Staphylococcus aureus Colonization in Atopic Skin Diseases

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An epidemiologic investigation on Staphylococcus aureus (S. aureus) colonization in atopic skin diseases was conducted at İnönü University Turgut Özal Medical Center. The incidence of unaffected skin, lesional skin, and nasal positivity for S. aureus was examined in a total of 60 patients with atopic dermatitis and other atopic skin diseases. 50 healthy subjects were studied as controls. Normal skin, lesional skin, and nasal nostrils were colonized with S. aureus in 25.0%, 40.0%, and 41.7% of patients, respectively. In controls, the colonization rate of S. aureus was 2% in healthy skin and 16% in nasal nostrils ($p<0.001$). According to the results of antibiogram; vancomycine, tobramycine, sulbactam-ampicilline, and gentamycine were considered as the most effective drugs against S. aureus. [Journal of Turgut Özal Medical Center 1996;3(4):299-302]

Key Words: *Staphylococcus aureus, colonization, atopic skin diseases*

Atopik deri hastalıklarında *Staphylococcus aureus* kolonizasyonu

Atopik deri hastalıklarında Staphylococcus aureus kolonizasyonu sıklığını belirlemek amacıyla İnönü Üniversitesi Turgut Özal Tıp Merkezinde 60 hasta ve 50 sağlıklı kişi üzerinde epidemiyolojik bir çalışma yapıldı. Hastaların; sağlam derilerinde %25.0, lezyonlu derilerinde %40.0 ve burun deliklerinde %41.7 sıklıkla S. aureus kolonizasyonu olduğu görüldü. Kontrollerin sağlıklı derilerinde %2.0, burun deliklerinde ise %16.0 oranında S. aureus üredi ($p<0.001$). Yapılan antibiyogramlar, S. aureus'a karşı en etkili antibiyotiklerin; vankomisin, tobramisin, sulbaktam-ampisilin ve gentamisin olduğunu gösterdi. [Turgut Özal Tıp Merkezi Dergisi 1996;3(4):299-302]

Anahtar Kelimeler: *Staphylococcus aureus, kolonizasyon, atopik deri hastalıkları*

Normal skin flora is the term used to describe the various bacteria and fungi that are permanent residents of the skin. There is a distinction between the normal flora and carrier state. The term 'carrier' implies that an individual harbors a potential pathogen and therefore can be a source of infection for others. It is most frequently used in reference to asymptomatic infection or to a patient who has recovered from a disease but continues to carry the microorganisms and shed it for a long period. When an microorganism establishes more than a temporary relationship, the host is said to be colonized by that microorganism. *Staphylococcus epidermidis* is the most important microorganism at

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normal skin flora. Other microorganisms, such as *Staphylococcus aureus*, *Corynebacterium* (*diphtheroids*), various streptococci, *Pseudomonas aeruginosa*, anaerobes (eg. *Propionibacterium acnes, Peptostreptococcus*) and yeasts (eg. *Candida albicans*) are less important microorganisms. The nose is colonized by a variety of streptococcal and staphylococcal species, the most significant of which is the pathogen *S.aureus*. *S.epidermidis*, *diphtheroids* and various streptococci are also found in the nose.

The bacterial skin flora of patients with atopic dermatitis is different from that in healthy people. In addition, such patients more often suffer from microbial infections such as impetigo, folliculitis, and furunculosis. Differences in sebum and sweat secretion and increased bacterial adhesion to epithelial cells in atopic skin diseases may predispose to enhanced amounts of *Staphylococcus aureus* on the skin. *S. aureus* secrets exotoxins called superantigens, which stimulate a large proportion of T cells. On the other hand, staphylococcal antigens may induce allergic reactions (ie, the release of inflammatory mediators such as leukotrienes and histamine). In addition, protein A, a component of the cell wall of *S. aureus*, is a potent B cell mitogen. This understanding provides a rationale for attempting to reduce the staphylococcal skin colonization of patients with atopic eczema and correlates with the clinical observation in a number of situations of marked improvement in atopic dermatitis following antibiotic treatment (1-3).

This epidemiologic investigation was performed to establish the incidence of *S. aureus* colonization in atopic skin diseases.

MATERIAL AND METHODS

The bacterial floras of the healthy skin, lesional skin, and nasal nostrils were assessed quantitatively in 60 patients with atopic dermatitis and some other atopic skin diseases such as neurodermatitis, nummular eczema, lichen simplex, papular urticaria, and dyshidrotic eczema. The patients (35 females and 25 males) were aged 11 months to 63 years. Fifty nonatopic and healthy controls (28 females and 22 males) between 3 to 57 years were also studied. Cotton-tipped swabs and contact agar discs were taken from the worst affected area of atopic skin disease, from an uninvolved site (the inner site of the forearm), and from the nose in patients. Swabs were taken from the forearm and from the nose in controls. Aerobic cultures were performed from these samples. *Staphylococcus* species were selectively sampled by a plate-contact technique using mannitol salt medium-coated film. *Staphylococcus* species were isolated from the film after the number of colonies was counted, and the species were identified by standart identification procedures (microskopic and colony morphology, staphylocoagulase and clumping factor). Antibiotic sensitivities of *S. aureus* strains from patients were tested for eleven antibiotics by Kirby-Bauer disk diffusion method. The differences between study and control groups were tested by chi-square test and the results were considered significant if p value is less than 0.05.

RESULTS

The members of the flora isolated from the patients were shown on Table 1 and from the controls on Table 2. The cultures from healthy skins of patients were negative in 36.7% of patients, *S. aureus* and coagulase negative staphylococcus (CNS) were both isolated from 25.0% of samples. Diphteroids and other members of normal skin flora were also isolated as 8.3% and 5.0%, respectively. From lesional skins of patients; *S. aureus*, CNS, diphteroids, and streptococci were isolated as 40.0%, 26.7%, 3.3%, and 3.3%, respectively. 6.7% of these cultures were evaluated as other normal skin flora members and 20.0% of these cultures were negative. *S. aureus*, CNS, beta-hemolytic

Table 1. The results of cultures in patients

	Healthy skin		**Lesional skin**		**Nasal mucosa**		**Total**	
	n	%	n	%	n	%	n	%
CNS	15	25.0	16	26.7	13	21.7	44	24.4
S.aureus	15	25.0*	24	40.0	25	41.7*	64	35.6
Diphtheroids	5	8.3	2	3.3	2	3.3	9	5.0
Streptococci	-	-	2	3.3	5	8.3	7	3.9
Other NFM	3	5.0	4	6.7	15	25.0	22	12.2
No growth	22	36.7	12	20.0	-	-	34	18.9
Total	60		60		60		180	

CNS: Coagulase negative staphylococcus, NFM: Normal flora members
* : $p<0.001$ when compared to healthy subjects

Table 2. The results of cultures in controls

	Healthy skin		Nasal mucosa		Total	
	n	%	n	%	n	%
CNS	16	32.0	2	4.0	18	18.0
S.aureus	1	2.0	8	16.0	9	9.0
Diphtheroids	-	-	-	-	-	-
Streptococci	-	-	-	-	-	-
Other NFM	3	6.0	40	80.0	43	43.0
No growth	30	60.0	-	-	30	30.0
Total	50		50		100	

CNS: Coagulase negative staphylococcus, NFM: Normal flora members

streptococcus, and diphteroids were the isolates from nostrils of patients as 41.7%, 21.7%, 8.3%, and 3.3%, respectively. In 25.0% of nose cultures were obtained other normal nose flora members. Cultures from healthy skins of controls were negative in 60% of samples. In 32.0% of these cultures CNS, in 2.0% of these samples *S. aureus*, and in 6.0% of them other flora members were isolated. The microorganisms from nasal mucosas of controls were identified *S. aureus* and CNS as 16.0% and 4.0%, respectively. Other normal flora members were isolated in 80.0% of samples.

Sixty four strains of *S. aureus* from patients and 9 strains from controls were isolated ($p<0.001$), and their sensitivities to eleven antibiotics were tested. Some strains were resistant in different degrees to some antibiotics (Table 3). Penicillin G had weak, rifampicin, oxacilline, sefalothin, and clindamycin had moderate, but vancomycine, tobramycine, sulbactam-ampicilline, and gentamycine had strong effects.

DISCUSSION

Atopic dermatitis is a hereditary and distinct form of eczema which may be associated with other atopic manifestations. The etiopathogenesis of atopic dermatitis is unknown and it is widely accepted that both intrinsic and extrinsic factors are involved. The production of bacterial toxins might be important for the pathophysiology of atopic eczema. Recent information implicates the Staphylococcus aureus antigens and exotoxins as likely factors in provoking the inflammatory response in atopic dermatitis.

Table 3. Antibiotic sensitivity of staphylococcus strains (n=64)

	Sensitive		Resistant	
	n	%	n	%
Clindamycin	45	70.3	19	29.7
Erithromycin	41	64.1	23	35.9
Gentamycin	54	84.4	10	15.6
Oxacilline	50	78.1	14	21.9
Penicillin G	9	14.1	55	85.9
Rifampicin	52	81.3	12	18.7
Sefalothin	49	76.6	15	23.4
Sul-ampicil.	56	87.5	8	12.5
TM-Sulpha.	42	65.6	22	34.4
Tobramycin	59	92.2	5	7.8
Vancomycin	61	95.3	3	4.7
Total	518	73.6	186	26.4

Bacterial colonization of the healthy skin and nasal mucosa was consistently more common and greater in amount from patients compared with controls. *Staphylococcus aureus* was the most common pathogen isolated from patients; from the lesional skin in 40.0% of patients, from an uninvolved skin site in 25% of patients, and from nasal nostrils in 41.7% of patients. These values were 2.0% for healthy skin and 16.0% for nasal mucosa in controls ($p<0.001$). In a similar study, these values were 74% in lesional skin and 30% in an uninvolved skin site of patients (4). The reason of the lower percentage for lesional skin in our study may be the inclusion of all the atopic skin diseases besides atopic dermatitis. In another study, the numbers of *S. aureus* colonies in the samples from the forearm and face skins of the patients with atopic dermatitis were significantly greater than from the healthy subjects and there were no significant seasonal differences in *S. aureus* numbers on the skin of patients (5). Similar results were obtained from some other studies (6,7). Leung et al, isolated *S. aureus* in 57.1% of patients with atopic dermatitis (6). Namura et al, found *S. aureus* positivity of nasal nostrils in 40.8% of inpatients and in 95.5% of outpatients with atopic dermatitis and 54.5% of atopic dermatitis patients were positive for *S. aureus* on subungual skin regions. They concluded that subungual spaces seemed to be havens of *S. aureus* besides nostrils, which have been much discussed as a reservoir of *S. aureus* (7). On the other hand, a secondary bacterial infection should be considered a likely cause of relapse or worsening of atopic dermatitis (8).

A trend toward increasing resistance of *Staphylococcus aureus* to standard antibiotic therapy has been reported (9). We observed resistance to some antibiotics in different percentages. Penicillin G had weakest effect on

staphylococcus strains (14.1%), whereas vancomycine and tobramycine were strongest antibiotics against this pathogen (95.3% and 92.2%, respectively). In one study, resistance to penicillin was present in 88% and to two or more antibiotics in 38% of strains (4). In another study, penicillin type antibiotics had weak effects, but cephem type had strong effects on *S. aureus* (5). Erythromycin was effective on 64.1% of strains isolated in our study. Misko et al, reported 22 (26%) of the 85 cultures that grew *S. aureus* were resistant to erythromycin. Treatment failure occurred in one of these patients. They concluded that despite significant erythromycin resistance, there was a low frequency of treatment failure and erythromycin may still be a reasonable agent in the treatment of uncomplicated superficial skin infections (9). The addition of an anti-infective to a topical glucocorticoid preparation for superinfected atopic eczema is still controversial. Korting et al, have found no difference between the groups treated with a topical corticosteroid and corticosteroid plus an anti-infective. They concluded that the addition of an anti-infective to a topical corticosteroid preparation is not to be generally recommended (10). Many other studies have suggested that so many clinical observations present on marked improvement in atopic dermatitis following antibiotic treatment and an attempt to reduce the staphylococcal skin colonization of patients with atopic eczema is necessary (1-3). We also have same conclusion.

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