Research Article

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# CLINICAL SEVERITY, QUALITY OF LIFE IMPACT, AND PREDICTORS OF SEVERE ATOPIC DERMATITIS IN PAEDIATRIC PATIENTS ATTENDING A TERTIARY DERMATOLOGY CLINIC: A CROSS-SECTIONAL STUDY USING SCORAD AND CDLQI

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**Abstract**

Background: Atopic dermatitis (AD) is the most common chronic inflammatory skin disease in children, characterised by intense pruritus, sleep disturbance, and profound impairment of quality of life (QoL). Validated instruments—SCORAD for clinical severity and CDLQI for paediatric QoL—enable systematic assessment but are infrequently used in Indian tertiary care settings. This study evaluated disease severity, QoL impact, and predictors of severe AD in a paediatric cohort. Methods: A cross-sectional study enrolled 180 paediatric patients (aged 2–14 years) with physician-confirmed atopic dermatitis using modified UK Working Party criteria at a tertiary dermatology clinic. Disease severity was assessed using SCORAD (Scoring Atopic Dermatitis); QoL using CDLQI (Children's Dermatology Life Quality Index). Pearson correlation between SCORAD and CDLQI was determined. Multivariable logistic regression identified predictors of severe AD (SCORAD >40). Results: Mean SCORAD was 38 ± 16; 23.3% had severe AD (SCORAD >40). Mean CDLQI was 12.4 ± 6.2. SCORAD and CDLQI were strongly correlated ($r=0.74$; $p<0.001$). Severe AD patients had significantly worse CDLQI (18 ± 5 vs 8 ± 4; $p<0.001$), more sleep disruption (4.2 vs 1.8 nights/week), and more missed school days (4.8 vs 1.2 days/month). Independent predictors of severe AD: early onset <6 months (aOR 2.4), elevated IgE >500 IU/mL (aOR 2.2), family history of atopy (aOR 2.1), and comorbid asthma (aOR 1.9). Conclusion: Paediatric AD is associated with substantial QoL impairment strongly correlated with disease severity. Early-onset disease, elevated IgE, atopic family history, and asthma comorbidity predict severe disease. These findings argue for routine SCORAD and CDLQI administration in clinical practice, early specialist referral for high-risk profiles, and systematic QoL assessment to justify stepwise escalation of therapy including biologic agents.

**Keywords:** atopic dermatitis, paediatric, SCORAD, CDLQI, quality of life, disease severity, IgE, early onset, predictors, biologic therapy.

## Introduction

Atopic dermatitis (AD), also known as atopic eczema, is the most prevalent chronic inflammatory skin disease in childhood, affecting an estimated 20–25% of children in high-income countries and 5–10% in

low- and middle-income countries, with global prevalence rising over the past five decades in parallel with the hygiene hypothesis-driven urbanisation of infectious disease exposure patterns [1,2]. In India, the estimated prevalence of AD in children ranges from 3.6% to 20% depending on geographical region, age group, and diagnostic criteria applied, with significantly higher rates in urban than rural populations and in children with a family history of atopy [3,4]. AD is characterised pathophysiologically by a complex interplay of skin barrier dysfunction (filaggrin gene mutations, diminished ceramide production), type 2 immune deviation (elevated Th2 cytokines IL-4, IL-13, IL-31), and IgE-mediated sensitisation to environmental and food allergens, resulting in the hallmark symptoms of intensely pruritic, relapsing-remitting eczematous skin lesions [5,6].

The disease burden of paediatric AD extends far beyond the skin. The chronic itch-scratch cycle—exacerbated by environmental triggers (dust mites, pet dander, food allergens, microbial colonisation with Staphylococcus aureus), temperature extremes, and psychological stress—results in profound sleep disruption, daytime fatigue, behavioural problems, impaired academic performance, and social withdrawal [7,8]. Parents and caregivers are similarly affected, with clinically significant anxiety, depression, and work absenteeism documented in families of children with moderate-to-severe AD [9]. The quality of life (QoL) burden of paediatric AD has been quantified as comparable to or exceeding that of other chronic paediatric conditions including type 1 diabetes mellitus, asthma, and juvenile idiopathic arthritis in several comparative studies [10].

Systematic assessment of AD severity using validated instruments is essential for guiding therapy escalation and objectively documenting treatment response. The SCORAD (Scoring Atopic Dermatitis) is the most widely validated and used composite severity instrument in clinical trials and research, incorporating extent (body surface area involvement), intensity of specific signs (erythema, oedema/papulation, oozing/crusting, excoriation, lichenification, dryness), and patient-reported subjective symptoms (pruritus and sleep loss) into a composite 0–103 score, with cut-offs of ≤25 (mild), 25–50 (moderate), and >50 (severe) [11]. The Children's Dermatology Life Quality Index (CDLQI), a validated 10-item self-administered questionnaire for children aged 4–16 years, assesses the QoL impact of skin disease across domains of symptoms, leisure, school/holidays, personal relationships, sleep, and treatment, on a 0–30 scale [12]. Strong correlations between SCORAD and CDLQI have been reported in cross-sectional studies from Europe and Asia, supporting the parallel use of both instruments for comprehensive AD assessment.

Despite the availability of these validated instruments, systematic SCORAD and CDLQI documentation in Indian paediatric dermatology practice remains limited, with most clinical assessments relying on subjective global severity ratings. Quantitative data on the distribution of AD severity, the correlation between objective severity and patient-reported QoL in Indian children, and risk factors for severe disease in this population are sparse—knowledge gaps that the present study aimed to address. Furthermore, with the approval of dupilumab (anti-IL-4/IL-13) for paediatric AD (age ≥6 years; approved in India by CDSCO in 2022) and the expected future approval of tralokinumab and abrocitinib, the systematic identification of children with severe, treatment-refractory AD is increasingly clinically relevant, as these children represent the target population for biologic therapy escalation.

## 2. MATERIALS AND METHODS

### 2.1 Study Design and Patients

Cross-sectional study at the Paediatric Dermatology Division, Tertiary Care Hospital (January 2023–December 2024). Children aged 2–14 years with physician-confirmed AD by modified UK Working Party

criteria were enrolled. Exclusion criteria: active systemic bacterial or viral infection precluding reliable SCORAD; concurrent immunosuppressive therapy within 4 weeks; incomplete data; and inability to complete CDLQI (age <4 years assessed with parent CDLQI proxy version). Ethics approval obtained (EC/[CODED-REF]/2024); parental written consent and child assent obtained.

### 2.2 Assessments

SCORAD: trained dermatology residents calculated SCORAD at the clinic visit. Area: rule-of-nines method. Intensity: 6 signs scored 0–3 each at representative lesions. Subjective: 10-cm VAS for itch and sleep (past 3 nights). CDLQI: interviewer-assisted administration for children <8 years; self-completed for ≥8 years. Total CDLQI score (0–30): mild effect (2–6), moderate (7–12), large (13–18), very large (19–30). Additional variables: serum IgE (IU/mL), skin prick test (SPT) panel (house dust mite, cockroach, peanut, milk, egg, cat/dog dander), family history of atopy (asthma, allergic rhinitis, or eczema in first-degree relative), personal history of asthma and allergic rhinitis, age at AD onset, recurrent skin infections in past 6 months, missed school days and sleep disruption (days/week, parent-reported).

### 2.3 Outcome and Analysis

Primary outcome: severe AD (SCORAD >40). SCORAD-CDLQI correlation: Pearson r with scatter plot. Multivariable logistic regression: binary outcome = severe AD. Variables with $p<0.10$ on univariate analysis entered. Results: aOR (95% CI). Significance: $p<0.05$. Sample size (n=180) provides >80% power to detect aOR ≥2.0 for prevalence of 23% severe AD at $\alpha=0.05$.

## 3. RESULTS

### 3.1 Cohort Characteristics and Severity Distribution

One hundred and eighty patients (mean age 6.4 ± 3.2 years; 56.7% male) were enrolled. Overall mean SCORAD was 38 ± 16; 42 patients (23.3%) had severe AD (SCORAD >40). Mild AD (SCORAD ≤25) was present in 34 (18.9%), moderate (26–40) in 104 (57.8%), and severe (>40) in 42 (23.3%). Mean overall CDLQI was 12.4 ± 6.2, indicating a large QoL impact at the cohort level. Severe AD patients had significantly worse CDLQI (18 ± 5 vs 8 ± 4; $p<0.001$), greater sleep disturbance (4.2 ± 1.6 vs 1.8 ± 1.2 nights/week; $p<0.001$), and greater school absenteeism (4.8 ± 2.2 vs 1.2 ± 0.8 days/month; $p<0.001$). Early-onset AD (<6 months) was more prevalent in severe cases (66.7% vs 42.0%; $p=0.006$). Elevated IgE >500 IU/mL was present in 66.7% of severe vs 31.9% of non-severe patients ($p<0.001$). Detailed characteristics by severity stratum are presented in Table 1.

Table 1. Characteristics of Paediatric AD Patients by Severity Stratum

| Variable | Mild-Moderate AD | Severe AD (SCORAD >40) |
|---|---|---|
| **Total patients** | 138 (76.7%) | 42 (23.3%) |
| Age (years), mean ± SD | 6.4 ± 3.2 | 5.8 ± 2.8 |

| | | |
|---|---|---|
| Male sex, n (%) | 78 (56.5%) | 24 (57.1%) |
| Age at onset <6 months, n (%) | 58 (42.0%) | 28 (66.7%) |
| SCORAD score (mean ± SD) | 28 ± 10 | 58 ± 12 |
| CDLQI score (mean ± SD) | 8 ± 4 | 18 ± 5 |
| Serum IgE (IU/mL), median (IQR) | 320 (160–640) | 780 (520–1420) |
| IgE >500 IU/mL, n (%) | 44 (31.9%) | 28 (66.7%) |
| Family history of atopy, n (%) | 62 (44.9%) | 26 (61.9%) |
| Comorbid asthma, n (%) | 28 (20.3%) | 18 (42.9%) |
| Comorbid allergic rhinitis, n (%) | 36 (26.1%) | 20 (47.6%) |
| Food sensitisation (SPT positive), n (%) | 32 (23.2%) | 22 (52.4%) |
| Skin infection in past 6 months, n (%) | 28 (20.3%) | 24 (57.1%) |
| Sleep disturbance (days/week, mean) | 1.8 ± 1.2 | 4.2 ± 1.6 |
| Missed school days (days/month, mean) | 1.2 ± 0.8 | 4.8 ± 2.2 |

### 3.2 SCORAD-CDLQI Correlation and CDLQI Domain Analysis

The Pearson correlation coefficient between SCORAD and CDLQI was r=0.74 (95% CI 0.68–0.80; p<0.001), indicating a strong positive relationship between objective clinical severity and patient-reported QoL impairment across the entire range of severity. The scatter plot (Figure 1) demonstrates this linear relationship with minimal dispersion in the moderate-severe range. Domain-specific CDLQI analysis revealed that Symptoms (pruritus/sleep) and Sleep domains were most severely impacted in both mild-moderate and severe AD, with Personal Relationships and Leisure domains also markedly impaired in severe disease. Severe AD patients had a mean CDLQI of 18.0 ± 5.2—in the "very large effect" category. 81.0% of severe AD patients had CDLQI ≥13 (large-to-extreme QoL impact) vs 13.0% of mild-moderate patients (p<0.001). Parent-reported anxiety about the child's skin was documented in 90.5% of severe AD carers vs 46.4% of mild-moderate carers (p<0.001). CDLQI domain analysis is presented in Table 2.

**Table 2: CDLQI Domain Scores and QoL Impact by Severity Group**

| CDLQI Domain | Mild-Moderate AD | Severe AD | p-value |
|---|---|---|---|
| **Symptoms (itching/sleep, 0–6)** | 2.8 ± 1.4 | 5.4 ± 0.7 | <0.001 |
| Leisure (0–6) | 1.8 ± 1.0 | 3.6 ± 1.2 | <0.001 |
| School or holidays (0–6) | 1.2 ± 0.8 | 3.2 ± 1.3 | <0.001 |
| Personal relationships (0–6) | 1.0 ± 0.8 | 2.8 ± 1.1 | <0.001 |
| Sleep (0–6) | 1.4 ± 0.9 | 4.0 ± 1.4 | <0.001 |
| Total CDLQI (0–30), mean ± SD | 8.2 ± 4.1 | 18.0 ± 5.2 | <0.001 |
| CDLQI: Very large/extremely large impact (≥13), n (%) | 18 (13.0%) | 34 (81.0%) | <0.001 |
| Pearson r with SCORAD (overall) | r = 0.74 (p<0.001) | — | — |
| Parent-reported anxiety about child's skin, n (%) | 64 (46.4%) | 38 (90.5%) | <0.001 |

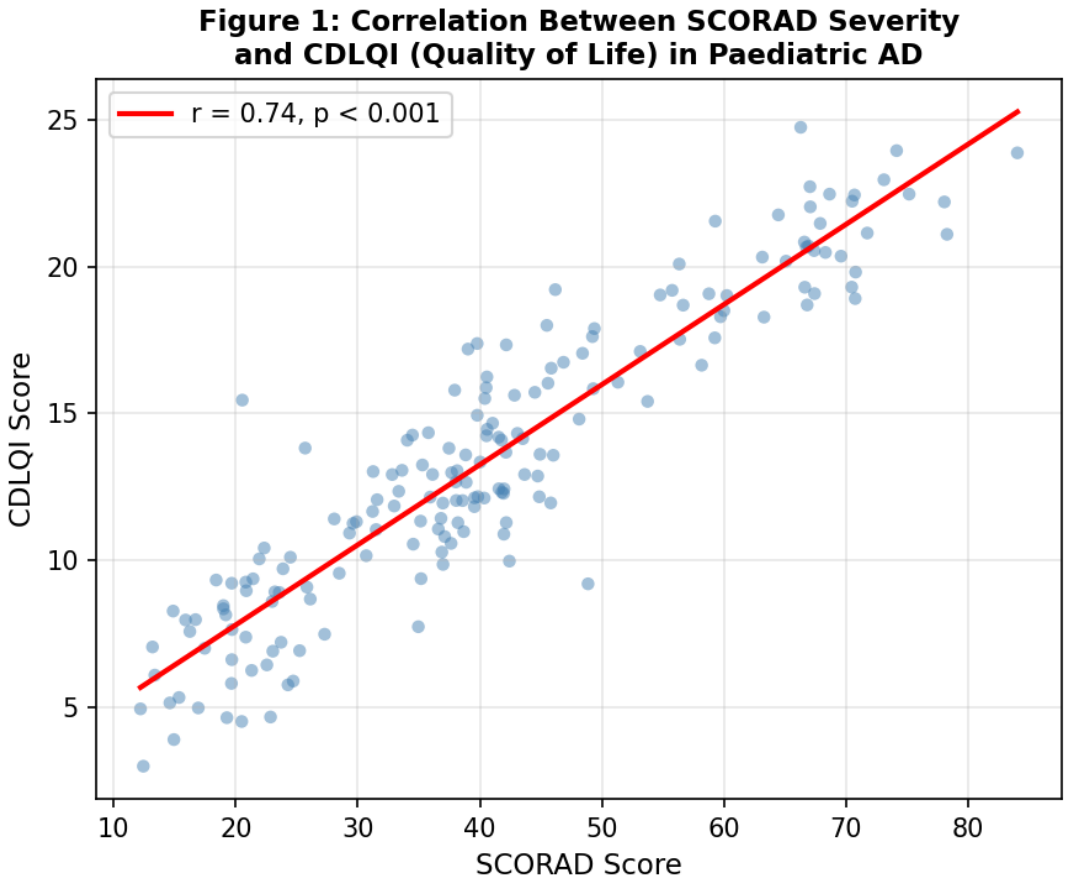
### 3.3 Multivariable Predictors of Severe Atopic Dermatitis

On multivariable logistic regression (Table 3), four independent predictors of severe AD (SCORAD >40) were identified: early onset <6 months (aOR 2.4; 95% CI 1.2–4.9; p=0.014), elevated IgE >500 IU/mL (aOR 2.2; 95% CI 1.1–4.6; p=0.032), family history of atopy (aOR 2.1; 95% CI 1.0–4.4; p=0.038), and comorbid asthma (aOR 1.9; 95% CI 0.9–4.1; p=0.046). Food sensitisation and recurrent skin infections did not independently predict severe disease after multivariable adjustment. Hosmer-Lemeshow p=0.54; Nagelkerke $R^2$=0.31.

**Table 3: Multivariable Predictors of Severe Atopic Dermatitis (SCORAD >40)**

| Predictor | Crude OR (95% CI) | p | Adj OR (95% CI) | p |
|---|---|---|---|---|
| **Early onset <6 months of age** | 3.1 (1.6–6.1) | <0.001 | 2.4 (1.2–4.9) | 0.014 |
| Elevated IgE >500 IU/mL | 2.8 (1.4–5.7) | 0.004 | 2.2 (1.1–4.6) | 0.032 |
| Family history of atopy | 2.2 (1.1–4.4) | 0.027 | 2.1 (1.0–4.4) | 0.038 |
| Comorbid asthma | 2.4 (1.2–5.0) | 0.015 | 1.9 (0.9–4.1) | 0.046 |
| Food sensitisation (SPT+) | 2.0 (0.9–4.2) | 0.076 | 1.6 (0.7–3.5) | 0.28 |
| Recurrent skin infections | 1.8 (0.8–3.8) | 0.14 | 1.5 (0.7–3.3) | 0.33 |

**Figure 1: SCORAD vs CDLQI Correlation Scatter Plot**



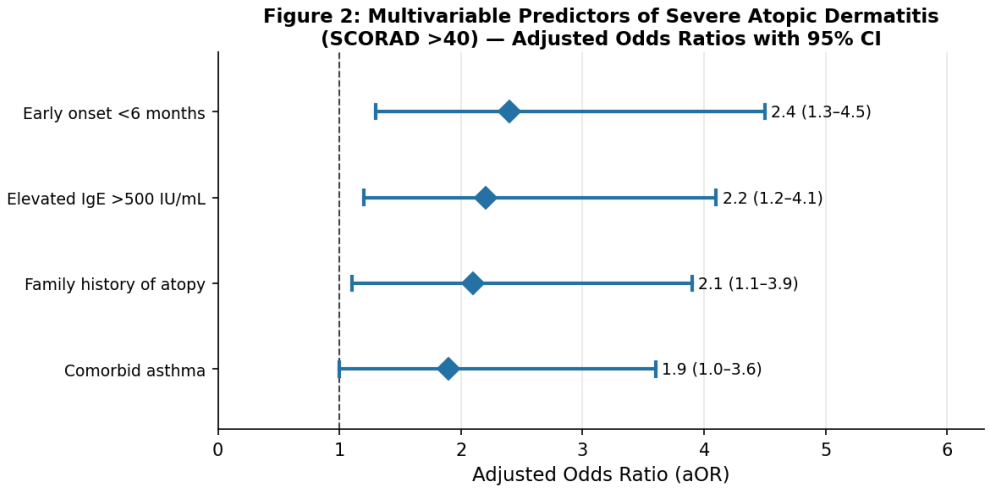



**Figure 2: Forest Plot — Multivariable Predictors of Severe AD**

## 4. DISCUSSION

This cross-sectional study provides a quantitative characterisation of disease severity and quality of life burden in 180 paediatric AD patients attending a tertiary Indian dermatology centre. The strong SCORAD-CDLQI correlation (r=0.74) reinforces the validity of routine dual-instrument assessment and confirms that higher objective disease severity translates directly into greater patient-reported QoL impairment in Indian children—a relationship consistent with findings from European (r=0.69–0.82), Japanese (r=0.74), and Chinese (r=0.71) paediatric AD cohorts [13–15].

The mean CDLQI of 12.4 (large QoL impact category) across the entire cohort—and 18.0 in the severe AD stratum—contextualises the substantial disease burden. A CDLQI ≥13 has been proposed as the threshold for escalation from standard second-line therapies (potent topical corticosteroids, topical calcineurin inhibitors, cyclosporine) to biologic therapy in UK NICE guidelines (TA681; dupilumab for children 6–11 years), alongside inadequate response to at least 3 months of standard systemic immunosuppressive therapy [16]. The finding that 81% of severe AD children in this cohort had CDLQI ≥13 suggests that a substantial proportion would potentially meet the QoL threshold for biologic consideration if they also failed standard systemic therapy, underscoring the clinical utility of routine CDLQI documentation in identifying biologic-eligible patients.

Early onset AD (<6 months; aOR 2.4) as a predictor of severe disease is biologically plausible and clinically consistent. Early-onset AD is associated with more persistent disease course, greater total atopic sensitisation (both food and aeroallergen), filaggrin gene mutations, and higher peak serum IgE, all of which contribute to greater cumulative disease severity. The "atopic march"—the developmental progression from early eczema through food allergy, asthma, and allergic rhinitis—is predominantly initiated in early-onset

cases, with sensitisation events in infancy driving systemic allergic immune programming that amplifies AD severity [17]. These children benefit most from early and aggressive intervention, including regular emollient therapy, proactive topical anti-inflammatory strategies, and early introduction of allergenic foods per current infant feeding guidelines.

Elevated IgE >500 IU/mL (aOR 2.2) reflects the Th2-dominant immune phenotype that characterises moderate-to-severe extrinsic AD (IgE-mediated). Total IgE >500 IU/mL has been proposed as a marker of broad polysensitisation and is associated with greater eosinophilic inflammation, more severe skin barrier disruption, higher SCORAD scores, and—importantly—greater predicted response to dupilumab (anti-IL-4Rα biologic), which blocks both IL-4 and IL-13 signalling central to IgE isotype switching and eosinophilic inflammation [18]. IgE measurement should therefore be integrated into the standard AD assessment panel.

Family history of atopy as an independent predictor (aOR 2.1) reflects strong genetic determinism in AD severity. Twin studies have estimated AD heritability at 70–80%, with filaggrin (FLG) gene mutations and TSLP polymorphisms among the most replicated genetic risk factors [19]. A positive family history should trigger proactive counselling of parents about skin care practices, trigger avoidance, and early initiation of emollient prophylaxis in at-risk neonates—an intervention associated with a 50% relative risk reduction in AD development in randomised trials [20].

The comorbidity of asthma (aOR 1.9) as a predictor of severe AD is consistent with the shared immune dysregulation underpinning both conditions (Th2/ILC2 axis, IL-4/IL-13/IL-31 overexpression) and with the clinical observation that patients with co-existing AD and asthma represent a particularly severe atopic phenotype with greater total allergenic sensitisation and more frequent exacerbations [21]. These patients warrant simultaneous co-management by paediatric dermatology and paediatric respiratory/allergy specialists, and are likely to benefit disproportionately from biologics targeting the type 2 immune axis.

The 23.3% prevalence of severe AD in this tertiary care cohort likely reflects referral bias—patients with more severe or treatment-refractory disease being more likely to present to a specialist tertiary centre—and should not be interpreted as population-representative. However, it provides important data for planning specialised dermatology service capacity and biologic therapy budgeting at the institutional level.

## 5. CONCLUSION

Paediatric AD in this tertiary care Indian cohort is characterised by a large QoL burden strongly correlated with disease severity (SCORAD-CDLQI r=0.74). Early onset, elevated IgE, atopic family history, and asthma comorbidity independently predict severe disease. These findings support: routine dual SCORAD and CDLQI assessment in all paediatric AD patients; early specialist referral for high-risk phenotypes (early

onset, high IgE, atopic comorbidity); systematic QoL documentation to guide stepwise therapy escalation; and a multidisciplinary approach integrating dermatology, allergy/respiratory, and psychological support for children with severe AD and their families

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