

PERSONALIZED MOLECULARLY GUIDED MANAGEMENT OF ATOPIC DERMATITIS: PROGNOSTIC STRATIFICATION AND CLINICAL OUTCOMES IN A 300-PATIENT COHORT

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Abstract. Background: Precision management of atopic dermatitis may be improved by integrating clinical severity, allergen components and FLG genotype. Objective: To summarize outcomes of a personalized algorithm in patients with atopic dermatitis. Methods: Aggregated findings from a 2022–2025 study of 150 personalized-care patients and 150 retrospective comparators were analyzed descriptively. Results: A reported risk model achieved AUC 0.88, sensitivity 84.3% and specificity 81.2%. Excellent immediate responses occurred in 14.7% versus 2.0%; excellent longer-term outcomes in 29.3% versus 6.0%. Six-month sustained remission was reported in 59.8% versus 31.2%. Conclusion: The findings suggest clinical utility but warrant controlled evaluation and external validation.

Keywords: atopic dermatitis; personalized medicine; component-resolved diagnostics; FLG; SCORAD; prognostic model; outcomes

Introduction

Atopic dermatitis (AD) is a heterogeneous, relapsing inflammatory skin disorder (1,2). Differences in epidermal barrier integrity, allergen-specific sensitization and clinical disease burden complicate uniform management strategies. Integrating clinical severity scores, component-resolved allergy testing and filaggrin (FLG) mutation status may support a more individualized treatment pathway.

This study aimed to describe the clinical outcomes and prognostic performance reported for a personalized diagnostic–therapeutic algorithm incorporating molecular sensitization, FLG status, patient age and regional characteristics in patients with AD.

Materials and methods

This manuscript is based on the aggregated results of a 2022–2025 clinical study performed at a republican clinical allergology center in Uzbekistan. The study included 300 patients with AD: a personalized-management group (n=150) and a retrospective conventional-care comparison group (n=150). Therefore, the comparison is non-randomized and susceptible to differences between contemporary and historical patient management.

Assessment involved SCORAD, Dermatology Life Quality Index (DLQI), clinical history, total IgE, component-specific IgE measured by ALEX² and Phadia-200, and FLG mutation analysis including R501X and 2282del4. The source also describes IL-4, transepidermal water loss (TEWL), skin pH and patient-reported symptoms. A short-term multi-indicator clinical response scale and reassessment at 3, 6 and 12 months were used. The published dissertation abstract was the sole dataset for this article; participant-level data and prespecified statistical models were unavailable for reanalysis.

Results

The source reports that a multivariable prognostic model incorporating FLG genotype, molecular sensitization profile, age and residence achieved an area under the receiver operating characteristic curve (AUC) of 0.88, sensitivity of 84.3% and specificity of 81.2%. These estimates are reported as stated in the dissertation abstract; the validation approach and confidence intervals were not available in the summarized source.

Immediate therapeutic response was rated excellent in 22/150 (14.7%) patients under personalized management versus 3/150 (2.0%) patients in the conventional-care comparison. Unsatisfactory immediate responses occurred in 6/150 (4.0%) and 31/150 (20.7%), respectively. The source reported an excellent long-term outcome in 44/150 (29.3%) versus 9/150 (6.0%), while an unfavorable long-term outcome occurred in 18/150 (12.0%) versus 45/150 (30.0%).

In another analysis reported in the abstract, sustained remission at six months was 59.8% in the personalized group and 31.2% in the historical comparison group. A reduction of 53.1% in SCORAD and 46.7% in DLQI was also reported. At 12 months, 67.1% of patients undergoing personalized treatment did not require systemic treatment. A separate summary records a 56.4% SCORAD reduction at 12 weeks; these different estimates refer to separately stated endpoints and should not be treated as identical time points.

*The source labels the middle long-term category “satisfactory,” while its explanatory prose calls it a favorable result. The numeric categories are retained without resolving that inconsistency.

Table 1. Immediate clinical response reported for each study group

Response category	Personalized (n=150)	Comparison (n=150)
Excellent	22 (14.7%)	3 (2.0%)
Good	83 (55.3%)	48 (32.0%)
Satisfactory	39 (26.0%)	68 (45.3%)
Unsatisfactory	6 (4.0%)	31 (20.7%)

Table 2. Reported long-term response categories

Response category	Personalized (n=150)	Comparison (n=150)
Excellent	44 (29.3%)	9 (6.0%)
Intermediate/satisfactory*	88 (58.7%)	96 (64.0%)
Unsatisfactory	18 (12.0%)	45 (30.0%)

Table 3. Summary metrics reproduced from the source

Metric	Reported value
Prediction model	AUC 0.88; sensitivity 84.3%; specificity 81.2%
Sustained remission at 6 months	59.8% vs 31.2%

SCORAD and DLQI improvement	53.1% and 46.7%, respectively
Without systemic therapy at 12 months	67.1% in personalized care

Discussion

The reported differences favor a structured clinical pathway linking symptom burden, barrier-related genetic susceptibility and clinically interpreted molecular sensitization. An important potential advantage is the identification of patients for whom intensified follow-up and individualized allergen counseling may be useful. Component-specific IgE must nevertheless be interpreted in the context of exposure history and clinical reactions; sensitization alone is not synonymous with clinically relevant allergy.

The observational and partly retrospective design prevents causal attribution of all observed improvements to the algorithm. Potential confounding, differences in background treatment, incomplete reporting of loss to follow-up, and unavailable uncertainty estimates may affect the reported effect sizes. The proposed prediction model requires transparent reporting of coefficients, missing-data handling, validation strategy, calibration and external validation before wider clinical adoption.

Conclusion

A personalized management algorithm incorporating FLG genotype, molecular sensitization and clinical severity was associated with more favorable reported outcomes than a historical conventional-care comparator in this 300-patient AD cohort. The prognostic AUC of 0.88 and improvements in clinical outcomes support further prospective evaluation, but do not establish efficacy in the absence of independent validation and randomized or appropriately controlled assessment.

References

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