



BACTERIAL SKIN INFECTION AND ATOPIC DERMATITIS ACCORDING TO SCORING ATOPIC DERMATITIS INDEX

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ABSTRACT

Introduction. Children with atopic dermatitis (AD) are more susceptible to skin infections, and vice versa. Skin infection can trigger and aggravate atopic dermatitis. This study was intended to determine the association between skin infection in atopic dermatitis with SCORing Atopic Dermatitis (SCORAD) index and to identify microorganisms which often infect skin in atopic dermatitis.

Methods. This study was a cross-sectional study of 80 children with atopic dermatitis who visited Helvetia and Padang Bulan Community Health Center, Medan from April to September 2016 for medical treatment. Index of SCORAD was used to evaluate the severity of AD, and skin lesion was smeared with cotton swabs to take sterile culture. Data were analysed using Chi square and Mann Whitney test with 95% CI. P value <0.05 was considered statistically significant.

Results. Total subjects in this study were 80 children, consisting of 47 girls and 33 boys. There were 50 subjects with atopic dermatitis suffered skin infection. The most common microorganism isolated from skin infection (mild, moderate, or severe) in atopic dermatitis was *Staphylococcus aureus*. The result showed there was a significant association between the classification SCORAD and bacterial infection in atopic dermatitis ($p = 0.011$) with OR (95% CI) = 2.06 (1.17-3.63).

Conclusion. There is a relationship between bacterial infection in atopic dermatitis with SCORAD index, where atopic dermatitis with bacterial infection had higher SCORAD index. In this study, *Staphylococcus aureus* is the most common cause of skin infections in atopic dermatitis.

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INTRODUCTION

Atopic Dermatitis (AD) or eczema is a chronic, relapsing inflammatory skin condition characterized by itchy, red rash, vary in appearance from edema, vesicles, exudate in the acute stage, and skin thickening (lichenification) on the chronic stage that often associated with other atopic disease such as allergic rhinitis and asthma, it causes pruritus and usually begins in early childhood.^{1,2} Atopic dermatitis can be the first step of atopic march that will continue to food allergy in infants and children, allergic rhinitis at school age, and asthma in children, teenagers and adults.^{3,4}

Atopic dermatitis is often recurrent even with adequate therapy, showing the need to look for possible precipitating factors for the prevention of AD recurrence. Infections of the skin can be caused by bacteria, viruses, fungi, or mix. Children with AD has a higher level of bacteria colony on the skin than the normal children and there after more vulnerable to skin infections, and vice versa. Skin infections can trigger and aggravate the atopic dermatitis.⁵ Severity of atopic dermatitis assessed by SCORing Atopic Dermatitis (SCORAD) index.

This study was intended to examine the relationship between bacterial skin infection and severity of AD by SCORAD index and to know the bacteria that most commonly cause skin infection of AD.

METHODS

This study was a cross-sectional study of 80 children younger than 18 years old with AD (confirmed by fulfilling at least three major and three minor of Hanifin and Rajka criteria) who visited *Puskemas* (Community Health Center) of Subdistrict Helvetia and Padang Bulan, Medan, Indonesia during April until September 2016. This study was conducted under the approval of the Research Ethics Committee of the Faculty of Medicine University of Sumatera Utara / Haji Adam Malik General Hospital, Medan, Indonesia. Children with malnutrition, chronic disease, taking long-term corticosteroids and oral/topical antibiotics over the last 10-14 days were excluded. After their parent gave informed consent, the subjects included in the study. Diagnosis of AD was made by the researchers and doctors who served in the health service. Weight, height, and nutritional status of these children were examined. Swab at AD skin lesion area were taken to know whether there was a bacterial infection of the skin, carried out directly by the researchers. Culture examinations were done using sterile cotton swab on the skin surface of AD and immediately transported to the laboratory of Faculty of Medicine, University of Sumatera Utara,

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Medan, Indonesia to be cultured in blood agar. The severity of AD classified into mild (SCORAD <25), moderate (SCORAD 25-50), and severe dermatitis (SCORAD > 50).

Data were analyzed using univariate and bivariate analysis. Bivariate analysis was carried out using was performed to assess the relationship between bacterial skin infection and severity of AD. The statistical test used in this study were Chi-square test, Unpairedt-test, and Mann-Whitney test. The level of significance is p<0.05 and 95% confidence intervals.

RESULTS

The total subjects in this study were 80 children, consist of 47 girls (58.75%) and 33 boys (41.25%). Among 47girls, there were 28 girls (59.6%) had skin infection in atopic dermatitis. Among male subjects, 22 boys (66.7%) had skin infection in atopic dermatitis. A total of 51 subjects (63.75%) had family history of atopy and 50 subjects (62.5%) were exclusive breast feeding. There was no association between gender, family history of atopy, history of exclusive breast feeding, and history of delivery with skin infection in atopic dermatitis (p> 0.05). There were no significant differences inage, weight, and height between infected and uninfected atopic dermatitis (p> 0.05) (Table 1).

Table 1 Characteristic of Subjects

Characteristics	Infected (n = 50)	Uninfected (n = 30)	P
Gender, n(%)			
Boy	22 (66.7%)	11 (33.3%)	0.519 ^a
Girl	28 (59.6%)	19 (40.4%)	
Age (yr), median (min – max)	4.3 (0.08-14)	5.8 (0.16-14)	0.081 ^b
Agegroup (yr), n (%)			
Less than 2 years	14 (77.8%)	4 (22.2%)	0.128 ^a
2 – 18 years	36 (58.1%)	26 (41.9%)	
Weight (kg), median (min-max)	14.5 (3.2-49)	16.8 (3-38)	0.152 ^b
Height (cm), mean (SD)	100.9(25.51)	111.9(24.81)	0.063 ^c
Family history of atopy, n(%)			
No history of atopy	21 (53.8%)	18 (46.2%)	0.119 ^a
History of atopy	29 (70.7%)	22 (29.3%)	
One parent	11 (64.7%)	6 (35.3%)	
Both parents	6 (66.7%)	3 (60.0%)	
Siblings	12 (80.0%)	3 (20.0%)	
History of breastfeeding, n(%)			
No exclusive breastfeeding	16 (53.3%)	14 (46.7%)	0.190 ^a
Exclusive breastfeeding	34 (68%)	16 (32%)	
Process of delivery, n(%)			
Vaginal	43 (67.2%)	21 (32.8%)	0.083 ^a
Sectiicaesarea	7 (43.8%)	9 (56.3%)	

^a Chi Square, ^b Mann-Whitney test, ^cunpaired T-test

Skin lesion cultures showed that *Staphylococcusaureus* was the most common bacteria found in atopic dermatitis, followed by *Escherichia coli*. Twenty four out of 50 subjects were infected by *Staphylococcus aureus*. *Escherichia coli* was infected 6 out of 50 subjects. *Staphylococcus aureus* was also most commonly found in every degree of atopic dermatitis, from mild (50%), moderate (45.16%), and severe atopic dermatitis (50%) (Table 2).

There was a significant association between classification of SCORAD with skin infection in atopic dermatitis (p = 0.011), patients with skin infection had 2.06 times higher risk having

severe atopic dermatitis (Table 3). There was significant difference in SCORAD index between infected and uninfected atopic dermatitis (p = 0.008) (Table 4).

Table 2 Microorganism of skin infections in atopic dermatitis according to classification of SCORAD

No	Classification of SCORAD	Microorganism	Total		
1	Mild (n = 14)	<i>Staphylococcus aureus</i>	7		
		<i>Escherichia coli</i>	2		
		<i>Staphylococcus saprophyticus</i>	1		
		<i>Klebsiella pneumonia</i>	1		
		<i>Staphylococcus hominis</i>	1		
		<i>Klebsiellaozaenae</i>	1		
		<i>Kocuriarosea</i>	1		
		2	Moderate (n = 30)	<i>Staphylococcus aureus</i>	14
				<i>Escherichia coli</i>	4
				<i>Pseudomonas sp</i>	4
<i>Klebsiella pneumonia</i>	2				
<i>Enterobacter cloacae</i>	2				
<i>Acinetobacter hwofii</i>	2				
<i>Citrobacterkoseri</i>	1				
<i>Burkholderiacepacia</i>	1				
3	Severe (n = 6)			<i>Staphylococcus aureus</i>	3
		<i>Klebsiellaoxytoca</i>	1		
		<i>Serratiaficaria</i>	1		
		<i>Proteus penneri</i>	1		

Table 3 The relationship between the classification of SCORAD with skin infections in atopic dermatitis

Infection Status	Classification of SCORAD		p-value	OR (95% CI)
	Moderate + Severe	Mild		
Infected	36 (72%)	14 (28%)	0.011*	2.06 (1.17 – 3.63)
Uninfected	13 (43.3%)	17 (56.7%)		

*p<0.05

Table 4 Comparison of SCORAD index between infectedanduninfected atopic dermatitis

Infection Status	SCORAD Index	p-value
Infected	30.9 (8.8 – 64.2)	0.008*
Uninfected	23.55 (11.1-64.4)	

*p<0.05

DISCUSSION

This study showed that prevalence of AD in boys were higher than girls. The previous studies reported inconsistent results. Yu *et al* reported that percentage of AD in boys was higher than girls.⁶ Pyun reported that percentage of AD in boys and girls were varies based on age. Boyswere more often to have atopic dermatitis in infant; but during adolescence, it was found that girls were more often.⁷

There was no significant difference in positive culture results between boys (66.7%) and girls (59.6%). This result was consistent with previous studies. Farajzadeh *et al* reported that there was no significant positive culture between boys and girls (77.9% vs 66.7%). The same result was reported by Hill *et al* and Alenizi *et al*.⁸⁻¹⁰

The prevalence of atopic dermatitis was high, especially in children under 6 years old and will decrease with age.¹¹ Zhao *et al* (2010) reported that peak incidence of AD was between

0-4 years. In this study, there was no association between the age and infected or uninfected a topic dermatitis ($p > 0.05$).¹² Farajzadehal so reported the same result that there was no association between age and bacterial skin infection in atopic dermatitis.⁸

There was no association between family history of atopy and skin infection in atopic dermatitis. History of atopy in the family has been known as one of the main factors of atopic dermatitis in children. But there has been no reports suggesting an association between family history of atopy and bacterial skin infection in atopic dermatitis.^{7,13}

The relationship between the incidence of atopic dermatitis and breast feeding was still controversy. Some studies showed that breast feeding was a protective factor against atopic dermatitis. But there were other studies showed that it can actually trigger the development of atopic dermatitis. There were several arguments that breast feeding might increase the risk of atopic dermatitis. Early infection can trigger maturation of immune system and prevent allergy, including atopic dermatitis. Because breast feeding will decrease children exposure to allergens which commonly found in solid foods or formula, their immune system cannot adequately to protect their body from antigen, which can cause atopic dermatitis.¹⁴ In this study, there was no statistically significant association between exclusive breast feeding and bacterial skin infection in atopic dermatitis. It was supported by Londero et al (2011) that there was no significant association between breast feeding and bacterial skin infection in atopic dermatitis. Twenty nine percents of 31 patients who exclusively breast fed had mucocutaneous infection, while 33.3% of 48 patients who are not exclusively breast fed experienced mucocutaneous infection ($p = 0.878$).¹⁵

The composition of intestinal flora in children can affect susceptibility to allergic diseases including atopic dermatitis. Beneficial intestinal flora were derived from maternal vagina and transferred during vaginal delivery. Renz-Polsteretal conducted research to compare the risk of atopic dermatitis in children who were born pervaginam and section caesarea. There was no significant relationship between process of delivery and atopic dermatitis.¹⁶ Along with Park et al and Bageretal, the risk of atopic dermatitis did not increase by the process of delivery (vaginal delivery or sectio caesarean).^{17,18} This study also found no association between process of delivery and skin infection in atopic dermatitis ($p > 0.05$).

This study found that *Staphylococcus aureus* was the most common microorganism that cause skin infection in atopic dermatitis. This result was in accordance with the results from Farajzadeh et al, Goh et al, Hoeger, Perera et al, and Abeck et al, that the most common cause of bacterial skin infection in AD is *Staphylococcus aureus*.^{8,19-22}

Gomes et al (2011) reported on 100 patients with atopic dermatitis, 57% had a skin infection by *Staphylococcus aureus*.²³ The prevalence rate of infection was different between studies. The lowest rate was 42.5% reported by Pezesk et al (2007) and 48.5% by Hon et al (2005), while the highest rate was 86% by Gilani et al (2005) and 100% by Guzik et al (2005).²⁴⁻²⁷

Alenizi et al found the incidence of infection in mild atopic dermatitis was 51.4% (18 of 35), moderate was 77.8% (14 of 18), and severe was 100% (7 of 7). Gomes et al (2011) reported prevalence of skin infection was 46% in mild atopic dermatitis, 73% in moderate atopic dermatitis, and 100% in severe atopic dermatitis.²³ Pascolin et al (2011) reported the prevalence of skin infection in atopic dermatitis was 15% in mild, 52% in moderate, and 77.5% in severe atopic dermatitis.²⁸

Farajzadeh et al (2008) analyzed swabs from 50 patients with atopic dermatitis in Iran and found positive results in 74% patients. Sixty six percent of skin infections were caused by *Staphylococcus aureus*.⁸ Goh found that *Staphylococcus aureus* isolated in 69.7% patients with eczematous lesions and 42.4% in non-eczematous. Goh reported *Staphylococcus aureus* isolated in 53% of patients with mild atopic dermatitis, and 100% in moderate and severe atopic dermatitis.¹⁹

Various microorganisms were reported to be the etiology of atopic dermatitis by Farajzadeh et al, such as *Staphylococcus aureus* beta-hemolytic *Streptococcus* group A and B, *Streptococcus viridans*, and *Enterobacter*, while Gong et al in China reported the colonization of *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Streptococcus haemolytic*, *Staphylococcus lugdunensis*, *S. capitis*, *E. coli*, *Micrococcus tetragenus*, *Enterobacter cloacae*, *Proteus* in patients with atopic dermatitis.²⁹ *Staphylococcus aureus* can colonize on the skin in healthy patients and become pathogenic in conditions of decreased immunity or damage the skin barrier.³⁰ Patients with atopic dermatitis are more susceptible to staphylococcal skin infections. The study reported between 80-100% of patients with atopic dermatitis showed skin colonization by *Staphylococcus aureus*, while colonization only seen in 5-30% in individuals without atopic dermatitis.³¹ Relationship between degree of atopic dermatitis and infections have been reported previously, that the presence of infection is an important mechanism to trigger or aggravate the disease atopic dermatitis.^{19,27,29}

CONCLUSIONS

This study had shown relationship between skin infections in atopic dermatitis with the SCORAD index, in which patients with skin infections would be 2.06 times more at risk to have a higher SCORAD value. This study has also found that most of the bacteria to cause skin infections in atopic dermatitis, whether mild, moderate, or severe is *Staphylococcus aureus*.

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References

1. Ring I, Alomar A, Bieber T. Guidelines for treatment of atopic eczema (atopic dermatitis)-part I. *J Europ Acad Dermatol Venereol*. 2012;26 :1045-60.
2. Watson W, Kapur S. Atopic dermatitis. *Allergy, Asthma & Clinical Immunology* 2011; 7(Suppl 1):S4.

3. Spergel JM: From atopic dermatitis to asthma: the atopic march. *Ann Allergy Asthma Immunol.* 2010;105:99–106.
4. Helmich LH, Linnerberg A, Thomsen SF, Glumer C. Association between parental socioeconomic position and prevalence of asthma, atopic eczema, and hay fever in children. *Scandinavian J Public Health.* 2014;42:120-7.
5. Evina B. Clinical manifestations and diagnostic criteria of atopic dermatitis. *J Majority.* 2015; 4(4):23-30.
6. Yu JS, Lee CJ, Lee HS, Kim J, H Y, Ahn K, et al. Prevalence of Atopic Dermatitis in Korea: Analysis by Using National Statistics. *J Korean Med Sci.* 2012;27:681-5.
7. Pyun BY. Natural history and risk factors of atopic dermatitis in children. *Allergy Asthma Immunol Res.* 2015;7(2):101-5.
8. Farajzadeh S, Rahnama ZZ, Kamyabi Z, Ghavidel B. Bacterial colonization and antibiotic resistance in children with atopic dermatitis. *Derm Online J.* 2008;14:21.
9. Alenizi DA. Prevalence of Staphylococcus aureus and antibiotic resistance in children with atopic dermatitis in Arar, Saudi Arabia. *J Dermatol Journal of Dermatology & Dermatologic Surgery.* 2014;18:22–6.
10. Hill SE, Yung A, Bademaker M. Prevalence of Staphylococcus aureus and antibiotic resistance in children with atopic dermatitis. *Australasian J Dermatol.* 2011;52:27-31.
11. Hong S, Son DK, Lim WR, Kim SH, Kim H, Yum HY, et al. The prevalence of atopic dermatitis, asthma, and allergic rhinitis and the comorbidity of allergic diseases in children. *Environment Health Toxicol.* 2012;27:e2012006.
12. Zhao J, Bai J, Shen K, Xiang L, Huang S, Chen A, et al. Self-reported prevalence of childhood allergic disease in three cities of China: a multicenter study. *BMC Public Health.* 2010;10:551
13. Baker BS. The role of microorganisms in atopic dermatitis. *ClinExpImmunol.* 2006;144:1-9.
14. Lien TY, Goldman RD. Breastfeeding and maternal diet in atopic dermatitis. *Can Fam Physician.* 2011;57(12):1403-5.
15. Londero RM, Giugliani ER, Bonamigo RR, Bauer VS, Cecconi MCP, Zubaran GM. Breastfeeding and mucosal and cutaneous colonization by Staphylococcus aureus in atopic children. *An Bras Dermatol.* 2011;86:435-9.
16. Renz-Polster H, David MR, Buist AS, Vollmer WM, O'Connor EA, Frazier EA, et al. Caesarean section delivery and the risk of allergic disorders in childhood. *Clin Exp Allergy.* 2005;35(11):1466-72.
17. Bager P, Wohlfahrt J, Westergaard T. Caesarean delivery and risk of atopy and allergic disease: meta-analyses. *Clin Exp Allergy* 2008;38:634-42.
18. Park YH, Kim KW, Choi BS, Jee HM, Sohn MH, Kim KE. Relationship between mode of delivery in childbirth and prevalence of allergic diseases in Korean children. *Allergy Asthma Immunol.* 2010;2(1):28-33.
19. Goh CL, Wong JS, Giam YC. Skin colonization of Staphylococcus aureus in atopic dermatitis patients seen at the National Skin Care, Singapore. *Int J Dermatol.* 1997;36:653-7.
20. Hoeger PH. Antimicrobial susceptibility of skin-colonizing S. aureus strains in children with atopic dermatitis. *Pediatr Allergy Immunol.* 2004;15:474-7.
21. Perera G, Hay R. A guide to antibiotic resistance in bacterial skin infections. *JEADV* 2005;19: 531-45.
22. Abeck D, Mempel M. Staphylococcus aureus colonization in atopic dermatitis and its therapeutic implications. *British J Dermatol.* 1998;139:13-16.
23. Gomes PL, Malavige GN, Fernando N, Mahendra MH, Kamaladasa SD, Seneviratne JK, et al. Characteristics of Staphylococcus aureus colonization in patients with atopic dermatitis in Sri Lanka. *ClinExpDermatol.* 2011;36(2):195–200.
24. Pezesk-Pour FZ, Miri S, Ghasemi R, Farid R, Ghenaat J. Skin colonization with Staphylococcus aureus in patients with atopic dermatitis. *Internet J Dermatol.* 2007;5(1):e93.
25. Hon KL, Lam MC, Leung TF, Kam WY, Li MC, Ip M, et al. Clinical features associated with nasal Staphylococcus aureus colonisation in Chinese children with moderate-to-severe atopic dermatitis. *Ann Acad Med Singapore.* 2005;34(10):602–5.
26. Gilani SJ, Gonzalez M, Hussain I, Finlay AY, Patel GK. Staphylococcus aureus re-colonization in atopic dermatitis: beyond the skin. *Clin Exp Dermatol.* 2005;30(1):10–3.
27. Guzik TJ, Bzowska M, Kasprowicza, Czerniawska-Mysik G, Wojcik K, Szmyd D, et al. Persistent skin colonization with Staphylococcus aureus in atopic dermatitis: relationship to clinical and immunological parameters. *ClinExp Allergy.* 2005;35 (4):448–55.
28. Pascolin C, Sinagra J, Pecelta S, Bordignon V, De Santis A, Cilli L, et al. Molecular and immunological characterization of Staphylococcus aureus in pediatric atopic dermatitis: implications for prophylaxis clinical management. *Clin Dev Immunol.* 2011;2011:718708.
29. Gong JQ, Lin L, Lin T, Hao F, Zeng FQ, Bi ZG, et al. Skin colonization by Staphylococcus aureus in patients with eczema and atopic dermatitis and relevant combined topical therapy: a double-blind multicentre randomized controlled trial. *Br J Dermatol.* 2006; 155:680–7.
30. Petry V, Bessa GR, Poziomczyk CS, de Oliveira CF, Weber MB, Bonamigo RR, et al. Bacterial skin colonization and infections in patients with atopic dermatitis. *An Bras Dermatol.* 2012;87(5):729-34.
31. Breuer K, Kappa A, Werfel T. Bacterial infections and atopic dermatitis. *Allergy.* 2001;56:1034-41.