ORIGINAL ARTICLE

Skin Colonization of Staphylococcus Aureus in Atopic Dermatitis Patients: A Case-Control Study at SMAMCH

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Abstract:

Background: Atopic dermatitis is a common chronic, relapsing inflammatory skin condition characterized by pruritus and erythematous patches with typical morphological features and distribution. Staphylococcus aureus is a gram-positive, round-shaped bacterium, with the ability to colonize the skin of patients with atopic dermatitis. A correlation between the severity of the eczema and colonization with S. aureus has been demonstrated. This study aims to evaluate the prevalence of staphylococcus aureus colonization on eczematous and non-eczematous skin with atopic dermatitis and the influence on atopic dermatitis severity. **Materials & Methods:** This was a case control study conducted among 30 diagnosed patients of atopic dermatitis as case and another 30 age and sex matched healthy individual as control. Both case and control were collected from the outpatient department of Shaheed Mansur Ali Medical College Hospital (SMAMCH) between the period of January 1st, 2012 and June 30th, 2021. For atopic dermatitis patients, two samples were collected using sterile cotton swab stick. One swab was taken from the eczematous lesion and the other from non-eczematous skin. Skin swabs for bacterial culture were sent to the Department of Pathology, Shaheed Monsur Ali Medical college & Hospital for the isolation and identification of recognized bacterial pathogens. **Results:** Patients were predominantly male, 17 (56.7%) male and 13 (43.3%) female. As for disease severity, 7 (23.3%) had mild disease, 19 (63.3%) had moderate disease and 4 (13.3%) had severe disease. Bacterial colonization by staphylococcus aureus was present among 23 (76.7%) of the patients. Staphylococcus aureus was isolated in 53.33% of the eczematous lesions and in 33.33% of non-eczematous skin of patients with atopic dermatitis. S. aureus was isolated in 3 (42.9%) patients with mild dermatitis, 16 (84.2%) with moderate dermatitis and in 4 (100.0%) with severe dermatitis. S. aureus was not isolated in non-eczematous skin of atopic patient. **Conclusion:** This study confirmed that the skin of patients with atopic dermatitis was more frequently colonized with S. aureus than that of non-atopic. The more severe the dermatitis, the higher the rate of colonization. S. aureus is also more often present in non-eczematous skin of atopic than of non-atopic.

Key words: Atopic dermatitis, Staphylococcus aureus

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Introduction

Atopic dermatitis (AD) is a multifactorial complex disease that causes skin barrier dysfunction with different etiologies and prognosis characterized by dryness, erythema, and extreme pruritus.[1-4] While some cases resolve spontaneously with time, others persist until adolescence and even develop into respiratory allergies such as asthma or allergic rhinitis.[5] Atopic dermatitis is a skin condition. Such bacterial infection is more frequently observed in patients with atopic dermatitis than in non-atopic individuals.

These infections may have a role in the exacerbation of atopic dermatitis.[6] Staphylococcus aureus has been found to be the predominant skin organism in atopic individuals.[7] The severity of AD may be attributed to many environmental factors including exposure to inhalant and food allergens, air pollution, meteorological factors, and the microbiota of the skin.[8] It is well known that S. aureus colonizes skin in 60%–100% of AD patients as compared to 5%–30% of healthy controls.[9-10]

In presenting study, we have investigated the prevalence and role of S. aureus skin colonization in patients with atopic dermatitis, and the relation between S. aureus skin colonization and clinical disease severity.

Materials & Methods

Patients who presented with atopic dermatitis at the outpatient of Shaheed Monsur Ali Medical College & Hospital, Dhaka, between January 1st, 2021 and June 30th, 2021 were included in the study. Thirty patients without atopic dermatitis, age and sex matched, were recruited at the same time to act as controls. All patients had received no oral or topical antibiotic therapy for 1 week prior to the study. The following definitions were used. Atopic dermatitis was defined as a skin condition with three or more of the following criteria: (i) Pruritus; (ii) Typical morphology and distribution of flexural lichenification in adults, or facial and extensor involvement in infants and children; (iii) Chronic or chronically relapsing dermatitis; (iv) Personal or family history of atopy (bronchial asthma, atopic dermatitis, and allergic rhinitis). Controls included healthy individual without atopic dermatitis. The severity of dermatitis was classified as (i) 'Mild' when there was less than 25% total body skin involvement, (ii) 'Moderate' when there was 25-50% involvement, and (iii) 'Severe' when there was more than 50% involvement. For atopic dermatitis patients, two swabs using sterile cottonfield swab sticks rolled over the skin surface twice were taken for culture and sensitivity studies; one swab was taken from the eczematous lesion and the other from noneczematous (normal looking) skin. One swab was taken from the forearm of normal looking skin of control patients. Skin swabs for bacterial culture were sent to the Department of Pathology Shaheed Monsur Ali medical college & Hospital for processing. Standard techniques were used for the isolation and identification of recognized bacterial pathogens.

The culture results of each subject were collared, and the prevalence of positive cultures was compared, according to age, sex, race, and severity of dermatitis, with the controls. All data were compiled and processed with the help of statistician and were analyzed using windows-based computer software with Statistical Packages for Social Sciences (SPSS-25) (SPSS Inc, Chicago, IL, USA). Quantitative data were expressed as mean & standard deviation. Categorical data were expressed as frequency and percentage. Association between categorical variables were seen by chi-square test. Comparison of continuous variables were done by Anova. For all statistical test, p-values less than 0.05 was considered significant.

Results

Thirty patients with atopic dermatitis were included in the study. Table 1 shows the demographic data of the patients. Their mean age was 12.7 ± 4.7 years. There were 17 males (56.7%) and 13 females (43.3%), with a 1.3: 1 male to female ratio. Thirty age- and sex-matched controls with a mean age of 13.7 ± 5.2 years were recruited.

Table 1: Age group & sex wise distribution of patients with atopic dermatitis (n = 30)

Age Group	Sex		
	Male	Female	Total
< 10 years	5 (29.4%)	6 (46.2%)	11 (36.7%)
11 – 19 years	5 (29.4%)	4 (30.8%)	9 (30.0%)
> 19 years	7 (41.2%)	3 (23.1%)	10 (33.3%)
Total	17 (56.7%)	13 (43.3%)	30 (100%)
Mean Age (in years)			12.7 ± 4.7

Data presented as n (%) or mean ± SD.

As for disease severity, 7 (23.3%) had mild disease, 19 (63.3%) had moderate disease and 4 (13.3%) had severe disease (Figure 1)

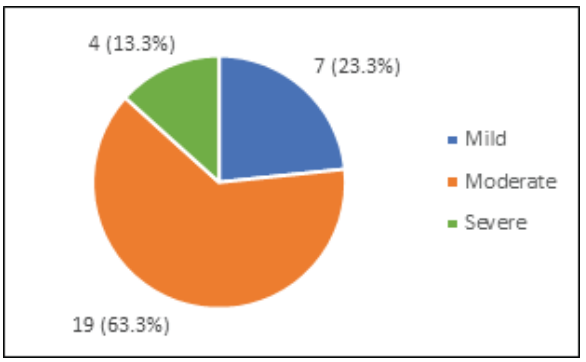

Figure 1: Distribution of study population according to disease severity

Table 2 shows the S. aureus skin colonization rate according to the severity of dermatitis and age group. Of the 30 patients with atopic dermatitis, 7 (23.3%) had mild disease, 19 (63.3%) had moderate disease and 4 (13.3%) had severe disease. These differences were statistically significant ($p < 0.05$).

Table 2: Distribution of atopic dermatitis patients according to bacterial colonization with disease severity (n = 30)

Severity of disease	Bacterial colonization	P
Mild disease (n = 7)	3 (42.9%)	
Moderate disease (n = 19)	16 (84.2%)	< 0.05
Severe disease (n = 4)	4 (100.0%)	
Total (n = 30)	23 (76.7%)	

Data presented as n (%)
* ANOVA was done. P values < 0.05 was considered statistically significant.

Study population were analyzed for bacterial colonization and 33.33% of non-eczematous skin of patients with atopic dermatitis had positive cultures for S. aureus and patient in the control group had a negative culture (Table 3). 53.33% positive cultures for S. aureus were obtained from the skin of patients with atopic dermatitis and negative cultures were obtained from the skin of the control group. Statistically significant ($p < 0.05$) relation was observed.

Table 3: Distribution of study population according to bacterial colonization on eczematous & non eczematous skin

Age group	Eczematous skin	Non eczematous Skin	Control	p
< 10 years	9/13	4/13	0	
11 – 19 years	7/10	5/10	0	< 0.05
> 19 years	3/7	1/7	0	
Total	16/30	10/30	0	
	53.33%	33.33%	0%	

Data presented as n (%)
* Chi-square test was done. P values < 0.05 was considered statistically significant.

Study population were analyzed for bacterial colonization and 33.33% of non-eczematous skin of patients with atopic dermatitis had positive cultures for S. aureus and patient in the control group had a negative culture (Table 3). 53.33% positive cultures for S. aureus were obtained from the skin of patients with atopic dermatitis and negative cultures were obtained from the skin of the control group. Statistically significant ($p < 0.05$) relation was observed.

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11 – 19 years	7/10	5/10	0	< 0.05
> 19 years	3/7	1/7	0	
Total	16/30	10/30	0	
	53.33%	33.33%	0%	

Data presented as n (%)

* Chi-square test was done. P values < 0.05 was considered statistically significant.

Discussion

Atopic dermatitis is a genetically determined skin disorder profoundly influenced by external/environmental factors.[11] The pathogenesis of atopic dermatitis is still unknown. Atopic dermatitis is frequently associated with elevated immunoglobulin E (IgE) levels. The disease usually arises during early infancy, childhood, or adolescence. Diagnosis is based on a combination of clinical and historic parameters, such as morphologic skin lesions, chronic relapsing course, pruritus (which is a hallmark), and a personal and/ or family history of atopy. Due to a decrease in cell-mediated immunity and malfunctioning chemotaxis, bacterial or viral infections are common in atopic dermatitis.[13] The density of S. aureus skin colonization is increased in atopic dermatitis patients, which may account for the frequent staphylococcal skin infections.[14] Leyden et al. isolated S. aureus from chronic lichenified plaques in 91% of 50 atopic dermatitis patients and from exudative lesions in 100% of 20 further atopic dermatitis patients. Aly et al. isolated S. aureus in 93% of eczematous skin, 76% of non-eczematous skin and in 79% of the anterior nares of patients with atopic dermatitis, compared with an isolation rate of less than 5% from normal individuals.[7] C L Goe et al. should thirty-three patients with atopic dermatitis were seen at the outpatient clinic during the study period. Staphylococcus aureus was isolated in 69.7% of the eczematous lesions and in 42.4% of non-eczematous skin of patients with atopic dermatitis. S. aureus was isolated in 53% of patients with mild dermatitis, and in 100% with moderate and severe dermatitis.[15] In our study, S. aureus was isolated in 53.33% of eczematous skin and 33.33% of non-eczematous skin of patients with atopic dermatitis and organism not isolated in controls. This difference was statistically significant. Our findings support previous results that S. aureus colonization is increased on eczematous and non-eczematous skin of patients with atopic dermatitis compared with normal individuals. The close association between the severity of dermatitis and the skin colonization of S. aureus was confirmed in our study. No difference was noted in the skin colonization rates of S. aureus indifferent age groups. The role of staphylococcal enterotoxins (superantigens) in skin inflammation is well established.[9,15] These superantigens are believed to play a role in eczema in atopic dermatitis. Elimination of S. aureus may help to eliminate these superantigens and to alleviate the severity of atopic dermatitis. The high prevalence of skin colonization of S. aureus in atopic dermatitis patients, and the higher rates in patients with severe dermatitis, may support the use of antimicrobials in the treatment of patients with severe dermatitis and of topical and/or systemic antimicrobials during the initial therapy of all atopic dermatitis to eradicate all S. aureus infection for the control of dermatitis.

Conclusion

This study confirmed that the skin of patients with atopic dermatitis was more frequently colonized with S. aureus than that of non-atopic. The more severe the dermatitis, the higher the rate of colonization. S. aureus is also more often present in non-eczematous skin of atopic than of non-atopic.

References

1. Hanifin JM. Pathophysioiogy of atopic dermatitis. In: •Soter NA, Howard BP, eds. Pathophysioiogy of Dermatologic Diseases. New York: McGraw-Hill, 1991: 169-177.

2. Kim BE, Leung DY. Significance of skin barrier dysfunction in atopic dermatitis. Allergy Asthma Immunol Res. 2018; 10:207–215.

3. Leung DY. Atopic dermatitis: more than a rash. Ann Allergy Asthma Immunol. 2018; 120:555–556.
4. Leung DY. The effect of being African American on atopic dermatitis. Ann Allergy Asthma Immunol. 2019; 122:1.
5. Tham EH, Leung DY. Mechanisms by which atopic dermatitis predisposes to food allergy and the atopic march. Allergy Asthma Immunol Res. 2019; 11:4–15.
6. Hauser C, Wulthrich B, Matter L, et al. Staphylococcus aureus skin colonization in atopic dermatitis patients. Dermatologica 1985; 170: 35-39.
7. Leyden JJ, Marples RR, Kligman AM. Staphylococcus aureus in the lesion of atopic dermatitis. Br J Dermatol 1974; 90: 525-530.
8. Williams RE, Gibson AG, Aitchison TC, et al. Assessment of a contact plate sampling technique and subsequent qualitative bacterial studies in atopic dermatitis. Br J Dermatol 1990; 123: 493-501.
9. Marrack P, Kappler J. The staphylococcal enterotoxins and their relatives. Science 1990; 248: 705-711. Leung DY, Gately M, Tramble A, et al. Bacterial superantigen induced T cell expression of the skin selective homing receptor, the cutaneous lymphocytes-associated antigen, via stimulation of IL2 production. / Exp Med 1995; 181:747-753.
10. Breuer K, HÄussler S, Kapp A, Werfel T. Staphylococcus aureus: colonizing features and influence of an antibacterial treatment in adults with atopic dermatitis. Br J Dermatol. 2002; 147:55–61.
11. White MI, Noble WC. Consequences of colonization and infection by Staphylococcus aureus in atopic dermatitis. Clin Exp Dermatol 1986; 11: 34-40.
12. Hanifin JM, Rajka G. Diagnostic features of atopic dermatitis. Acta Dermatovenereol (Stockholm) Suppl 1980; 92: 44-47.
13. Vieluf D, Ruzicka TH. Complication and diseases ^, j associated with atopic eczema. In: Ruzicka TH, ed. Handbook of Atopic Eczema. Berlin: Springer, 1991: 54.
14. Aly R, Maibach HI, Shinefield HR. Microbial flora of atopic dermatitis. Arch Dermatol 1977; 113: 780-782.
15. C L Goh, J S Wong, Y C Giam. Skin colonization of Staphylococcus aureus in atopic dermatitis patients seen at the National Skin Centre, Singapore.