and quality of life with combinations of systemic immunosuppressants (e.g., cyclosporine, methotrexate [MTX]), phototherapy, or add-on treatment with JAK inhibitors to biologics in AD[16,37]. Korean studies have reported baricitinib add-on therapy as effective for aggravation, hyper-eosinophilia, or

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insufficient response to dupilumab[16]. As these combinations are off-label, careful patient monitoring and thorough explanation are essential[14]. Larger prospective studies are needed to identify suitable candidates for add-on therapy.

7) Switching to another biologic agent or an oral JAK inhibitor

We suggest considering switching to another biologic or oral JAK inhibitor in patients with moderate to severe AD if there is an insufficient response* to biologic therapy or an inability to use current biologic treatment due to side effects (Recommendation strength: B, Grade of evidence: biologics 4 / JAK inhibitors 1b, % of respondents [agreement score ≥7]: 96%).

Biologic treatments like dupilumab are effective for moderate to severe AD, but some patients experience insufficient responses or localized persistent symptoms, impacting quality of life. Side effects such as conjunctivitis, blepharitis, and head and neck erythema may also limit treatment continuation. Additionally, the lack of predictive biomarkers and restrictions under the current Korean insurance system including the copayment assistance policy for severe AD, which prevents switching between biologics or JAK inhibitors, poses challenges in managing severe AD.

Switching to upadacitinib or abrocitinib after insufficient response or side effects from dupilumab has shown additional improvement. The Heads Up[38] and JADE EXTEND studies[39], along with cohort and case series, support the benefits of switching from biologics to JAK inhibitors[40-50]. In the JADE-EXTEND trial, over 50% of dupilumab non-responders achieved EASI-90 with abrocitinib[39]. In addition, dupilumab-induced conjunctivitis and dupilumab-associated head and neck dermatitis were successfully treated by switching from dupilumab to upadacitinib[47-50].

Switching from dupilumab to other biologics like tralokinumab may benefit patients with insufficient response or side effects from dupilumab[25,51-54]. Although some of these reports are cohort studies, case series, or case reports, and therefore provide a low level of evidence, these guidelines suggest considering a switch to another biologic agent or an oral JAK inhibitor in patients with moderate to severe AD if there is an insufficient response or an inability to use current biologic treatments due to side effects, based on clinical needs and the aforementioned evidence.

8) Adjustment of administration intervals for biologics

We suggest considering selective dosing intervals according to the patient's symptoms in patients with AD on biologics (Recommendation strength: B, Grade of evidence: tralokinumab 1b/lebrikizumab 1b/dupilumab 4, % of respondents [agreement score ≥7]: 90%).

Biologics for AD, including dupilumab, tralokinumab, and lebrikizumab, may allow dosing interval adjustments based on patient response. Studies show effective maintenance with extended intervals.

The ECZTRA 3 trial found that patients achieving clear or almost clear skin after 16 weeks of tralokinumab could consider Q4W dosing. At week 32, 90.8% maintained EASI-75 with Q4W dosing and TCS, supporting its use as a maintenance option[23]. Similarly, lebrikizumab maintained efficacy with both Q2W and Q4W dosing after a 16-week induction[55]. A real-world study reported that 47.4% of dupilumab-treated patients achieving EASI-75 with Q4W dosing over 16 weeks[56]. These findings support adjusting dosing intervals based on individual patient needs.

JAK inhibitors

1) Baricitinib

We recommend the use of baricitinib, an oral JAK 1/2 inhibitor, in adult patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: A, Grade of evidence: 1a, % of respondents [agreement score ≥7]: 94%).

Baricitinib, a selective JAK1/JAK2 inhibitor, reduces inflammation and regulates immune cell activity. In long-term RCTs, 45.7%–46.3% of patients achieved vIGA-AD 0/1 (0, clear; 1, almost clear) with 2 mg or 4 mg doses at week 16, increasing to 47.1%–59.3% at week 68[57]. In a meta-analysis of five clinical trials, baricitinib 4 mg and 2 mg daily showed slightly less reduction in EASI at 16 weeks compared with dupilumab, with mean differences of −3.2 and −5.2 points, respectively.[58]. This pattern of results was similar for changes in Patient-Oriented Eczema Measure, Dermatology Life Quality Index (DLQI), and peak pruritus numerical rating scale (PP-NRS)[58]. Baricitinib showed faster itch relief compared to dupilumab in indirect comparisons[59].

A direct meta-analysis showed no significant increase in treatment-emergent adverse events versus placebo over 16 weeks[60]. Common side effects included increased low-density lipoprotein (LDL) cholesterol, upper respiratory infections, and headaches[61].

A long-term real-world safety data from 2,636 patients over 3.9 years showed low adverse event rates, with a 3.4% discontinuation rate[62]. Reported events included herpes infections, cardiovascular events, and pulmonary emboli[62].

2) Upadacitinib

We recommend the use of upadacitinib, an oral JAK1 inhibitor, in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates

for topical therapies (Recommendation strength: A, Grade of evidence: 1a (adult)/1b (adolescent), % of respondents [agreement score ≥7]: 96%).

Upadacitinib, a selective JAK1 inhibitor, effectively targets pro-inflammatory cytokine signaling. In a pooled analysis of 11 trials (6,254 patients), upadacitinib 30 mg demonstrated the highest efficacy across endpoints (IGA 0/1, EASI-75/90, PP-NRS 4) at weeks 12–16, followed by abrocitinib 200 mg and upadacitinib 15 mg, though with more adverse events[63]. A long-term treatment (112 weeks) showed consistent safety for both 15 mg and 30 mg doses[64].

In the phase 3b Heads Up study, upadacitinib achieved higher EASI-75 (71%) and EASI-90 (61%) rates at week 16 than dupilumab (61% and 39%, respectively)[65].

Common adverse events included acne, headache, shingles, and upper respiratory infections[66]. In trials with 552 adolescents with severe AD, upadacitinib showed similar safety and efficacy to adults, with dose-related adverse events primarily acne and elevated CPK[67]. These findings highlight a favorable benefit-risk profile for upadacitinib in moderate to severe AD, including adolescents.

3) Abrocitinib

We recommend the use of abrocitinib, an oral JAK1 inhibitor, in adult and adolescent patients with moderate to severe AD who are not adequately controlled by or are not candidates for topical therapies (Recommendation strength: A, Grade of evidence: 1a (adult)/1b (adolescent), % of respondents [agreement score ≥7]: 94%).

Abrocitinib, a selective JAK1 inhibitor, regulates inflammatory cytokines like IL-4, IL-13, IL-31, and interferon, as well as signaling molecules involved in these pathways. In the JADE-REGIMEN trial, 64.7% of the 1,233 patients achieved IGA 0/1 and EASI-75 during a 12-week induction with abrocitinib 200 mg[68]. A meta-analysis of 23 RCTs confirmed high efficacy in EASI-75 and IGA 0/1 improvements with both monotherapy and TCS combination[69]. In a head-to-head trial, abrocitinib 200 mg showed faster itch relief (PP-NRS4 at week 2: 48.2% vs. 25.5%) and higher EASI-90 rates at week 4 (28.5% vs. 14.6%) compared to dupilumab[70].

Common non-serious adverse events include nausea, headache, acne, and herpes infections[71]. A report indicates that abrocitinib transiently reduces platelet count by week 4, which then gradually return to baseline, while cholesterol levels increased dose-dependently without affecting the HDL/LDL ratio[71]. Hemoglobin, neutrophil, and lymphocyte counts showed no significant changes.

4) Monitoring of JAK inhibitors

We recommend periodic monitoring and careful consideration of the benefit-risk ratio when using oral JAK inhibitors for maintenance therapy in patients with moderate to severe AD (Recommendation strength: A, Grade of evidence: 1b, % of respondents [agreement score ≥7]: 96%).

In clinical practice, baseline screening for JAK inhibitors should include complete blood count (CBC), renal and liver function tests, lipid profile, CPK levels, and screening for hepatitis and tuberculosis, including a chest radiograph.

For monitoring, CBC, liver function tests should be checked during routine patient care, with an additional CBC check at 4 weeks for those taking abrocitinib. Lipid profiles should be measured every 3 months, with an additional check at 4 weeks for those taking abrocitinib.

One European guideline practically recommends checking CBC, renal function, liver function, lipid profile, and CPK at 4 weeks after the initiation of therapy and then every 3 months for the duration of all JAK inhibitor treatments[72].

5) Special consideration of comorbidities when using JAK inhibitors

Patients with AD who have coexisting inflammatory conditions such as rheumatoid arthritis, ankylosing spondylitis, and psoriatic arthritis are more likely to benefit from baricitinib and upadacitinib. Abrocitinib, primarily approved for moderate to severe AD, has limited data for other inflammatory diseases.

6) Switching to biologics or another JAK inhibitor

We suggest considering switching to another biologic or oral JAK inhibitor in patients with moderate to severe AD if there is an insufficient response to an oral JAK inhibitor or an inability to use current oral JAK inhibitor due to side effects (Recommendation strength: B, Grade of evidence: 4, % of respondents [agreement score ≥7]: 92%).

For moderate to severe AD, switching to biologics or alternative JAK inhibitors should be considered when oral JAK inhibitors are ineffective or cause side effects. Studies show improved outcomes after such switches: a multicenter cohort found baricitinib non-responders achieved effective responses with upadacitinib[42], and another cohort reported significant disease severity reduction after switching to abrocitinib due to insufficient response or side effects from baricitinib, dupilumab, or tralokinumab[73].

Although some of these studies are cohort studies, and therefore provide a low level of evidence, these guidelines suggest

considering switching to another biologic or oral JAK inhibitor in patients with moderate to severe AD if there is an insufficient response to an oral JAK inhibitor or an inability to use current oral JAK inhibitor due to side effects, based on clinical needs and the aforementioned evidence.

7) Dose adjustment for JAK inhibitors

> We suggest considering selective dose adjustments according to the patient's symptoms in patients with AD on oral JAK inhibitors (Recommendation strength: B, Grade of evidence: 5, % of respondents [agreement score ≥7]: 90%).

A few RCTs have investigated dose reductions of baricitinib (from 4 mg to 4 mg, 2 mg, or placebo) and abrocitinib (from 200 mg to 200 mg, 100 mg, or placebo), primarily noting efficacy recapture upon re-administration during flares[68,74]. Regarding upadacitinib, the usual initial dose is 15 mg in adolescent patients over 40 kg, with 30 mg available for patients with a high disease burden[66]. Further studies on maintenance, dose reduction, and discontinuation strategies for JAK inhibitors in moderate to severe AD are needed.

Requirements to meet when switching agents

Switching medications during advanced AD treatment may be necessary due to insufficient therapeutic benefit, safety concerns, or tolerability issues. There are no universally accepted guidelines, but several consensus recommendations provide guidance. The 2019 consensus Korean diagnostic guidelines define treatment refractory AD as an AD who failed to reach to EASI-50 or at least one of the following conditions after 3 months of appropriate treatment: a. daytime or nighttime itch with NRS score ≥4; b. DLQI ≥6[1]. Further details on treatment goals and recommendations for considering a switch, as provided by other countries and organizations are discussed in the **Supplementary Data 1**.

Other therapies

Various therapies are commonly used in the treatment of AD, including phototherapy, systemic antihistamines, antimicrobial treatments, allergen-specific immunotherapy, and adjunctive therapies such as probiotics, essential fatty acids, and vitamin D. These therapies are discussed in detail in the **Supplementary Data 2** for further reference.

Special considerations for children, adolescents, elderly patients, and pregnant or breastfeeding women

Special considerations for children, adolescents, elderly patients, and pregnant or breastfeeding women in the treatment of AD require tailored approaches due to unique physiological and clinical challenges. These topics are discussed in detail in the **Supplementary Data 3** for further reference.

DISCUSSION

The updated national consensus-based guidelines for the management of AD reflect a careful and comprehensive evaluation of the latest scientific literature, offering an evidence-based approach to optimize patient care. As illustrated in **Fig. 1**, the treatment algorithm is organized into 2 categories: mild AD and moderate to severe AD, with recommendations tailored to disease severity.

For mild AD, the emphasis is placed on basic therapies such as moisturizers, allergen avoidance, and structured educational programs. Topical therapies, including TCSs and TCIs (both reactive and proactive), as well as wet wrap therapy for acute cases, are recommended as primary interventions.

For moderate to severe AD, a broader range of treatments is highlighted to address the complexities of disease management. In addition to basic and topical therapies, systemic options include conventional immunosuppressive agents (e.g., cyclosporine and MTX) and short-term corticosteroids. The guidelines also feature advanced therapies such as biologics and JAK inhibitors, which represent significant advancements in personalized and targeted care for refractory or severe cases. Other therapies, such as phototherapy and allergen-specific immunotherapy, are also discussed as adjunctive options, particularly for certain patient populations. The guideline also provides treatment considerations for a variety of AD patients, including special populations such as children, adolescents, elderly patients and those who are pregnant or breastfeeding.

Treatment accessibility is influenced by reimbursement policies. In South Korea, biologics and JAK inhibitors are reimbursed under strict eligibility criteria, which differ by age group. For adolescents (≥12 years) and adults, eligibility requires severe AD persisting for ≥3 years, failure to respond to ≥4 weeks of moderate-to-high potency TCS or TCI, and inadequate response to ≥3 months of systemic immunosuppressants, with an EASI score ≥23. For children (6–11 years), eligibility applies to severe AD lasting ≥1 year, failure to respond to ≥4 weeks of TCS or TCI, and an EASI score ≥21. For infants and young children (6 months–5 years), the same criteria apply as for children aged 6–11 years, except that the 1-year disease duration requirement is waived. Patients meeting these criteria receive partial reimbursement with a 10% co-payment, while those who do not qualify must cover the full treatment cost. These restrictions significantly impact treatment accessibility, influencing treatment adherence and decision-making in clinical practice.

While these guidelines integrate both RCTs and real-world data, the latter is inherently subject to selection bias, confounding

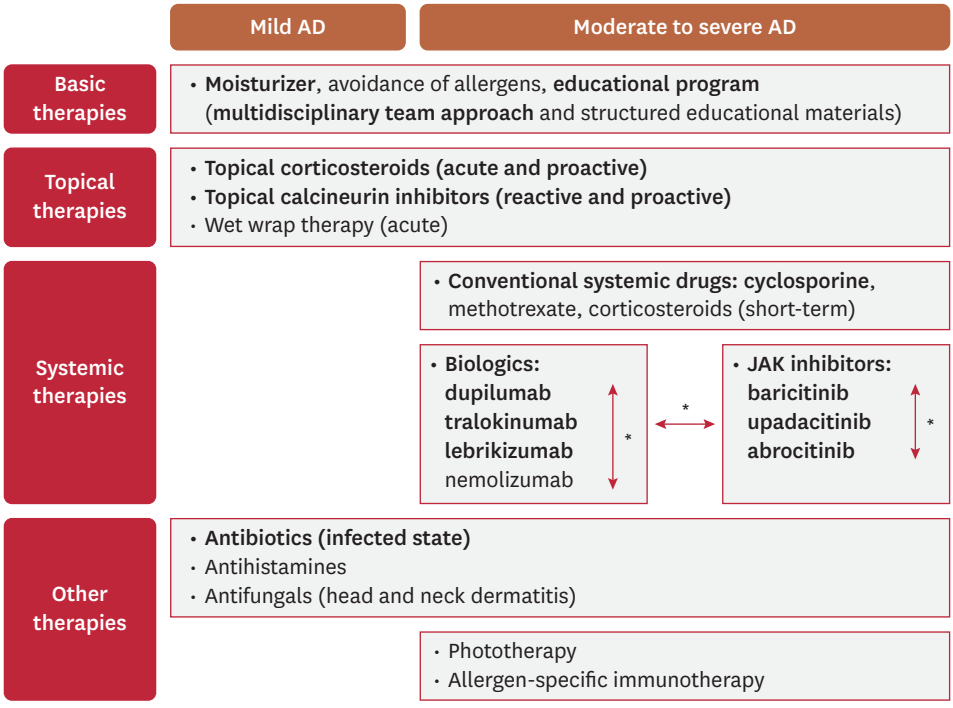

Fig. 1. Treatment algorithm for patients with atopic dermatitis in Korea. The text in bold indicates a treatment with recommendation A (strong recommendation for using an intervention).
AD: atopic dermatitis, JAK: Janus-kinase.
*Switchable in insufficient response† or intolerable due to adverse effects.
†Insufficient response is defined as an AD patient who fails to achieve Eczema Area and Severity Index 50, or meets one or more of the following criteria after 3 months of appropriate treatment: a daytime or nighttime itch numeric rating scale score ≥4, or a Dermatology Life Quality Index ≥6.

variables, and heterogeneity in clinical practice. Consequently, recommendations based on real-world evidence or case series should be interpreted with caution, with explicit acknowledgment of their level of certainty. Further prospective studies and rigorously designed RCTs are required to validate these findings and strengthen evidence-based treatment strategies for moderate to severe AD. As new therapeutic modalities continue to emerge, these guidelines will undergo updates to incorporate the latest advancements in AD management.

In conclusion, KADA's updated guideline is the result of a thorough and careful assessment of the current state of the art in the treatment of AD, based on the latest scientific evidence and expert consensus. We believe that this guideline will serve as an important tool for clinicians, promoting optimized patient care and helping to manage the dynamic landscape of AD.

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DATA SHARING STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

SUPPLEMENTARY MATERIALS

Supplementary Table 1

Expert consensus recommendations for the treatment of AD

Supplementary Data 1

Requirements to meet when switching agents

Supplementary Data 2

Other therapies

Supplementary Data 3

Special considerations for children, adolescents, elderly patients, and pregnant or breastfeeding women

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