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Research Article

Methicillin-Resistant Staphylococcus Aureus Colonization in Lesional and Non-Lesional Skin of Children with and without Atopic Dermatitis

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Abstract

Background: Staphylococcus aureus (SA) is highly susceptible for colonization in atopic dermatitis (AD) lesions, leading to the aggravation of the disease. We aimed to determine the extent of SA and MRSA colonization in lesional and non-lesional skin of children with AD and its comparison with that of healthy children, as well as to investigate the antibiotic sensitivity of isolated SA.

Method: In this cross-sectional study, skin swabs from both lesional and non-lesional regions of 100 children with AD (case group) and one region of the skin of 100 children without AD (control group) were collected and investigated in terms of the existence of SA and MRSA. Antibiotics susceptibility tests were performed on isolated SA. The severity of disease was determined based on the SCORAD criteria.

Results: The rates of SA colonization in the case and control groups were 21 and 4%, respectively. The MRSA colonization rates in lesional and non-lesional skin were 36.7 and 4.16%, respectively, in the case group, compared with 25% in the control group, which was not statistically significant. All isolated MRSA were community-acquired. SA and MRSA colonization rates had a direct relationship with disease severity. A family history of atopy increased the rate of SA colonization. The highest and lowest antibiotics resistances were reported for penicillin (100%) and vancomycin (0%), respectively.

Conclusion: Considering the high resistance to conventional antibiotics (penicillin, oxacilin, and erythromycin), cultures and antibiotics susceptibility tests are recommended for the treatment of secondary AD infections.

Keywords: MRSA colonization; Atopic dermatitis; Children

Introduction

Colonization and infections with staphylococcus aureus (SA) have increased in recent years in children with atopic dermatitis (AD) [1,2]. SA has been introduced as an activating factor for AD [3-5]. These bacteria typically colonize in the nose, groin and perineum [4,6].

The existence of these bacteria in skin lesions and their super antigen and toxin production leads to the aggravation of dermatitis with AD. SA colonization has been observed in lesional and non-lesional regions of the skin in patients suffering from AD. However, bacteria concentration in lesional regions is higher [2,3,6,7]. Eczema lesions in these patients are a source for SA transfer [8].

Antibiotics resistance and emergence of multi-drug resistant SA including MRSA or fusidic acid SA (FRSA) or mopirucin are serious problems in patients with AD [6].

During the 1960s, a strain of SA called methicillin-resistant staphylococcus aureus (MRSA) was found as a serious threat for the patients' health worldwide [9]. MRSA included two species called healthcare associated MRSA (HA-MRSA) and community acquired MRSA (CA-MRSA) which were transferred easily resulting in lethal and severe infections [10,11]. Chronic skin diseases such as eczema are related to MRSA colonization [9].

Effective factors on MRSA colonization in patients with AD include history of hospital admission [8,11,12], severity of disease [3,5], age, sex, taking topical steroids in combination with topical Calcineurin Inhibitors (TCI) (8), excessive and prolonged dose of antibiotics such as flucloxacillin [5,13].

We aimed to identify SA and MRSA colonization rates in lesional and non-lesional skin of children with AD as well as investigate the antibiotics resistance pattern of native strains in our region.

Patients and Methods

In this cross-sectional study, 100 children with AD aged 3 months to 17 years referred to Afzalipoor Hospital, Kerman, Iran, during November 2011 to December 2012 were assessed using the successive sampling method. After receiving their biographies and clinical examination, AD was diagnosed based on the UK working party standard. Then, all demographic and clinical characteristics of the patients and disease severity were registered by a single researcher in prepared standard forms based on the SCORAD (severity scoring of AD) system [14]. 100 healthy children in terms of skin disease

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who did not have any allergies and atopic history in their families were selected from daycare centers and schools as the control group. Written informed consent was obtained from children older than 15 or from parents of younger children.

The exclusion criteria included safety deficiency, an acute viral, bacterial of fungal disease, history of taking antibiotics topically during 2 prior weeks or orally or via injection during 4 prior weeks, concurrence of another skin disease, and employment of parents in hospitals and healthcare centers. Two samples were obtained from each child with AD, one from lesional regions (eczematus) and the other from non-lesional regions. These two samples were taken from 2 anatomically similar areas. One skin sample was also taken from each healthy child.

Healthy children were matched with the children in the case group in terms of sampling region, age, and sex. Sampling regions in each group included scalp, face, neck, front and back of the body, upper and lower extremities. Sampling was not conducted from groin and interior nose.

For sampling, a sterile cotton swab was soaked in normal saline and was rubbed on the respective lesion using the standard sampling method [15] for 5-10 times and 15-20 minutes. Then, the swab was put in testing tube with Stuart culture (product of Merk Co.). All sampling steps were performed next to alcohol light. The samples were transferred to the laboratory within 18 hours for respective investigations. During these hours, the samples were kept in the refrigerator. The samples in the lab were inoculated in bloody agar culture and Eosin-Methylen Blue (EBM). After 24 to 48 hours of incubation at 37°c, colonies suspected of SA were investigated with gram staining, catalase and coagulase test, and then their activities in mannitol salt agar culture were carefully evaluated. After the definite confirmation of SA, antibiogram tests using Kirby Bauer diffusion disk were performed to determine sensitive conditions to Oxacillin and identify MRSA and MSSA strains.

In order to investigate resistance to Methicillin and MRSA isolation from other bacteria, MRSA isolation was conducted using Oxacillin disks (Padtan Teb, Iran) on Moler Hinton culture and 6 μg/ml Oxacilin with 4% salt were added to identify real MRSAs in this culture [15]. Antibiotic applied in antibiogram included penicillin, oxacilin, vancomycin, gentamycin, ciprofluxacin, erythromycin, and clindamycin.

Data were analyzed using SPSS software, version 16. Chi-square test was used to compare colonization rate in both groups and Spearman's correlation coefficient was used to examine the relationship among MRSA colonization, disease severity and the patients' age.

Results

In our study, 200 cultures from patients with AD (from lesional and non-lesional regions) were obtained. The mean±SD age of the children in case group was 4.7±4.73 and half the children were girls. 54% of the control groups were girls and 46% were boys with a mean±SD age of 4.73±5.6 years.

The demographic and clinical characteristics of the participants were presented in Table 1. In the case group, of the 100 lesional samples, 16% showed SA colonization, 37.5% of which were MRSA. Of 100 sample cultures of non-lesional skin, only 12% were positive for MRSA. Of the 100 children in the control group, 4 (4%) had SA colonization among whom only one person (25%) had MRSA colonization.

Table 1: Demographic data.

Characteristics	Percentages
Patients	100
Female / Male	50% / 50%
Age range	3 months up to 17 years
Place of attendance (Private medical office/ Public medical office)	55% / 45%
Location of lesion (Extremities/trunk/head and neck)	69% / 38% / 50%
Type of lesion (Extensor/flexor)	54% / 45%
Family history (Allergic rhinitis/asthma/atopic dermatitis)	26% / 17% / 31%
Personal history (Allergic rhinitis/asthma)	49% / 18%
Admission in hospital (No/Yes)	92% / 8%
Drug history (Topical/Systemic/Both)	69% / 3% / 11%
SCORAD criteria (Severe/Intermediate/Mild)	9% / 25% / 66%
Location of obtaining sample (Trunk/Neck/Face/Head/ extremities)	18% / 4% / 23% / 3% / 52%

Of all 200 cultures obtained from the case group, 40% (n=80) of the samples were positive for microbial culture, of which 35% were colonized with SA. Other bacteria included bacillus (27.5%), staph. epidermis (21.2%) and coagulase negative staph (16 .2%).

Generally, we can state that 21%of the patients had at least one positive culture of SA colonization, as follows: 9% of the patients had SA colonization on lesional regions, 33.35% of which were MRSA-SA, 5% of the patients had SA colonization on non-lesional region, 40% of which were MRSA, 7% of the patients had SA colonization on both lesional and non-lesional regions, 42.8% of which were MRSA. Of the 66% of the patients that had AD, only 3 patients showed SA colonization, none of which were MRSA. Of the 25% of the patients with moderate AD, 10 (40%) had SA colonization among which only 3 (30%) were colonized with MRSA. Of the 9% of patients with acute AD, 8 (89%) had SA colonization, among which 62.5% were colonized with MRSA. We found no statistically meaningful difference between SA colonization rate on lesional and non-lesional regions in children with AD (p=0.4). However, the SA colonization rate on lesional regions in the case group compared with the control group and this ratio between non-lesional regions in the case and control group were statistically significant (P=0.02 and P=0.03, respectively). Generally, there was a significant difference between SA colonization in children with AD (21%) and the healthy ones (4%). We found no significant difference between MRSA colonization on lesional and nonlesional regions in the case group (P=0.82, Chi-square test). Also, no significant difference was observed with respect to MRSA colonization in lesional skin (37.5%) and nonlesional (41.6%) skin in the children of the case group compared with those of the control group (25%, P=0.9).

The relationship between the existence of SA and disease severity was evaluated using Spearman's correlation coefficient (based on SCORAD) which was statistically significant (p=0.37). Moreover, a

Table 2: Sensitivity/resistance to antibiotics in SA isolated from both groups.

Antibiotic	SA isolated from case group	SA isolated from control group
	Sensitive / Resistant	Sensitive / Resistant
Ciprofluxacin	82% / 18%	100% / 0%
Gentamicin	86% / 14%	100% / 0%
Erythromycin	64% / 36%	50% / 50%
Oxacillin	61% / 39%	75% / 25%
Clindamycin	82% / 18%	75% / 25%
Penicillin	0% / 100%	0% / 100%
Vancomycin	100% / 0%	100% / 0%

Table 3: SA sensitivity/resistance to antibiotics in *MRSA/MSSA in both groups.

Antibiotic	Case group		Control Group	
	MRSA	MSSA	MRSA	MSSA
	Sensitive/ resistant	Sensitive/ resistant	Sensitive/ resistant	Sensitive/ resistant
Ciprofluxacin	100% / 0%	100% / 0%	54.5% / 45.5%	100% / 0%
Gentamicin	100% / 0%	100% / 0%	63.6% / 36.4%	100% / 0%
Erythromycin	0% / 100%	66.6% / 33.3%	9% / 91%	100% / 0%
Oxacillin	0% / 100%	100% / 0%	0% / 100%	100% / 0%
Clindamycin	100% / 0%	66.6% / 33.3%	54.5% / 45.5%	100% / 0%
Penicillin	0% / 100%	0% / 100%	0% / 100%	0% / 100%
Vancomycin	100% / 0%	100% / 0%	100% / 0%	100% / 0%

*MRSA: Methicillin Resistant Staphilococcus Areous
*MSSA: Methicillin Sensitive Staphilococcus Areous

significant relationship was observed between disease severity and MRSA colonization (P=0.016 and P=0.45) so that as disease severity increased, SA and MRSA colonization increased as well. We found a significant relationship between SA colonization and age (P=0.04, R= 0.87, Spearman's correlation coefficient); so that SA colonization increased with age. However, there was no relationship between MRSA colonization rate and age. We found no statistically significant difference in SA and MRSA colonization rates with respect to sex, lesions' region (head and neck, body), type of lesion (flexor, extensor), history of taking medicine and its type, and history of hospital admission. Only 8 patients had a history of hospital admission over the recent year among whom only 3 patients were colonized with SA.

In patients with SA colonization, we noticed that 75% of them had a family history of atopic diseases (allergic rhinitis, asthma, and atopic dermatitis) (P=0.04, Chi-square test). 54% of the patients with SA colonization against 27.3% without SA colonization had family history of atopic dermatitis (P=0.005); however, no significant difference was found in terms of asthma (P=0.9) and allergic rhinitis (P=0.3). Therefore, patients with a family history of atopic dermatitis were more likely to be colonized by SA. 67.9% of the patients with a self-reported history of other atopic diseases (asthma and allergic rhinitis) were colonized with SA compared with 32.1% of the patients with AD (p=0.113). Also, no significant difference was found in this group with respect to asthma and allergic rhinitis (P=0.9 and P=0.2). It was observed that 91% of the patients with MRSA colonization against 59% of those without MRSA colonization had a family history of atopic diseases (P=0.06). Family history was compared with MRSA colonization in terms of asthma, atopic dermatitis and allergic rhinitis which was not significant. There was no significant relationship between MRSA colonization and self-reported history of other atopic diseases (P=0.93).

Antibiogram

As shown Table 2, all isolated SAs were resistant to penicillin (100%) and then the highest antibiotic resistance was related to oxacillin (39%) and erythromycin (36%) in the case group. In the control group the highest antibiotic resistance was related to erythromycin (50%), followed by oxacillin (25%) and clindamycin (25%). The highest antibiotics sensitivity in case group was related to vancomycin (100%) followed by gentamicin (86%), clindamycin and ciprofluxacin (82%). The highest antibiotics sensitivity in the control group was related to ciprofluxacin and gentamicin (100%). Antibiotics sensitivities are presented in Table 3. As shown, all isolated MRSAs were resistant to the tested antibiotics except vancomycin while MSSA was only resistant to penicillin.

Discussion

In our study, 21% of the patients had SA colonization compared with only 4% in the control group. In another study in Sri Lanka dermal SA colonization in patients with AD was reported to be 57% [3]. Also, in a study in Iran, the SA colonization rate was reported 66% [16]. In these studies, dermal swabs were collected from two lesional regions but in our study, a swab of one lesion was taken and was inoculated in bloody agar culture and EMB.

We found that on lesional skin, 36.7% of the patients had MRSA colonization compared with only 25% in the control group. Another study by Suh and colleagues in Philadelphia reported an SA colonization rate of 80%, 16% of which was MRSA [8]. In the mentioned study, the samples were collected simultaneously from skin and nose and the results are not independent for these regions. Since nostrils are the main source of SA colonization [3,5,17], this may lead to the high reported prevalence in that study. Prior hospital admission [8] and chronic eczema lesions [3,5] are risk factors for SA colonization. In our research, all people were outpatients, most of which had no history of hospital admission (92%). Geographical factors may have contributed to the lower prevalence of colonization in our study. SA colonization in was 4% in the control group compared with 2-40% reported in Sri Lanka (3).

In other studies, it has been observed that SA colonization is directly related to AD severity and age which was consistent with our study. MRSA colonization rate was significantly related to the severity of AD which is also confirmed in studies conducted in the U.S [5] and Sri Lanka [3].

In this study, SA colonization in lesional skin of children with AD was 16% which was reported 87% in a study by Kedzeirska and co-workers in the U.S [5]. However, in the mentioned study, samples of lesional skin were obtained from 4 regions while in our study; only a sample of one lesion was taken. In the same study [5], SA colonization in nonlesional skin of patients with AD was reported 44% compared with 12% in our study. However, various studies show colonization rates of 2-25% in nonlesional skin [1,2] (18). Regarding these percentages, SA colonization in lesional and nonlesional skin of children with AD did not show significant difference although

SA is more colonized in lesional skin [1,2] (2, 5, and 6). Gomez and colleagues obtained similar results in Sri Lanka [3].

In our study, MRSA/SA colonization rates were 37.5% and 41.6% in lesional and nonlesional skin, respectively; compared with 7.4% and 4% in a study in the U.S [19]. In south Korea [20], MRSA/SA colonization rate on AD skin was 18.3%. Another recent study in Taiwan reported a colonization rate of 30.8% [1,2] (17). On the other hand, a research conducted in Canada in 2011, this value was only 0.5% (12). MRSA/SA colonization rate obtained in our study was 38%. It has been proved that antibiotic sensitivities are different based on geographical locations [4,21]. Geographical factors might have lead to the higher prevalence of MRSA in our study.

We found that MRSA colonization was 25% on the skin of healthy children. This value was reported 1-3% and 5.1% in the U.S [8] and South Korea [20], respectively which is not statistically significant with respect to the lower prevalence of positive SA samples obtained from healthy children (4 cases). Comparison of MRSA/SA colonization between lesional (37.5%) and non-lesional skin (41.6%) in children with AD was not significantly difference but this difference was significant in other studies [3,5] which may be due to the severity of inflammation in skin lesions. The ratio of MRSA/SA colonization in lesional and non-lesional skin of children with AD was compared to that of healthy children which did not differ significantly (P=0.9). It is worthy of noting that the SA colonization rate in children with AD was much higher compared with healthy children.

MRSA colonization rate had no relationship with sex [4,22], the site of involvement and the type of lesions [3], type of medication [22] and usage in other studies which was consistent with our study. SA colonization is higher in individuals with a history of atopic diseases. Among atopic diseases, people with family history of atopic dermatitis are more likely to be colonized with SA but there was no relationship between SA colonization and self-reported history of other atopic diseases in our study. There was no significant relationship between personal and family history of atopic diseases. In a study in the U.S, history of hospital admission was related to SA/MRSA colonization and it was introduced as a risk factor for such colonization [8]. We found no relationship between hospital admission and SA/MRSA colonization which may be due to the small number of positive cases.

MRSAs obtained in our study were of the CA-MRSA type. Considering that all SAs were related to outpatients and they had no history of prior infection with MRSA, invasive surgery [3], and hospital admission during the last year [3,20], such results were expected.

We found that all SAs were resistant to penicillin. In a study by Farajzadeh and co-workers in Iran [16], the degree of resistance was reported to be 90% compared with 88% in studies conducted in the U.S [5] and Taiwan [17] suggesting an incremental trend of resistance to penicillin in this area.

Resistance to erythromycin was 33.7%. Other studies in Iran [16] and the U.S [5] have reported resistance rates of 66.7% and 25%, respectively; the reason for which is not clear. Reduced used of erythromycin, as a result of taking other oral and topical antibiotics, may justify this issue. The highest sensitivity in our study was related to vancomycin which may be used for the treatment of antibiotics-resistant SAs.

Cloxacillin is the initial treatment of choice for children with AD who require systemic antibiotic therapy in Iran. As the rate of resistance to ciprofloxacin was lower than oxacillin it may be a good choice as an empiric antibiotic therapy in these patients. Although the rate of resistance to gentamycin and clindamycin was as almost similar to ciprofloxacin, considering the side effects of these drugs and the point that gentamycin is not available orally, ciprofloxacin is preferred as the first line antibiotic therapy in this area.

Side effects of clindamycin include diarrhea (2-20%), pseudomembranous colitis (0.01–10%), skin rash (10%), Stevens-Johnson syndrome, elevated liver enzymes, cytopenia, neuromuscular blockade and anaphylaxis [23]. The side effects of gentamicin include nephrotoxicity, ototoxicity, local inflammation at the site of intrathecal and intraventricular injection leading to radiculitis, neuromuscular blockade, and anaphylaxis [24].

The MRSA isolates found in our study showed some degree of resistance to all of the antibiotics except vancomycin. A 100% resistance to penicillin was found in both MRSA and MSSA.

Existence of Multi-drug resistant SA can create acute infections resistant to treatment. In a study conducted in Taiwan the degree of multi-drug resistant SA in patients with AD was reported 70% [17] which is higher compared with our study in which 39.2% of SAs were resistant to two antibiotics as a result of MRSA tags found in that area or excessive use of antibiotics. High CA/MRSA colonization in children with AD is worrying because they are susceptible to aggressive skin infections [20]. Prolonged and unnecessary antibiotics treatment in patients with AD increases resistance and MRSA and multi-drug resistant SA [5]. As a result, we must be careful in prescribing antibiotics to such patients to prevent higher antibiotics resistance [5,22].

One of the limitations in our study was that we were not able to determine MRSA tags because of lack of sufficient facilities.

Conclusion

It can be concluded that in our study SA/MRSA colonization did not show significant difference between lesional and non-lesional skin of the children with atopic dermatitis and healthy children.

Considering lower resistance rate to ciprofloxacin in our study, it can be a good choice for empirical therapy in patients with atopic dermatitis and vancomycin should be considered for cases with antibiotics-resistant SA_S.

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