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VIEWPOINT ARTICLE

Clinical features and *Staphylococcus aureus* colonization/infection in childhood atopic dermatitis

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Abstract

Introduction: *Staphylococcus aureus* (SA) colonization/infection is important in the pathophysiology of childhood atopic dermatitis (AD). This study evaluated which clinical features may predict presence of SA colonization/infection and reviewed antimicrobial sensitivity of SA in patients with AD. **Methods:** The associations between bacteriologic culture results of skin swabs (taken at the most severely affected area and at the antecubital fossa) and SCORing-Atopic-Dermatitis (SCORAD), skin hydration, transepidermal water loss (TEWL), and quality of life were evaluated. **Results:** Moderate-to-heavy growth of SA was present in 31% of the swabs of the most severe area and in 16% of the flexural (antecubital fossae) areas of 95 AD patients (12.5 ± 4.8 years). Binomial logistic regression showed moderate-to-heavy growth of SA in the severe area were associated with objective SCORAD ($p = 0.004$) and lesion intensity [erythema ($p = 0.022$) and lichenification ($p = 0.035$)] and excoriation ($p = 0.024$) and TEWL ($p = 0.009$) in the antecubital fossa. The relative risk of isolating moderate-to-heavy growth of SA in the most affected area in patients with severe disease (objective SCORAD >40) is 2.73 (1.43–5.21, $p = 0.001$). Any growth of SA in either swab sites was associated with objective SCORAD and lesion intensity ($p = 0.001$ – 0.019). SA had no association with quality of life and other clinical parameters. All specimens of methicillin-sensitive SA were sensitive to cloxacillin. All methicillin-resistant SA (MRSA) (5.7%) was sensitive to co-trimoxazole and fusidic acid. **Conclusions:** Clinical features, especially severity and lesion intensity, are useful in “predicting” moderate-to-heavy SA colonization/infection in AD patients. Cloxacillin has a favorable sensitivity profile for MSSA, and co-trimoxazole and fusidic acid for MRSA. As colonization and infection are ambiguous and potentially overlapping clinical states, we recommend to abandon these terms and propose to describe quantitatively/semi-quantitatively SA isolation as none, mild, scanty, moderate or heavy growth instead in clinical trials.

Keywords

Atopic dermatitis, children, flexures, quality of life, SCORAD, *Staphylococcus aureus*

History

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Introduction

Atopic dermatitis (AD) is a common chronic relapsing disease. It was estimated that about 15% of children suffer from this disease (1–4). Bacterial colonization/infection due to *Staphylococcus aureus* (SA) is considered an important factor in the pathophysiology of AD (5–8). Physician caring for patients with severe disease or with acute exacerbation in ambulatory clinic settings often has to decide if symptomatology is due to SA colonization/infection. Swabs may have to be taken for bacteriology, and an empirical course of antibiotic prescribed pending bacteriology results in these patients (5–8). Treatment targeted to reduce SA colonization/infection may improve quality of life and ameliorate disease severity and intensity (9,10). This study aims to (i) evaluate which clinical features may predict presence of SA colonization/infection and (ii) review antimicrobial sensitivity of SA in patients with AD.

Methods

Swabs taken from a typical flexure (right antecubital fossa) (7) and the worst affected skin areas (defined as acute lesional skin with erythema, papulation, excoriation, oozing, or crusting) of AD children managed at the pediatric dermatology service of a university teaching hospital over a 3-year period were reviewed. Body swabs (COPAN Innovation, Italy) were taken by either one of the two staff at the clinic (5 seconds of rubbing). Patients were excluded from the study if they suffered from other inflammatory dermatitides (such as psoriasis, seborrheic dermatitis, ichthyosis) or had been treated with systemic antibiotics (such as cloxacillin or erythromycin) in the previous 4 weeks. Bacterial cultures of the swabs were carried out by standard laboratory techniques and sensitivity towards common antibiotics examined. Bacterial growth was classified as scanty ($<10^4$ colony-forming units per milliliter), moderate (10^4 to 10^5), or heavy ($>10^5$). We defined moderate-to-heavy growth as significant growth, and scanty or no growth as negative (7). AD severity was routinely assessed by a clinician with the SCORAD index (11,12). Long-term disease severity (Nottingham Eczema Severity Score, NESS) (13,14), quality of life (Children's Dermatology Life Quality Index,

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CDLQI) (15,16) and biophysiologic parameters [skin hydration (SH) and transepidermal water loss (TEWL)] (17) were also recorded. This review was approved by the Clinical Research Ethics Committee of the University. Kruskal–Wallis *H* test and Mann–Whitney *U* test were used to analyze quantitative traits, and chi-square test was used to compare proportions between groups. Binomial logistic regression was performed using IBM SPSS 20.0 to correct for multiple comparisons. All comparisons were two-sided, and *p*-values less than 0.05 were considered to be statistically significant.

Results

Ninety-five AD patients (4 mild, 56 moderate, and 35 severe AD as assessed using objective SCORAD) had body swabs taken between January 2012 and January 2015. The patients had been treated with combinations of emollients, topical corticosteroids, oral antihistamines, or topical antibiotic on an as-needed basis.

Moderate-to-heavy growth of *SA* was present in 31% of the swabs of the most severe area and in 16% of the flexural (antecubital fossae) areas of the 95 AD patients (Tables 1 and 2).

Binomial logistic regression showed moderate-to-heavy growth of *SA* were associated with objective SCORAD ($p=0.004$) and lesion intensity [erythema ($p=0.022$) and lichenification ($p=0.035$)] in the severe area (Table 3); and excoriation ($p=0.024$) and TEWL ($p=0.009$) in the antecubital fossa (Table 4). The relative risk of isolating moderate-to-heavy growth of *SA* in the most affected area in patients with severe disease (objective SCORAD >40) is 2.16 (1.38–3.40, $p=0.001$). A receiver operating characteristic (ROC) curve for objective SCORAD and lesion intensity was constructed (Figure 1; area under curve = 0.76 and 0.78, respectively). Any growth (scanty + moderate + heavy growth) of *SA* in either swab sites was associated with objective SCORAD and intensity (Tables 2 and 4; $p=0.001$ – 0.019). All specimens of methicillin-sensitive *SA* were sensitive to cloxacillin (Table 5). Methicillin-resistant *SA* (MRSA) (5.7%) was sensitive to co-trimoxazole and fucidin.

Discussion

As demonstrated in many studies, *SA* is the most important pathogen associated with severe AD (6–8). Physician caring for

Table 1. Comparison of clinical features between AD patients with no, scanty or moderate-to-heavy growth of *SA* in the most severely affected skin site ($n=95$).

	Group I	Group II	Group III	<i>p</i> Value		
	No growth ($n=27$)	Scanty growth ($n=39$)	Moderate-to-heavy growth ($n=29$)	I versus II versus III	(I + II) versus III	I versus (II + III)
Male (%)	16 (59.3)	28 (71.8)	13 (44.8)	0.080	0.045	0.926
Age (y)	11.5 $\pm$ 5.8	12.5 $\pm$ 4.2	13.4 $\pm$ 4.5	0.504	0.357	0.298
BMI (kg/m^2)	19.4 $\pm$ 4.2	19.5 $\pm$ 4.3	18.0 $\pm$ 3.2	0.339	0.143	0.458
Topical corticosteroid use (days/week)	3.7 $\pm$ 2.8	3.8 $\pm$ 2.7	4.2 $\pm$ 2.9	0.798	0.510	0.694
Topical antibiotic use (days/week)	1.2 $\pm$ 2.4	0.8 $\pm$ 1.8	2.0 $\pm$ 3.0	0.182	0.080	0.835
Topical immunomo- dulant use (days/ week)	0.4 $\pm$ 1.0	1.4 $\pm$ 2.4	1.6 $\pm$ 2.4	0.121	0.312	0.040
Oral antihistamine use (days/week)	2.3 $\pm$ 2.6	2.4 $\pm$ 2.9	2.8 $\pm$ 2.9	0.868	0.609	0.934
Emollient frequency ≥ 3 times/day (%)	18 (66.7)	22 (56.4)	17 (58.6)	0.693	0.856	0.097
SH	37.4 $\pm$ 13.2	31.9 $\pm$ 14.7	29.7 $\pm$ 11.5	0.062	0.195	0.020
TEWL	11.3 $\pm$ 2.2	10.9 $\pm$ 2.4	12.2 $\pm$ 2.1	0.038	0.018	0.931
SCORAD						
Objective SCORAD	26.7 $\pm$ 12.5	34.6 $\pm$ 13.8	46.1 $\pm$ 14.9	<0.0005	<0.0005	<0.0005
Extent, % body sur- face area	34.5 $\pm$ 16.7	41.1 $\pm$ 21.0	52.9 $\pm$ 20.9	0.009	0.005	0.022
Intensity						
Erythema	5.7 $\pm$ 2.8	7.5 $\pm$ 2.8	10.1 $\pm$ 3.1	<0.0005	<0.0005	<0.0005
Edema/papulation	0.8 $\pm$ 0.6	1.0 $\pm$ 0.7	1.6 $\pm$ 0.7	<0.0005	<0.0005	0.009
Oozing/crust	1.3 $\pm$ 0.7	1.6 $\pm$ 0.6	2.0 $\pm$ 0.6	0.001	0.001	0.002
Excoriation	0.4 $\pm$ 0.8	0.8 $\pm$ 0.8	1.5 $\pm$ 0.9	<0.0005	<0.0005	0.001
Lichenification	1.2 $\pm$ 0.6	1.6 $\pm$ 0.5	1.9 $\pm$ 0.6	<0.0005	0.001	<0.0005
Dryness	0.9 $\pm$ 0.7	1.2 $\pm$ 0.6	1.7 $\pm$ 0.7	<0.0005	<0.0005	0.006
Pruritus	1.0 $\pm$ 0.6	1.4 $\pm$ 0.6	1.6 $\pm$ 0.6	0.009	0.035	0.004
Sleep loss	5.6 $\pm$ 1.7	6.1 $\pm$ 1.9	7.2 $\pm$ 2.3	0.010	0.004	0.039
NESS	4.3 $\pm$ 3.2	3.8 $\pm$ 3.2	6.3 $\pm$ 2.9	0.008	0.002	0.470
CDLQI ($n=85$)	11.9 $\pm$ 2.1	12.0 $\pm$ 2.4	12.9 $\pm$ 2.1	0.085	0.028	0.237
	8.7 $\pm$ 4.6 ($n=22$)	8.9 $\pm$ 6.3 ($n=37$)	11.4 $\pm$ 6.9 ($n=26$)	0.257	0.104	0.706

Objective SCORAD: I versus III ($p<0.0005$), II versus III ($p=0.003$).

Affected area: I versus III ($p=0.002$), II versus III ($p=0.045$).

Intensity: I versus II ($p=0.031$), I versus III ($p<0.0005$), II versus III ($p=0.001$).

Erythema: I versus III ($p<0.0005$), II versus III ($p=0.001$).

Edema/papulation: I versus III ($p=0.001$), II versus III ($p=0.047$).

Oozing/crust: I versus III ($p<0.0005$), II versus III ($p=0.008$).

Excoriation: I versus II ($p=0.019$), I versus III ($p<0.0005$).

Lichenification: I versus III ($p<0.0005$), II versus III ($p=0.007$).

Dryness: I versus III ($p=0.003$).

Pruritus: I versus III ($p=0.007$).

Sleep loss: I versus III ($p=0.047$), II versus III ($p=0.005$).

Table 2. Comparison of clinical features between AD patients with no, scanty or moderate-to-heavy growth of SA in the right antecubital fossa (n = 95).

	Group I	Group II	Group III	p Value		
	No growth (n = 41)	Scanty growth (n = 39)	Moderate-to-heavy growth (n = 15)	I versus II versus III	(I + II) versus III	I versus (II + III)
Male (%)	28 (68.3)	20 (51.3)	9 (60.0)	0.300	>0.999	0.151
Age (years)	12.1 ± 4.9	12.7 ± 4.6	12.9 ± 5.2	0.912	0.721	0.724
BMI (kg/m ²)	19.1 ± 3.4	18.3 ± 4.0	20.5 ± 4.9	0.150	0.232	0.330
Topical corticosteroid use (days/week)	4.0 ± 3.0	3.7 ± 2.6	3.9 ± 3.0	0.880	0.975	0.649
Topical antibiotic use (days/week)	1.1 ± 2.3	1.2 ± 2.4	2.0 ± 2.7	0.175	0.062	0.474
Topical immunomod- ulant use (days/week)	0.9 ± 2.0	1.2 ± 2.0	1.9 ± 2.8	0.436	0.354	0.238
Oral antihistamine use (days/week)	2.0 ± 2.9	2.8 ± 2.8	2.7 ± 2.6	0.246	0.647	0.096
Emollient frequency ≥ 3 times/day (%)	24 (58.5)	21 (53.8)	12 (80.0)	0.207	0.036	0.014
SH	37.2 ± 13.4	31.3 ± 13.1	24.6 ± 11.3	0.006	0.014	0.004
TEWL	10.7 ± 2.5	11.5 ± 2.0	12.8 ± 1.8	0.002	0.003	0.002
SCORAD						
Objective SCORAD	26.6 ± 11.7	40.7 ± 12.4	48.6 ± 18.3	<0.0005	0.003	<0.0005
Extent, % body surface area	32.9 ± 17.6	48.1 ± 17.3	56.3 ± 26.3	<0.0005	0.024	<0.0005
Intensity	5.7 ± 2.5	8.9 ± 2.7	10.7 ± 3.8	<0.0005	0.001	<0.0005
Erythema	0.8 ± 0.6	1.3 ± 0.7	1.4 ± 0.8	0.001	0.078	<0.0005
Edema/papulation	1.3 ± 0.7	1.8 ± 0.5	2.1 ± 0.7	<0.0005	0.010	<0.0005
Oozing/crust	0.4 ± 0.6	1.2 ± 0.9	1.6 ± 0.9	<0.0005	0.001	<0.0005
Excoriation	1.3 ± 0.6	1.7 ± 0.5	2.1 ± 0.6	<0.0005	0.001	<0.0005
Lichenification	0.9 ± 0.6	1.4 ± 0.7	1.7 ± 0.6	<0.0005	0.008	<0.0005
Dryness	1.1 ± 0.6	1.4 ± 0.6	1.7 ± 0.7	0.001	0.015	0.001
Pruritus	5.6 ± 1.7	6.7 ± 2.2	7.1 ± 1.9	0.013	0.073	0.004
Sleep loss	3.2 ± 3.0	5.6 ± 3.1	6.6 ± 2.5	<0.0005	0.020	<0.0005
NESS	11.5 ± 2.1	12.7 ± 2.1	13.1 ± 2.3	0.004	0.068	0.001
CDLQI (n = 85)	7.4 ± 4.3 (n = 36)	11.1 ± 6.8 (n = 37)	11.5 ± 7.2 (n = 12)	0.026	0.346	0.007

SH: I versus III ($p = 0.005$).
TEWL: I versus III ($p = 0.006$).
Objective SCORAD: I versus II ($p < 0.0005$), I versus III ($p < 0.0005$).
Affected area: I versus II ($p = 0.002$), I versus III ($p < 0.0005$).
Intensity: I versus II ($p < 0.0005$), I versus III ($p < 0.0005$).
Erythema: I versus II ($p = 0.001$), I versus III ($p = 0.006$).
Edema/papulation: I versus II ($p = 0.001$), I versus III ($p = 0.004$).
Oozing/crust: I versus II ($p < 0.0005$), I versus III ($p < 0.0005$).
Excoriation: I versus II ($p = 0.003$), I versus III ($p < 0.0005$), II versus III ($p = 0.040$).
Lichenification: I versus II ($p = 0.003$), I versus III ($p < 0.0005$).
Dryness: I versus II ($p = 0.012$), I versus III ($p = 0.001$).
Pruritus: I versus III ($p = 0.034$).
Sleep loss: I versus II ($p = 0.002$), I versus III ($p = 0.001$).
NESS: I versus II ($p = 0.032$), I versus III ($p = 0.043$).
CDLQI: I versus II ($p = 0.019$).

patients with moderate-to-severe and chronic disease in ambulatory clinic settings has to make a number of clinical decisions, namely, (i) if routine management with topical emollient and corticosteroid and adherence is optimal, (ii) whether superimposed or secondary SA colonization/infection is present which may account for the moderate-to-severe disease state, (iii) if further investigations such as skin swabs for bacteriology and antimicrobial sensitivity are to be taken, (iv) which sites the swabs are to be taken, and (v) what antibiotic and escalation of treatment modalities to be prescribed. These decisions may impose inconvenience and additional costs to the patient/family especially those in the private setting. Even if swabs are to be taken, it will take 48–72 h before culture results are available. Physician may need to make clinical decision based on symptomatology and start a course of oral antibiotic pending bacteriology.

Our results indicate that clinical features such as disease severity (objective SCORAD) and lesion intensity are associated with moderate-to-severe SA growth in lesional skin. Therefore,

clinical features can aid physician to determine if SA colonization/infection is present. Hauser et al. (18) reported that the frequency of SA carriage was significantly higher in the apparently normal skin of AD patients than in healthy individuals. Compared to the normal skin of patients, SA density was 100–1000 times higher in three different kinds of lesional skin (dermatitic, lichenified, and impetiginized sites). However, even in the most affected skin areas, significant isolates of SA was only found in approximately one-third of the specimens, which in part implies direct pathogen invasion may not be the sole factor in mediating AD (7). Acute exacerbations of eczema often present with oozing due to acute inflammation alone, but not infection, especially on the faces of young children (7).

Based on the current findings, physicians at our busy outpatient clinics may consider a course of cloxacillin or equivalent as a therapeutic option when managing a patient with moderate-to-severe disease if he/she has high objective SCORAD and “intense lesions.” In patients with history of

Table 3. Binomial logistic regression for growth of SA in the most severely affected skin site.

<i>No-to-scanty growth (I + II) versus moderate-to-heavy growth (III)</i>	
Method 1: objective SCORAD as a whole	Logistic regression to ascertain the effects of sex, TEWL, objective SCORAD, pruritus, sleep loss, and NESS on the likelihood that patients have moderate-to-heavy SA colonization in their most severely affected skin site: increasing objective SCORAD was associated with an increased likelihood of carrying moderate-to-heavy growth of SA (adjusted odds ratio is 1.080 with a 95% CI of 1.025 to 1.138; $p=0.004$).
Method 2: split objective SCORAD to extent and intensity	Logistic regression to ascertain the effects of sex, TEWL, extent, intensity, pruritus, sleep loss, and NESS on the likelihood that patients have moderate-to-heavy SA colonization in their most severely affected skin site: increasing intensity was associated with an increased likelihood of carrying moderate-to-heavy growth of SA (adjusted odds ratio is 1.721 with a 95% CI of 1.166 to 2.540; $p=0.006$).
Method 3: split intensity to components of intensity	Logistic regression to ascertain the effects of sex, TEWL, extent, erythema, edema/papulation, oozing/crust, excoriation, lichenification, dryness, pruritus, sleep loss, and NESS on the likelihood that patients have moderate-to-heavy SA colonization in their most severely affected skin site: increasing erythema (adjusted odds ratio is 5.183 with a 95% CI of 1.274 to 21.081; $p=0.022$) and increasing lichenification (adjusted odds ratio is 3.823 with a 95% CI of 1.097 to 13.326; $p=0.035$) were associated with an increased likelihood of carrying moderate-to-heavy growth of SA.
<i>No growth (I) versus any growth (II + III)</i>	
Method 1: objective SCORAD as a whole	Logistic regression to ascertain the effects of SH, objective SCORAD, and pruritus on the likelihood that patients have any SA colonization in their most severely affected skin site: increasing objective SCORAD was associated with an increased likelihood of carrying SA (adjusted odds ratio is 1.058 with a 95% CI of 1.016 to 1.101; $p=0.007$).
Method 2: split objective SCORAD to extent and intensity	Logistic regression to ascertain the effects of SH, extent, intensity, and pruritus on the likelihood that patients have any SA colonization in their most severely affected skin site: decreasing extent (adjusted odds ratio is 0.938 with a 95% CI of 0.890 to 0.990; $p=0.020$) and increasing intensity (adjusted odds ratio is 1.990 with a 95% CI of 1.338 to 2.959; $p=0.001$) were associated with an increased likelihood of carrying SA.
Method 3: split intensity to components of intensity	Logistic regression to ascertain the effects of SH, extent, erythema, edema/papulation, oozing/crust, excoriation, lichenification, dryness, and pruritus on the likelihood that patients have any SA colonization in their most severely affected skin site: decreasing extent was associated with an increased likelihood of carrying SA (adjusted odds ratio is 0.929 with a 95% CI of 0.872 to 0.990; $p=0.023$).

Table 4. Binomial logistic regression for growth of SA in the right antecubital fossa.

<i>No-to-scanty growth (I + II) versus moderate-to-heavy growth (III)</i>	
Method 1: objective SCORAD as a whole	Logistic regression to ascertain the effects of SH, TEWL, emollient frequency, objective SCORAD, and sleep loss on the likelihood that patients have moderate-to-heavy SA colonization in their right antecubital fossa: increasing TEWL was associated with an increased likelihood of carrying moderate-to-heavy growth of SA (adjusted odds ratio is 1.394 with a 95% CI of 1.033 to 1.881; $p=0.030$).
Method 2: split objective SCORAD to extent and intensity	Logistic regression to ascertain the effects of SH, TEWL, emollient frequency, extent, intensity, and sleep loss on the likelihood that patients have moderate-to-heavy SA colonization in their right antecubital fossa: increasing TEWL was associated with an increased likelihood of carrying moderate-to-heavy growth of SA (adjusted odds ratio is 1.364 with a 95% CI of 1.011 to 1.842; $p=0.042$).
Method 3: split intensity to components of intensity	Logistic regression to ascertain the effects of SH, TEWL, emollient frequency, extent, edema/papulation, oozing/crust, excoriation, lichenification, dryness, and sleep loss on the likelihood that patients have moderate-to-heavy SA colonization in their right antecubital fossa: increasing TEWL (adjusted odds ratio is 1.736 with a 95% CI of 1.146 to 2.629; $p=0.009$) and increasing excoriation (adjusted odds ratio is 79.601 with a 95% CI of 1.786 to 3547.658; $p=0.024$) were associated with an increased likelihood of carrying moderate-to-heavy growth of SA.
<i>No growth (I) versus any growth (II + III)</i>	
Method 1: objective SCORAD as a whole	Logistic regression to ascertain the effects of SH, TEWL, emollient frequency, objective SCORAD, pruritus, sleep loss, and NESS on the likelihood that patients have moderate-to-heavy SA colonization in their right antecubital fossa: increasing objective SCORAD (adjusted odds ratio is 1.071 with a 95% CI of 1.011 to 1.134; $p=0.019$) and increasing sleep loss (adjusted odds ratio is 1.401 with a 95% CI of 1.059 to 1.854; $p=0.018$) were associated with an increased likelihood of carrying SA.
Method 2: split objective SCORAD to extent and intensity	Logistic regression to ascertain the effects of SH, TEWL, emollient frequency, extent, intensity, pruritus, sleep loss, and NESS on the likelihood that patients have moderate-to-heavy SA colonization in their right antecubital fossa: increasing intensity (adjusted odds ratio is 1.720 with a 95% CI of 1.121 to 2.637; $p=0.013$) and increasing sleep loss (adjusted odds ratio is 1.366 with a 95% CI of 1.019 to 1.832; $p=0.037$) were associated with an increased likelihood of carrying SA.
Method 3: split intensity to components of intensity	Logistic regression to ascertain the effects of SH, TEWL, emollient frequency, extent, erythema, edema/papulation, oozing/crust, excoriation, lichenification, dryness, pruritus, sleep loss, and NESS on the likelihood that patients have moderate-to-heavy SA colonization in their right antecubital fossa: increasing oozing/crust (adjusted odds ratio is 2.936 with a 95% CI of 1.126 to 7.655; $p=0.028$) and increasing sleep loss (adjusted odds ratio is 1.369 with a 95% CI of 1.010 to 1.856; $p=0.043$) were associated with an increased likelihood of carrying SA.

Figure 1. Receiver operating characteristic curve for objective SCORAD and lesion intensity (area under curve = 0.76 and 0.78, respectively).

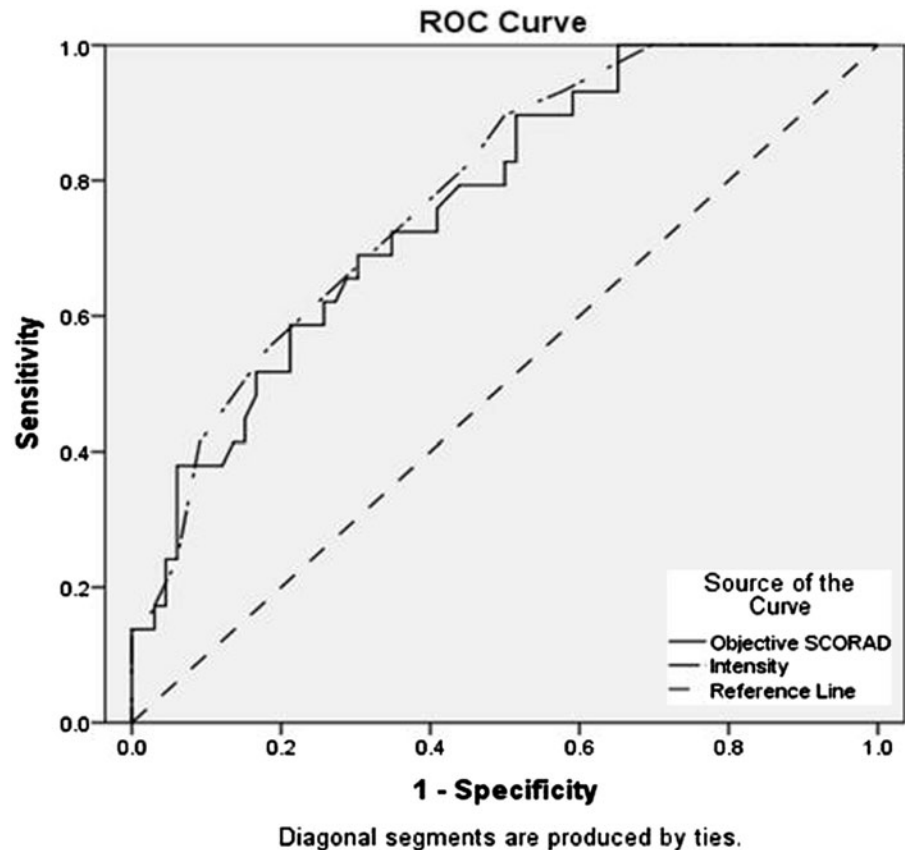


Table 5. Antibiotics-resistant pattern of isolated SA.

	Total	Growth of SA in most severely affected site	Growth of SA in right antecubital fossa
All SA	<i>n</i> = 122	<i>n</i> = 68	<i>n</i> = 54
Resist to clindamycin (%)	13 (10.7)	6 (8.8)	7 (13.0)
Resist to cloxacillin (%)	7 (5.7)	4 (5.9)	3 (5.6)
Resist to co-trimoxazole (%)	1 (0.8)	0 (0.0)	1 (1.9)
Resist to erythromycin (%)	23 (18.9)	11 (16.2)	12 (22.2)
Resist to fusidic acid (%)	22 (18.0)	11 (16.2)	11 (20.4)
Methicillin-sensitive SA (% among all SA)	<i>n</i> = 115 (94.3)	<i>n</i> = 64 (94.1)	<i>n</i> = 51 (94.4)
Resist to clindamycin (%)	10 (8.7)	4 (6.3)	6 (11.8)
Resist to cloxacillin (%)	0 (0.0)	0 (0.0)	0 (0.0)
Resist to co-trimoxazole (%)	1 (0.9)	0 (0.0)	1 (2.0)
Resist to erythromycin (%)	20 (17.4)	9 (14.1)	11 (21.6)
Resist to fusidic acid (%)	22 (19.1)	11 (17.2)	11 (21.6)
Methicillin-resistant SA (% among all SA)	<i>n</i> = 7 (5.7)	<i>n</i> = 4 (5.9)	<i>n</i> = 3 (5.6)
Resist to clindamycin (%)	3 (42.9)	2 (50.0)	1 (33.3)
Resist to cloxacillin (%)	7 (100.0)	4 (100.0)	3 (100.0)
Resist to co-trimoxazole (%)	0 (0.0)	0 (0.0)	0 (0.0)
Resist to erythromycin (%)	3 (42.9)	2 (50.0)	1 (33.3)
Resist to fusidic acid (%)	0 (0.0)	0 (0.0)	0 (0.0)

penicillin allergy, erythromycin, clindamycin, and co-trimoxazole may be options for the empirical treatment of suspected bacterial infection at our clinic. Despite disease severity and preference of MRSA in other childhood conditions in our locality, MRSA in childhood AD is not prevalent (7,9,19,20). MRSA (5.7%) was sensitive to co-trimoxazole and fucidin in the present cohort.

The study has some limitations. It did not confirm the pathogenic role of SA since no comparison was made between the severity of AD before and after the eradication of the SA. Besides,

the numbers of patients included were relatively small and there was no healthy control.

We conclude that clinical features, especially severity and intensity, are useful in “predicting” presence of SA colonization/ infection in patients with AD. These findings will facilitate physicians to manage patients before bacteriology results become available. We would recommend that cultures be considered not only in children with chronically extensive eczema, but also in children with an acute worsening of the intensity or severity of

their eczema. Cloxacillin has a favorable sensitivity profile. Thus far there is no evidence for a positive effect of oral or topical antibiotics in non-infected AD, and the clinical effects of antibiotics on infected AD are likely to be transient (9,21). Further studies are required to investigate if treatment targeting at reducing disease severity may improve quality of life and reduce SA colonization/infection without use of antibiotics or antiseptics. As *colonization* and *infection* are ambiguous and potentially overlapping clinical states, we recommend to abandon these terms and propose to describe quantitatively/semi-quantitatively SA *isolation* as none, mild, scanty, moderate or heavy growth instead in clinical trials.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

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