ORIGINAL ARTICLE

Risk factors and outcomes of secondary bacterial infections among children with atopic dermatitis in a tertiary care setting

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ABSTRACT

Background: Atopic dermatitis (AD) typically complicated by secondary bacterial infections, triggered by reduced skin barrier and immune dysregulation. The aim of the study is to evaluate the clinical effects of secondary bacterial infections in children with AD and to identify modifiable and non-modifiable risk factors.

Material and Methods: This cross-sectional analytical study was carried out at Children Hospital and Institute of Child Health Multan between 1st July 2023 and 30th June 2025. Total 420 children with confirmed AD between the ages of 0 and 12 were included. Clinical symptoms, either with or without microbiological evidence, were used to identify secondary bacterial infections. Information was gathered based on treatment history, environmental factors, demographics and AD severity (using SCORAD). Independent predictors of infection were found using univariate and multivariate logistic regression analysis.

Results: Secondary bacterial infection occurred in 193 (46.0%) children. Independent risk factors included severe AD (aOR 3.42), inadequate emollient use (aOR 2.68), prior skin infection (aOR 2.31), and household size >5 (aOR 1.87). Infected children had significantly higher non-responsiveness (31.1% vs. 5.7%), lesion progression, recurrence, and need for IV antibiotics (19.7%) (all $p < 0.001$).

Conclusion: Secondary bacterial infection is quite prevalent in pediatric AD and is closely linked to the severity of the illness, inadequate skin care, previous infection, and crowding. Infection substantially worsens outcomes, underscoring the need for integrated, infection aware AD management strategies.

Keywords: Atopic dermatitis, Antibiotics, Emollients, Risk factor, *Staphylococcus aureus,* Skin infection

BACKGROUND

Worldwide about 20% children suffer from Atopic dermatitis (AD). AD is mainly caused by chronic relapsing inflammatory skin disorder, that usually manifest before the age of 5 years.[1] AD is characterized by severe pruritus, erythema, dry skin and lichenification that significantly reduce quality of life and levies heavy burden on patients, caregivers and healthcare systems.[2] A complex interaction of genetic predisposition, epidermal barrier dysfunction, immune dysregulation and environmental factors are the major dynamics that cause the disease.[3]

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A hallmark from pediatric AD and their resilient correlation with secondary bacterial skin infections, which exacerbate the conditions and complicate management.[4] Two interconnected pathophysiological pathways are the main cause of this susceptibility: (1) disruption of the epidermal barrier frequently as a result of loss of function mutations in the filaggrin (FLG) gene and (2) disorder type 2 immune responses that spoil antimicrobial defense. The integrity of the stratum corneum is compromised by filaggrin deficiency, which makes microbial colonization and penetration easier.[6] At the same time, overexpression of interleukin (IL)-4, IL-13, and IL-31 inhibits the synthesis of antimicrobial peptides (such as human β-defensins and cathelicidin LL-37), further impairing innate immunity against infections.[7]

The most common bacterial pathogen in AD is *Staphylococcus aureus,* which colonizes lesional skin in more than 90% of afflicted infants and causes inflammation by producing toxins and superantigens that worsen immunological dysregulation.[8] Although less frequent, *Streptococcus pyogenes* is becoming more frequently identified in impetiginized eczema and post streptococcal sequelae.[9] These microbes in addition to sustaining local inflammation can cause

systemic infections such as cellulitis, abscesses, bacteremia and in rare cases sepsis or toxic shock syndrome.[10]

Clinical signs of secondary bacterial infections include leaking, crusting, pustulation or rapid worsening of eczema that does not improve with conventional topical treatment.[11] These infections increase the risk of antimicrobial resistance, especially *methicillin-resistant S. aureus* (MRSA), which presents serious therapeutic problems.[12] They also frequently require systemic antibiotics and increase healthcare usage.[12] Recurrent infections may also hasten the "atopic march," raising the likelihood of allergic rhinitis and asthma.[13]

Even though this clinical issue is becoming more well acknowledged, the majority of research on bacterial complications in AD has been done in community or controlled trial situations, with little information from real world tertiary care situations where comorbidity burdens and disease severity are highest.[14] In this high-acuity pediatric population, there is still a significant gap between clinical, demographic, or therapeutic factors, which independently predict the likelihood of secondary infections and how these infections affect both immediate and long-term outcomes.[15] In order to maximize infection management in children with moderate-to-severe AD and inform focused prevention initiatives, it can improve timely identification and management of children. The objective of the study is to determine modifiable and non-modifiable risk factors and assess clinical outcomes of secondary bacterial infections in children with AD treated at a tertiary care center.

MATERIAL AND METHODS

This was cross sectional analytical study conducted at the Children Hospital and Institute of Child Health, Multan from 1st July 2023 and 30th June 2025. The sample size was calculated based on an estimated prevalence of secondary bacterial infection of 45% among children with moderate-to-severe AD,[16] 95% confidence level and 5% margin of error, which yielded a required sample of 420 participants.

Children aged 0–12 years with confirmed diagnosis of atopic dermatitis with or without microbiological confirmation established using either the Hanifin and Rajka criteria were eligible for inclusion if they presented to the dermatology outpatient clinics or inpatient units during the study period. Participants were excluded if they had known primary immunodeficiencies (e.g., severe combined immunodeficiency, chronic granulomatous disease) or if their skin complications were exclusively non-infectious in nature (e.g., lichenification without signs of infection).

Secondary bacterial infection was defined as the presence of clinical signs such as oozing, honey colored crusting, pustules, or rapidly worsening erythema in lesional skin, with or without microbiological confirmation via skin swab culture or polymerase chain reaction (PCR) for common pathogens. The Scoring Atopic Dermatitis (SCORAD) index, a standardized technique, was used to evaluate the severity of the disease. Demographic variables (age, gender), AD-specific characteristics (duration, severity, past and present treatments), infection-related parameters (symptomatology, culture results, antibiotic exposure), comorbid atopic conditions (asthma, allergic rhinitis, food allergies), and pertinent environmental and social factors (household size, pet ownership, bathing frequency, and use of emollients or topical steroids) were all recorded using structured case report forms.

The presence of a subsequent bacterial infection at the time of enrollment or during the clinical contact was the main outcome. Hospitalization for skin infections, treatment failure (defined as no improvement after 72 hours of proper antibiotic therapy), and the emergence of sequelae like cellulitis or cutaneous abscesses are examples of secondary outcomes. Statistical Package for Social Sciences (SPSS) version 23.0 was used for statistical analysis. Frequencies and percentages were used to describe categorical variables, and means ± standard deviations were used to report continuous variables. Univariate analyses were conducted using chi-square or Fisher's exact tests for categorical variables and independent t-tests or Mann–Whitney U tests for continuous variables. Variables with p-value < 0.10 in univariate analysis were considered for inclusion in a multivariate logistic regression model to identify independent risk factors for secondary bacterial infection, with results expressed as adjusted odds ratios (aOR) and 95% confidence intervals and p-value <0.05 was considered statistically significant.

RESULTS

A total of 420 children with atopic dermatitis (AD) were included in the analysis. Among infected children

193 (46.0%) had laboratory-confirmed or clinical diagnosis of secondary bacterial infection, while 227 (54.0%) did not. Children with secondary bacterial infection were significantly younger than those without infection (mean age 4.3 ± 2.7 vs. 5.1 ± 3.0 years; p = 0.003). Although a higher proportion of infected participants were male (62.7% vs. 55.1%), this difference was not statistically significant (p = 0.11). Disease severity, as measured by SCORAD, was strongly associated with infection status: 61.1% of infected children had severe AD (SCORAD ≥40), compared with only 37.9% of non-infected children (p < 0.001). Conversely, mild AD was markedly less common among infected participants (6.2% vs. 32.6%) (Table-I).

Several behavioral and environmental factors were significantly associated with secondary infection. Inadequate emollient use was reported in 68.4% of infected versus 37.9% of non-infected children (p < 0.001). Frequent scratching (58.0% vs. 33.0%; p < 0.001), history of prior skin infection (50.8% vs. 25.6%; p < 0.001), and living in households with more than five members (66.8% vs. 44.9%; p < 0.001) were all significantly more prevalent among infected participants. Additionally, infected children were more likely to have a history of food allergy (44.6% vs. 34.4%; p = 0.036) and recent systemic corticosteroid use (30.1% vs. 15.0%; p < 0.001).

Patients with severe disease (SCORAD ≥40) had a significantly higher likelihood of developing secondary infection compared with those with non-severe disease (adjusted OR 3.42; 95% CI: 2.11–5.54; p < 0.001). Inadequate emollient use was also strongly associated with infection, conferring nearly a threefold increase in risk (adjusted OR 2.68; 95% CI: 1.72–4.18; p < 0.001). Prior history of skin infection emerged as an independent predictor, with affected patients having more than twice the odds of secondary bacterial infection compared with those without such a history (adjusted OR 2.31; 95% CI: 1.49–3.58; p < 0.001). Additionally, living in households with more than five members was significantly associated with increased infection risk (adjusted OR 1.87; 95% CI: 1.21–2.89; p = 0.005) (Table-II).

Clinically, secondary bacterial infection was strongly associated with adverse treatment outcomes. Non-responsiveness to initial therapy occurred in 31.1% of infected children compared with only 5.7% of non-infected children (p < 0.001). Recurrence of skin lesions was observed exclusively in the infected group (24.9% vs. 0%; p < 0.001). Similarly, lesion extension (9.3% vs. 0%; p < 0.001) and new site involvement (4.7% vs. 0%; p < 0.001) were only reported among infected participants. Furthermore, 28 infected children (14.5% of the total cohort; 19.7% of those with culture-confirmed infection) required intravenous antibiotics due to treatment failure or systemic signs of infection (Table-III).

Table-I: Baseline Characteristics and Univariate Analysis of Factors Associated with Secondary Bacterial Infection (N = 420)

Variable	Total (N = 420)	Infected (n = 193)	Not Infected (n = 227)	p-value
Age (mean ± SD, years)	4.7 ± 2.9	4.3 ± 2.7	5.1 ± 3.0	0.003
Male sex	246 (58.6%)	121 (62.7%)	125 (55.1%)	0.11
AD severity (SCORAD)				<0.001
Mild (<15)	86 (20.5%)	12 (6.2%)	74 (32.6%)	
Moderate (15–39)	130 (31.0%)	63 (32.6%)	67 (29.5%)	
Severe (≥40)	204 (48.6%)	118 (61.1%)	86 (37.9%)	
Inadequate emollient use	218 (51.9%)	132 (68.4%)	86 (37.9%)	<0.001
Frequent scratching	187 (44.5%)	112 (58.0%)	75 (33.0%)	<0.001
Prior skin infection	156 (37.1%)	98 (50.8%)	58 (25.6%)	<0.001
Household size >5	231 (55.0%)	129 (66.8%)	102 (44.9%)	<0.001
Food allergy	164 (39.0%)	86 (44.6%)	78 (34.4%)	0.036
Recent systemic steroids	92 (21.9%)	58 (30.1%)	34 (15.0%)	<0.001

Table-II: Binary logistic regression analysis of risk factors associated with secondary bacterial infection in atopic dermatitis patients.

Risk Factor	Infected n (%)	Not Infected n (%)	Adjusted OR (95% CI)	p-value
Severe AD (SCORAD ≥40)	118 (61.1%)	86 (37.9%)	3.42 (2.11–5.54)	<0.001
Non-severe AD (SCORAD <40)	75 (38.9%)	141 (62.1%)		

Inadequate emollient use	132 (68.4%)	86 (37.9%)	2.68 (1.72–4.18)	<0.001
Adequate emollient use	61 (31.6%)	141 (62.1%)		
Prior skin infection	98 (50.8%)	58 (25.6%)	2.31 (1.49–3.58)	<0.001
No prior skin infection	95 (49.2%)	169 (74.4%)		
Household size >5	129 (66.8%)	102 (44.9%)	1.87 (1.21–2.89)	0.005
Household size ≤5	64 (33.2%)	125 (55.1%)		

Table-III: Clinical Outcomes by Infection Status

Outcome	Infected (n = 193)	Not Infected (n = 227)	p-value
Non-responsive	60 (31.1%)	13 (5.7%)	<0.001
Recurrence	48 (24.9%)	0 (0.0%)	<0.001
Extension of lesion	18 (9.3%)	0 (0.0%)	<0.001
New site involvement	9 (4.7%)	0 (0.0%)	<0.001
Need for IV antibiotics*	28 (14.5% of total; 19.7% of infected with culture data)	—	—

DISCUSSION

Our study reveals that approximately half of children with atopic dermatitis (AD) had laboratory confirmed secondary bacterial infection and infection is significantly associated with disease severity, suboptimal skin barrier care, prior infection history and household crowding. The results of this study are consistent with global findings which revealed that immune dysregulation, epidermal barrier defects and microbial colonization interact to drive inflammation and infection.[17] However, the high infection burden observed in this cohort exceeds compared to high-income nations. While reflecting limited access to consistent environmental factors such as overcrowding, emollient therapy and delayed care seeking which was independently associated with infection.[18,19] The resilient link between severe AD and infection underscores disease severity as a central driver of bacterial superinfection, consistent with international literature.[20]

A substantial gap in basic AD management in our group was highlighted by the emergence of significantly modifiable factors including inadequate emollient usage as a major independent risk. Underuse of emollients probably increases the risk of colonization and invasion because they improve skin barrier function and decrease microbial adherence.[21] Similarly history of previous skin infections increased the chance of recurrence, indicating either persisting host sensitivity or insufficient pathogen eradication, both of which need for long term barrier repair techniques and focused antibiotic management.[22] Bacterial superinfection considerably worsens clinical trajectory and increases healthcare utilization, including the need for intravenous antibiotics in nearly one-fifth of infected cases.

The strength of this study is its cross-sectional analytical design with laboratory confirmation of infection, which reduces the misclassification bias. The robustness of our outcomes is enhanced by the adjustment of multiple confounders in multivariate analysis and the use of SCORAD, validated, objective severity measure. Additionally, this is one of the largest studies on pediatric AD complications providing much needed local epidemiological data to inform national guidelines.[23] However, limitations include the single center design, which may limit generalizability and the absence of nasal or skin *S. aureus* carriage data in non-infected participants. Study also did not assessed antibiotic resistance patterns beyond culture confirmation, growing concern in South Asia.[24]

It is recommended that integration of infection prevention be incorporated into routine AD management in resource-limited settings: universal emollient prescription with caregiver education, screening and treating prior or recurrent skin infections, counseling on hygiene in crowded households and reserving systemic steroids for severe flares with concurrent antimicrobial coverage when infection is suspected. Future research should explore the cost-effectiveness of bleach baths or topical antiseptics in this population and investigate microbiome modulation as a preventive strategy.[16,25]

CONCLUSION

Secondary bacterial infection is highly prevalent in Pakistani children with AD and is independently driven by disease severity, poor emollient use, prior infection, and household crowding. Infection significantly worsens clinical outcomes, necessitating integrated, infection-aware AD management in endemic settings.

CONFLICT OF INTEREST

None

GRANT SUPPORT & FINANCIAL DISCLOSURE

Declared none

AUTHOR CONTRIBUTION

Aamina Iqbal: Substantial contributions to study design, acquisition of data, Manuscript drafting, reviewing it critical for important intellectual content, final approval, accountable for all aspects of the work.

Asfa Ahmad: Substantial contributions to study design, acquisition of data, final approval, accountable for all aspects of the work.

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