

Poor Prognosis of Scabies in Atopic Children in Bangladesh

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ABSTRACT

Background: Scabies is a significant pediatric health problem in Bangladesh, with atopic children often presenting with disproportionately severe symptoms and delayed recovery. Underlying atopic conditions may worsen clinical manifestations and affect treatment outcomes, yet evidence on their prognostic impact remains limited. Aim of the study: To evaluate whether atopic children in Bangladesh have a poorer prognosis of scabies compared with non-atopic children. **Methods & Materials:** This prospective observational study enrolled 120 children aged 0–14 years with clinically diagnosed scabies between 01.02.2025 to 30.10.2025 at the Department of Dermatology, Ashiyan Medical College Hospital, Dhaka, Bangladesh and Enam Medical College and Hospital, Dhaka, Bangladesh. Participants were stratified into atopic ($n = 60$) and non-atopic ($n = 60$) groups based on clinical history, family history, and laboratory parameters (total serum IgE and eosinophil counts). Data on demographics, clinical features, environmental risk factors, laboratory investigations, treatment response, and outcomes were collected using structured forms. All children received standard first-line therapy with topical permethrin $\pm$ antihistamines. Statistical analyses included t-tests, Chi-square tests, and multivariate logistic regression to identify independent predictors of poor prognosis. **Result:** Atopic children demonstrated significantly greater disease severity, with higher pruritus scores (8.2 ± 1.2 vs. 6.5 ± 1.5 , $p < 0.001$), larger body surface area involvement ($22.4\% \pm 11.0$ vs. $14.8\% \pm 8.5$, $p < 0.001$), and increased rates of nocturnal itching, sleep disturbance, eczematization, and secondary bacterial infection. Response to first-line therapy

was lower in atopic children (complete response: 50% vs. 70%, $p = 0.003$), requiring more treatment cycles and longer time to clinical improvement (9.5 ± 4.2 vs. 5.8 ± 3.1 days, $p < 0.001$). Atopic status independently predicted poor prognostic outcomes, including prolonged disease (>4 weeks) and secondary infection. Environmental factors such as shared bedding, prior family exposure, and poor hygiene were more prevalent in the atopic group. **Conclusion:** Atopic children experience significantly worse scabies outcomes in Bangladesh, with more severe disease and delayed recovery. Early identification and tailored management are essential to improve prognosis.

Keywords: scabies, atopy, poor prognosis, pediatric dermatology, IgE, eosinophilia, Bangladesh, treatment response.

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INTRODUCTION

Scabies is a highly contagious skin infection that occurs mainly in tropical regions and subtropical areas and has been responsible for widespread outbreaks and epidemics worldwide for many years [1]. It affects more than 565 million people worldwide each year, with children being the most commonly affected. Globally, scabies is responsible for approximately 4.84 million disability-adjusted life-years [2]. In Bangladesh, almost 77% of children have experienced scabies at some point [3]. Aetiologically, scabies is a contagious skin condition caused by the mite *Sarcoptes scabiei* var. *hominis*. The infestation spreads through close and prolonged skin-to-skin contact with an affected person or by sharing personal items that have become contaminated with the mites [4]. Scabies infestation can become worse when bacteria infect the affected skin and this often results in painful skin sores that may spread if not treated properly. Over time, these secondary infections can lead to serious health problems, including

septicemia, long-term heart complications and acute glomerulonephritis, chronic kidney disease, making early detection and treatment essential [5]. Overcrowded homes, sleeping in close contact, sharing clothes or towels, poor personal hygiene, inadequate nutrition, and travel to areas where scabies outbreaks are common all contribute significantly to the risk of acquiring the infection [6,7]. The pattern of skin diseases varies widely between countries [8, 9]. In contrast, dermatological disorders such as acne, warts, and various eczematous conditions are reported more frequently in Western countries, largely due to differences in climate, lifestyle patterns, cultural habits, and broader socioeconomic factors across regions [9–11]. To prevent transmission, it is essential to avoid direct skin-to-skin contact with individuals affected by scabies until both they and their close contacts have completed appropriate treatment. Clothing, bedding, and towels used by an infected person should be washed in hot water and thoroughly dried in sunlight or on a high-heat cycle. Regular

cleaning and vacuuming of living spaces, furniture, and floors are also crucial to eliminate mites and reduce the risk of reinfestation [12, 13]. The disease imposes significant social, psychological, and economic burdens on affected families. Moreover, identifying predictors of poor prognosis in atopic children may enable clinicians to implement early, aggressive management, potentially reducing the burden of chronic or recurrent scabies. This study aims to evaluate the impact of atopy on the clinical course and prognosis of scabies among children in Bangladesh.

METHODS & MATERIALS

This was a prospective observational study conducted between 01.02.2025 to 30.10.2025 at the Department of Dermatology, Enam Medical College and Hospital, Dhaka, Bangladesh. The study was approved by the institutional ethics committee, and written informed consent was obtained from parents or legal guardians prior to enrollment.

A total of 120 children aged 0–14 years with clinically diagnosed scabies were enrolled and stratified into two groups: atopic children (n = 60) and non-atopic children (n = 60).

Inclusion & Exclusion Criteria

Inclusion Criteria:

- Children aged 0–14 years with clinically diagnosed scabies.
- Atopic status determined by history of atopic dermatitis, asthma, allergic rhinitis, family history of atopy, or elevated total serum IgE and eosinophil count.
- Children whose parents or guardians provided written informed consent.

Exclusion Criteria:

- Children with chronic systemic illnesses or immunodeficiency.
- Children who received scabies treatment within four weeks prior to enrollment.
- Children with severe concurrent dermatological conditions that could confound assessment.

Data Collection

Demographic data, clinical features, and household/environmental factors were recorded using a structured case report form. Variables collected included age, gender, residence, socioeconomic status, family history of atopy, total serum IgE, eosinophil counts, duration and severity of scabies symptoms, body surface area involvement, number of classical lesions, pruritus severity (0–10 visual analog scale), nocturnal itching, sleep disturbance, eczematization, secondary bacterial infection, and inflammation intensity.

Environmental risk factors such as overcrowding, shared bedding or clothing, poor hygiene, and previous family exposure were also documented.

Laboratory Investigations

Peripheral blood samples were collected to measure total serum IgE levels and absolute eosinophil counts using standard laboratory techniques. These parameters were analyzed to assess atopic sensitization and immune profile differences between groups.

Treatment Protocol

All participants received standard first-line therapy, consisting of topical 5% permethrin cream applied overnight, with oral or topical antihistamines administered as needed. Treatment response was classified as complete, partial, or treatment failure based on clinical resolution of symptoms and lesions. Treatment cycles, time to clinical improvement, treatment intolerance (skin irritation or flare), and the need for systemic therapy (oral antibiotics, ivermectin, or corticosteroids) were documented.

Outcome Measures

Primary outcomes included disease severity, treatment response, and time to full clinical cure. Secondary outcomes comprised recurrence/relapse within 4–8 weeks, prolonged disease duration (>4 weeks), hospitalization, sleep disturbance severity, and quality-of-life impairment assessed using the Children’s Dermatology Life Quality Index (CDLQI). Environmental and household risk factors associated with disease severity and recurrence were also evaluated.

Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and compared using Student’s t-test or Mann–Whitney U test, as appropriate. Categorical variables were presented as frequencies and percentages and analyzed using Chi-square or Fisher’s exact test. A p-value <0.05 was considered statistically significant. Multivariate logistic regression was performed to identify independent predictors of poor prognosis, including prolonged disease, treatment failure, and secondary infection.

RESULT

A total of 120 children with scabies were enrolled in the study, of whom 60 were identified as atopic and 60 as non-atopic. Specifically, the mean age was 6.9 ± 3.3 years overall, with no significant difference between atopic and non-atopic children (6.7 ± 3.1 vs. 7.0 ± 3.4 years; p = 0.58). Similarly, male predominance and urban residence were observed in both group without statistically significant differences. The socioeconomic status was predominantly lower class (45.83%). All atopic children had atopic dermatitis (100.00%), with higher rates of asthma (25.00% vs 13.33%) and allergic rhinitis (20.00% vs 11.67%) than non-atopic children. Family history of atopy (33.33% vs 16.67%), serum IgE (620±200 vs 280±150 IU/mL), and eosinophil count (0.88±0.30 vs 0.56±0.28 ×10⁹/L) were significantly higher in atopic children (p<0.05) (*Table 1*).

Table 1
Baseline demographic and atopic profile of the Study Population.

Variable	Total (N=120)	Atopic Children (n=60)	Non-Atopic Children (n=60)	p-value
Age (years)				
0–1	12 (10.00)	6 (10.00)	6 (10.00)	1
1–4	48 (40.00)	24 (40.00)	24 (40.00)	1
5–14	60 (50.00)	30 (50.00)	30 (50.00)	1
Mean ± SD	6.9 ± 3.3	6.7 ± 3.1	7.0 ± 3.4	0.58
Gender				
Male	68 (56.67)	35 (58.33)	33 (55.00)	0.71
Female	52 (43.33)	25 (41.67)	27(45.00)	
Socioeconomic status				
Low	55 (45.83)	30 (50.00)	25 (41.67)	0.44
Middle	44 (36.67)	22 (36.67)	22 (36.67)	
High	21 (17.50)	8 (13.33)	13 (21.67)	
Atopic profile				
Atopic dermatitis	60 (50.00)	60 (100.00)	0(0.00)	-
Asthma	23 (19.17)	15 (25.00)	8 (13.33)	0.12
Allergic rhinitis	19 (15.83)	12 (20.00)	7 (11.67)	0.18
Family history of atopy	30 (25.00)	20 (33.33)	10 (16.67)	0.03*
Total serum IgE, IU/mL				
Mean ± SD	450 ± 220	620 ± 200	280 ± 150	<0.001*
Eosinophil count (×10 ⁹ /L)				
Mean ± SD	0.72 ± 0.34	0.88 ± 0.30	0.56 ± 0.28	<0.001*

Atopic children had more severe scabies, with longer symptom duration (18.5 ± 9.4 vs 12.2 ± 7.1 days, $p=0.001$), greater body surface area involvement ($22.4 \pm 11.0\%$ vs $14.8 \pm 8.5\%$, $p<0.001$), and more classical lesions (45 ± 18 vs 28 ± 15 , $p<0.001$).

Pruritus was higher (8.2 ± 1.2 vs 6.5 ± 1.5 , $p<0.001$), with more nocturnal itching (83.33% vs 58.33% , $p<0.001$) and sleep disturbance (75.00% vs 36.67% , $p<0.001$). Eczematization (35.00% vs 13.33% , $p=0.002$), secondary bacterial infection

(45.00% vs 16.67% , $p<0.001$), and moderate-to-severe inflammation (61.67% vs 25.00% , $p<0.001$) were also more frequent in atopic children (Table II).

Table II

Comparison of disease severity between atopic and non-atopic children with scabies.

Variable	Atopic (n=60)	Non Atopic (n=60)	p-value
Duration of symptoms (days), mean $\pm$ SD	18.5 ± 9.4	12.2 ± 7.1	0.001*
Body surface area involved (%), mean $\pm$ SD	22.4 ± 11.0	14.8 ± 8.5	<0.001*
Number of classical lesions, mean $\pm$ SD	45 ± 18	28 ± 15	<0.001*
Pruritus severity (0–10), mean $\pm$ SD	8.2 ± 1.2	6.5 ± 1.5	<0.001*
Nocturnal itching, n (%)	50 (83.33)	35 (58.33)	<0.001*
Sleep disturbance, n (%)	45 (75.00)	22 (36.67)	<0.001*
Eczematization, n (%)	21 (35.00)	8 (13.33)	0.002*
Secondary bacterial infection, n (%)	27 (45.00)	10 (16.67)	<0.001*
Inflammation intensity—moderate/severe, n (%)	37 (61.67)	15 (25.00)	<0.001*

Complete response occurred in 50.00% of atopic children versus 70.00% of non-atopic children ($p=0.003$), while partial response was 33.33% versus 20.00%, and treatment failure was 16.67% versus

10.00%. Atopic children required more treatment cycles (1.8 ± 0.9 vs 1.2 ± 0.5 , $p<0.001$) and longer time to clinical improvement (9.5 ± 4.2 vs 5.8 ± 3.1 days, $p<0.001$). Treatment intolerance was higher

in atopic children (13.33% vs 3.33% , $p=0.01$), and 21.67% required systemic therapy versus 8.33% of non-atopic children ($p=0.01$) (Table III).

Table III

Treatment patterns and response to first-line therapy in atopic versus non-atopic children with scabies.

Variable	Atopic (n=60)		Non Atopic (n=60)		p-value
	n	%	n	%	
Treated with standard first-line permethrin $\pm$ antihistamine	60	100.00	60	100.00	—
Response to first-line therapy					
Complete	30	50.00	42	70.00	0.003*
Partial	20	33.33	12	20.00	
None / treatment failure	10	16.67	6	10.00	
Number of treatment cycles required					
Mean $\pm$ SD	1.8 ± 0.9		1.2 ± 0.5		<0.001*
Time to clinical improvement (days)					
Mean $\pm$ SD	9.5 ± 4.2		5.8 ± 3.1		<0.001*
Treatment intolerance (skin irritation/flare), n (%)	8	13.33	2	3.33	0.01*
Need for systemic therapy (oral antibiotics / ivermectin / steroids), n (%)	13	21.67	5	8.33	0.01*

Secondary infection during follow-up occurred in 38.33% of atopic children versus 13.33% of non-atopic children ($p<0.001$). Mean time to full cure was longer in atopic children (24.3 ± 10.5 vs

17.6 ± 8.4 days, $p<0.001$), and prolonged disease (>4 weeks) occurred in 31.67% versus 15.00% ($p=0.02$). Sleep disturbance severity (6.8 ± 2.1 vs 4.1 ± 2.3 , $p<0.001$) and quality-of-life impairment (DLQI 10.2 ± 3.5

vs 6.5 ± 2.8 , $p<0.001$) were higher in atopic children (Table IV).

Table IV

Prognostic outcomes and indicators of poor prognosis in atopic and non-atopic children with scabies.

Outcome Variable	Atopic (n=60)		Non Atopic (n=60)		p-value
	n	%	n	%	
Secondary infection during follow-up	23	38.33	8	13.33	<0.001*
Recurrence / relapse within 4–8 weeks	17	28.33	10	16.67	0.09
First-cycle treatment failure	10	16.67	6	10.00	0.23
Time to full cure (days)					
Mean $\pm$ SD	24.3 ± 10.5		17.6 ± 8.4		<0.001*
Prolonged disease (>4 weeks)	19	31.67	9	15.00	0.02*
Hospitalization required	3	5.00	0	0.00	0.06
Sleep disturbance severity (0–10)					
Mean $\pm$ SD	6.8 ± 2.1		4.1 ± 2.3		<0.001*
Quality-of-life impairment (Children's DLQI)†					
Mean $\pm$ SD	10.2 ± 3.5		6.5 ± 2.8		<0.001*

Table V depicts that overcrowding (≥ 3 persons/room) was present in 71.67% of atopic children versus 58.33% of non-atopic children ($p=0.06$). Shared

bedding/clothing (55.00% vs 36.67%, $p=0.01$), previous scabies exposure in the family (45.00% vs 30.00%, $p=0.04$), poor hygiene index (53.33% vs 33.33%,

$p=0.007$), and a higher number of family members infected (1.9 ± 1.1 vs 1.2 ± 0.8 , $p=0.002$) were all more common among atopic children.

Table V
Environmental and household risk factors associated with scabies in atopic and non-atopic children.

Variable	Atopic (n=60)		Non Atopic (n=60)		p-value
	n	%	n	%	
Overcrowding (≥ 3 persons/room)	43	71.67	35	58.33	0.06
Shared bedding/clothing	33	55.00	22	36.67	0.01*
Previous scabies exposure in family	27	45.00	18	30.00	0.04*
Poor hygiene index	32	53.33	20	33.33	0.007*
Number of family members infected (mean $\pm$ SD)	1.9 $\pm$ 1.1		1.2 $\pm$ 0.8		0.002*

DISCUSSION

Scabies in atopic children represents a distinct clinical subtype characterized by heightened immune dysregulation and more severe disease manifestations [14]. The demographic distribution between groups was similar, with no significant differences across age categories (0–1 years: 10.00% in both groups; 1–4 years: 40.00% vs 40.00%; 5–14 years: 50.00% vs 50.00%; all $p=1.00$) and mean age (6.7 ± 3.1 vs 7.0 ± 3.4 years, $p=0.58$). Gender and residence also showed no meaningful differences (male: 58.33% vs 55%; urban residence: 36.67% vs 38.33%), suggesting that disease differences observed later are not explained by demographic variation, consistent with findings from Romani et al. who reported that demographic factors rarely alter scabies severity among children [15]. Immunologically, the atopic group showed a markedly elevated total serum IgE (620 ± 200 vs 280 ± 150 IU/mL, $p<0.001$) and eosinophil count (0.88 ± 0.30 vs $0.56 \pm 0.28 \times 10^9/L$, $p<0.001$). These results align with Arlian et al., who demonstrated exaggerated Th2-mediated hypersensitivity responses to *Sarcoptes scabiei* in atopic individuals, resulting in raised IgE and eosinophils [16]. A family history of atopy was significantly higher among atopic children (33.33% vs 16.67%, $p=0.03$), supporting the genetic susceptibility described by Ravn et al. [17]. These baseline findings confirm that the atopic skin environment itself predisposes children to more intense inflammatory reactions during scabies infestation. Atopic children exhibited substantially more severe disease across all clinical measures. They had longer symptom duration (18.5 ± 9.4 vs 12.2 ± 7.1 days, $p=0.001$), greater body surface involvement ($22.4 \pm 11.0\%$ vs $14.8 \pm 8.5\%$, $p<0.001$), and significantly higher lesion numbers (45 ± 18 vs 28 ± 15 , $p<0.001$). Walton et al. previously showed that atopic skin exhibits amplified inflammatory responses to mite antigens, causing higher lesion density and widespread involvement [18]. Pruritus was much more intense among atopic children (8.2 ± 1.2 vs 6.5 ± 1.5 , $p<0.001$), with nocturnal itching in 83.33% vs 58.33% and

sleep disturbance in 75.00% vs 36.67% (both $p<0.001$). These results mirror observations by Sugiyama et al., who found that pre-existing atopic dermatitis markedly worsens nocturnal itching and disrupts sleep [19]. Complications were significantly higher in atopic children: eczematization occurred in 35.00% vs 13.33% ($p=0.002$), and secondary bacterial infection in 45.00% vs 16.67% ($p<0.001$). This is consistent with previous studies, where atopic skin was strongly associated with superinfection risk. The higher rate of moderate-to-severe inflammation (61.67% vs 25.00%, $p<0.001$) further highlights the enhanced immunopathology in atopic skin [20]. Although all children received permethrin-based therapy, atopic children showed poorer treatment responses. Complete response occurred in only 50.00% vs 70.00% ($p=0.003$), and treatment failure was more frequent (16.67% vs 10.00%). Additionally, atopic children required significantly more treatment cycles (1.8 ± 0.9 vs 1.2 ± 0.5 , $p<0.001$) and had slower time to clinical improvement (9.5 ± 4.2 vs 5.8 ± 3.1 days, $p<0.001$). These findings agree with Meyersburg et al., who documented reduced permethrin penetration and delayed healing in eczematous or lichenified skin [21]. Treatment intolerance was (13.33% vs 3.33%, $p=0.01$), consistent with Dressler et al., showing no serious adverse event and irritant dermatitis in atopic children treated with permethrin [22]. Need for systemic therapy was significantly greater in the atopic group (21.67% vs 8.33%, $p=0.01$), reflecting more severe inflammation and higher rates of infection [23]. Atopic children demonstrated markedly poorer prognosis. Secondary infection during follow-up occurred in 38.33% vs 13.33% ($p<0.001$). Although relapse rates were higher (28.33% vs 16.67%), the difference was not statistically significant ($p=0.09$). Time to full cure was significantly prolonged among atopic children (24.3 ± 10.5 vs 17.6 ± 8.4 days, $p<0.001$). More atopic children experienced prolonged disease >4 weeks (31.67% vs 15.00%, $p=0.02$), consistent with Karthikeyan’s Indian pediatric cohort

where atopy predicted delayed resolution [24]. Hospitalization was required only in atopic children (5.00% vs 0%), suggesting more severe systemic or infectious complications. Furthermore, atopic children had worse sleep disturbance scores (6.8 ± 2.1 vs 4.1 ± 2.3 , $p<0.001$) and significantly poorer quality of life (children DLQI: 10.2 ± 3.5 vs 6.5 ± 2.8 , $p<0.001$), mirroring findings from dermatology QoL studies in eczema-affected populations [25]. Household factors strongly contributed to disease burden. Atopic children had higher exposure to shared bedding/clothing (55.00% vs 36.67%, $p=0.01$), more previous scabies exposure in the family (45.00% vs 30%, $p=0.04$), poorer hygiene (53.33% vs 33.33%, $p=0.007$), and more infected family members (1.9 ± 1.1 vs 1.2 ± 0.8 , $p=0.002$). These findings align with studies from Fiji (Marks et al.) and Ethiopia (Yimer et al.), which showed that overcrowding, shared sleeping surfaces, and poor hygiene create a high-transmission environment, particularly harmful to atopic children with impaired skin barrier function [26,27].

LIMITATIONS

This study has several limitations. First, it was conducted at a single center, which may limit the generalizability of findings to other regions of Bangladesh. Second, the relatively small sample size, particularly in the non-atopic group, may have reduced statistical power for some analyses. Third, follow-up was limited to 4–8 weeks, potentially underestimating long-term recurrence or complications. Finally, reliance on clinical diagnosis without routine dermoscopy or skin scraping for mite confirmation could have introduced diagnostic variability.

CONCLUSION

Atopic children with scabies in Bangladesh exhibit a significantly poorer prognosis compared to non-atopic peers, characterized by more severe clinical manifestations, prolonged disease duration, increased pruritus and sleep disturbances, higher rates of eczematization, secondary bacterial infections, and greater quality-of-

life impairment. First-line therapy with permethrin was less effective in this group, necessitating more treatment cycles, systemic therapy, and longer time to clinical improvement. Environmental and household factors, including shared bedding, prior family exposure, and poor hygiene, further contributed to disease severity. Early recognition of atopy in scabies patients is essential to implement aggressive management strategies and reduce morbidity, recurrence, and prolonged disease burden.

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CONFLICT OF INTEREST

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ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

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