

Bacterial Skin Colonization In Patients With Atopic Dermatitis / Eczema Syndrome

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Citation

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Abstract

From a total of 286 cases of AD patients, 94.4% and 86.36% yielded positive cultures of eczematous lesions and healthy area of AD skin. Twenty various bacterial types and a total of 959 and 744 isolates were identified from each of above areas respectively. Staph. aureus was the predominant bacterial agent isolated from 60.48% of eczematous lesions, while Staph. epidermidis was predominantly from 57.34% of healthy areas of AD patients ($P < 0.001$). The bacterial numbers ranged from $(0.02-92.0) \times 10^5 \text{ cell/cm}^2$ in eczematous lesions and $(0.11-23.0) \times 10^3 \text{ cell/cm}^2$ in healthy areas of atopic skin ($P < 0.001$).

INTRODUCTION

Atopic dermatitis (AD) is a chronic, pruritic eczematous disease that early always begins in infancy and childhood and follows a remitting / flaring course that may continue throughout life⁽¹⁾. It develops as a result of a complex interrelationship of environmental, immunologic, genetic and pharmacologic factors⁽²⁾. It may be exacerbated by infection, psychological stress, seasonal of climate changes, irritants, and allergens⁽³⁾. The rapid rise in prevalence of AD is thought to be primarily related to changes in our environment. A number of factors can trigger AD, including irritants foods, aerollergens, and infection⁽⁴⁾.

The skin of patients with AD is characterized by higher levels of colonization by aerobes⁽⁵⁾. Significantly higher colony forming units per surface unit could be isolated from clinically normal skin of patients with AD than from corresponding skin sites in normal controls⁽⁶⁾. Acute exudative lesion yielded a mean of 10^7 colony forming units (CFU)/ cm^2 . Thus, the atopic skin must provide a favorable environment for the colonization and multiplication of aerobic bacteria⁽⁵⁾. Coagulase-positive staphylococci (Staph. aureus), which are usually not found on normal skin, accounted for the majority of CFU isolated from AD skin⁽⁷⁾. Staph. epidermidis was the predominant organism isolated from the clinically uninvolved skin of AD patients, whereas Staph. epidermidis was second to Staph. aureus found in lesional skin⁽⁸⁾. It is generally accepted that the normal resident skin flora includes micrococci, staphylococci (e.g. Staph. epidermidis and Staph. capitis), corynebacteria,

propionibacteria (e.g. pr.acnes and pr.granulosum) and Acetobacter. An aerobes out number aerobes by a factor of more than 10, the latter containing most of the pathogenic organisms⁽⁴⁾.

There is no previous study in Iraq - in especially - detailed bacterial colonization of atopic skin, so this study was found necessary to determine the bacterial types and numbers of each eczematous lesions and healthy areas from patients skin with atopic dermatitis.

MATERIALS & METHODS

PATIENTS

A total of (286) patients suffering from atopic dermatitis syndrome in various age groups and both sexes were included in this study. The patients attending the out patients of dermatology department of fourth main hospitals in Basrah providence (out patients based study), and AD diagnosed under supervision of dermatologists based on a criteria of (8,9,10). The study was carried out during period from November 2003 to July 2005.

PRIMARY ISOLATION

Brain Heart Infusion Broth (BHIB) saturated skin swabs from both eczematous lesions and healthy area were cultured on primary isolation media: Blood Agar Base BAB (Oxoid), MacConkey Agar MA and Nutrient Agar NA (Himedia) and then incubated at 37°C for 24-48 hrs aerobically⁽¹¹⁾. Samples that cultured on Chocolate Agar (CA) in addition to NA were incubated under CO_2 in candle jar at the same

temperature and period time mentioned above(11).

BACTERIAL COUNT

Enumeration of a total bacterial counts per cm² of the same above sites were carried by using a serial dilution technique of Nutrient Broth (HiMedia) and then culturing on NA(11).

IDENTIFICATION TECHNIQUES

Four types of Api-technique (bioMerieux, France) as a rapid identification system were used for identification the various bacterial isolates based on enclosed instructions of supplied company:

- Api Staph: identification system for staphylococci.
- Api 20 Strept: identification system for streptococci.
- Api 20 E: identification system for Enterobacteriaceae and other Gram-negative rods.
- And Api 20 A: identification system for anaerobes.

STATISTICAL ANALYSIS

Chi-Square test and ANOVA test were carried by using computer program , SPSS . ver.11.

RESULTS

Table (1) illustrate all bacterial types isolated from eczematous lesions and nearly healthy areas. The total numbers of positive cultures (270,247) cases from 286 studied AD cases in percentages (94.4, 86.36) from eczematous and healthy area respectively. In general, twenty bacterial types were isolated from both area separately and (959,744) isolates with isolation ratio (3.35:1, 2.6:1) isolates: case were identified in each above area respectively. The percentages of bacterial occurrence in eczematous lesions and healthy areas respectively were as follow: (P< 0.05) : Staph. aureus (60.48, 17.48), Staph. epidermidis (17.13, 57.34), Staph. xylosus (2.79) in each, Staph. saprophyticus (5.24, 10.48), Staph. capitis (2.79) in each, Staph. hominis (22.37, 9.44), Strept.pyogenes (17.13, 9.79), Strept. Faecalis (23.07, 17.83), Strept. Mutans (14.68, 9.44), E.coli (25.52, 33.21) Enterobacter sp. (5.59, 17.83), Klebsiella sp. (3.14, 1.39), Acinetobacter sp. (5.59, 3.49), Proteus sp. (5.94) in each, Pseudomonas aeruginosa (17.48, 5.59), Propionibacterium acnes (19.58, 3.49%), Pr.granulosum (20.27, 18.53), Haemophilus influenzae (21.32, 11.53), Bacteroid sp. (18.18, 3.84), and Corynebacterium sp. (26.92, 17.83).

Table (2) illustrated modes of isolation of bacterial types from both areas of AD patients. It has been found that the double bacterial agents was predominant in each eczematous

lesions and healthy area in percentages (44.11, 41.29) respectively followed by other modes, with significant differences between modes of isolation (P< 0.05).

Figure (1): showed distribution of bacterial types in eczematous lesions and healthy areas of skin of AD patients as previous described.

The means of bacterial number measured for studied areas of AD patients showed in figure (2). The bacterial numbers ranged from (0.02-92.0) X 10⁵ cell (CFU)/cm² in eczematous lesions and (0.11-23.0) X 10³ cell (CFU)/cm² in healthy areas with very highly significant differences between means of bacterial numbers within the same areas and between each studied area (P< 0.001).

Figure 1

Table 1: Illustrate bacterial types isolated from eczematous lesions and healthy areas of AD patients (P< 0.05)

| Bacterial types | No. of cases (%) from eczematous lesion | No. of cases (%) from healthy area |
|--------------------------------|---|------------------------------------|
| <i>Staph. aureus</i> | 173(60.48) | 50 (17.48) |
| <i>Staph. epidermidis</i> | 49 (17.13) | 164 (57.34) |
| <i>Staph. xylosus</i> | 8 (2.79) | 8 (2.79) |
| <i>Staph. saprophyticus</i> | 15 (5.24) | 30 (10.48) |
| <i>Staph. capitis</i> | 8 (2.79) | 8 (2.79) |
| <i>Staph. hominis</i> | 64 (22.37) | 27 (9.44) |
| <i>Strept.pyogenes</i> | 49 (17.13) | 28 (9.79) |
| <i>Strept. Faecalis</i> | 66 (23.07) | 51 (17.83) |
| <i>Strept. Mutans</i> | 42 (14.68) | 27 (9.44) |
| <i>E.coli</i> | 73 (25.52) | 95 (33.21) |
| <i>Enterobacter sp.</i> | 16 (5.59) | 51 (17.83) |
| <i>Klebsiella sp</i> | 9 (3.14) | 4 (1.39) |
| <i>Acinetobacter sp.</i> | 16 (5.59) | 10 (3.49) |
| <i>Proteus sp.</i> | 17 (5.94) | 17 (5.94) |
| <i>Pseudomonas aeruginosa</i> | 50 (17.48) | 16 (5.59) |
| <i>Propionibacterium acnes</i> | 56 (19.58) | 10 (3.49) |
| <i>Pr.granulosum</i> | 58(20.27) | 53(18.53) |
| <i>Haemophilus influenzae</i> | 61 (21.32) | 33 (11.53) |
| <i>Bacteroid sp.</i> | 52 (18.18) | 11 (3.84) |
| <i>Corynebacterium sp.</i> | 77 (26.92) | 51 (17.83) |
| No. of isolates | 959 | 744 |
| Average (isolate: case) | 3.35:1 | 2.6:1 |
| No. of -ve growth cultures | 16 (5.59) | 39 (13.63) |
| No. of +ve growth cultures | 270 (94.4) | 247 (86.36) |
| Total no. of cultures | 286 | |

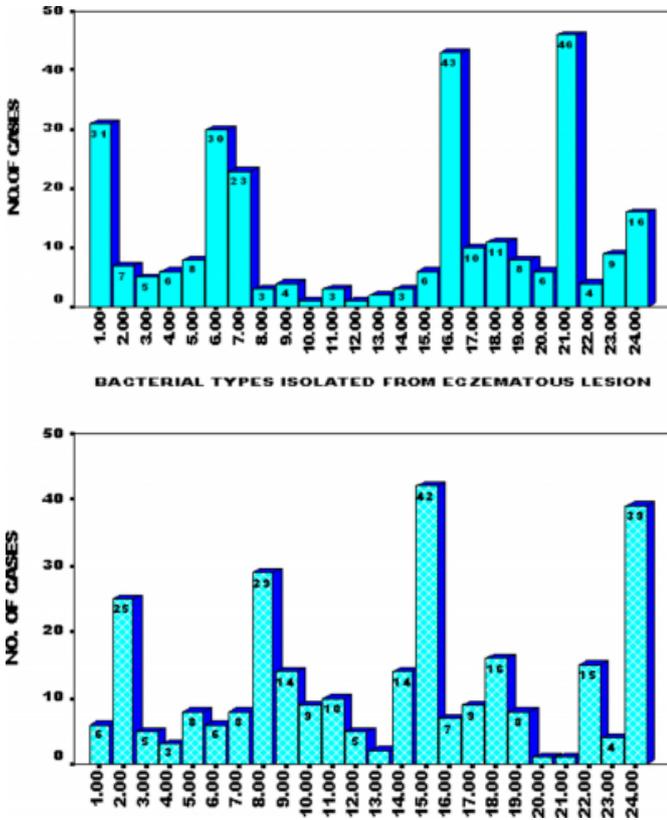
Figure 2

Table 2: Illustrate modes of isolation of bacterial types from eczematous lesions and healthy areas of AD patients (P< 0.05)

| Mode of isolation | No. of cases (%) from eczematous lesions | No. of cases (%) from healthy areas |
|---|--|-------------------------------------|
| Single | 38 (14.07) | 31 (12.55) |
| Double | 75 (44.11) | 102(41.29) |
| Third | 22 (12.94) | 19 (7.69) |
| Fourth | 19 (11.17) | 61 (24.69) |
| Fifth | 59 (34.7) | 17 (6.88) |
| Sixth and over | 57 (33.52) | 17 (6.88) |
| No. of + ve growth culture (from total No. of modes) | 270 (94.4) | 247 (86.36) |
| No. of - ve growth culture | 16 (5.59) | 39 (13.63) |
| Total no. of cases | 286 | 286 |

Figure 3

Figure 1: illustrated isolation modes of bacterial types from eczematous lesions (above) & healthy area (below) of ad patients skin . (p < 0.001)

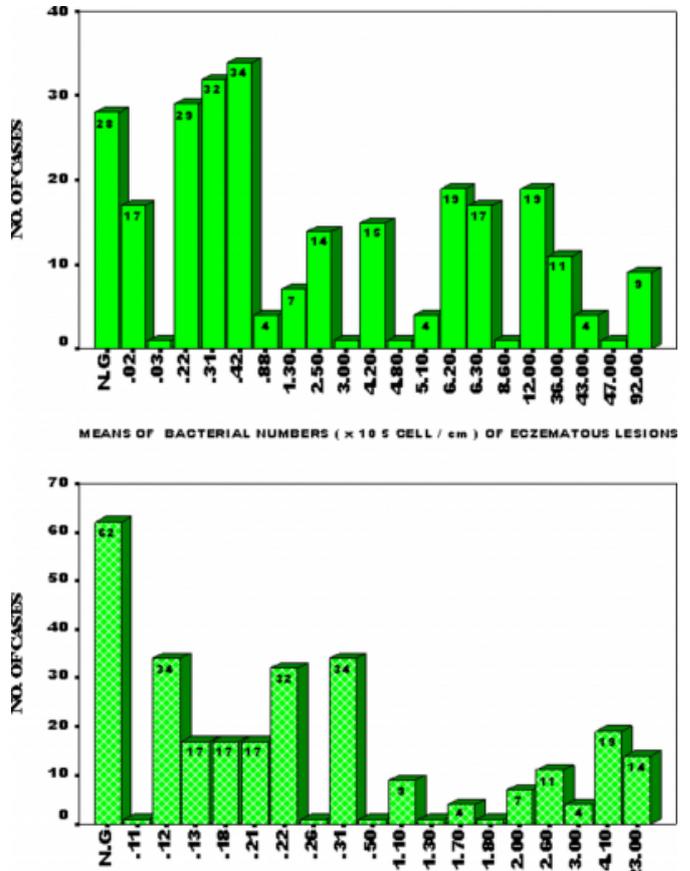


BACTERIAL TYPES : 1 : STAPH AUREUS , 2 : STAPH EPIDERMIDIS , 3: STAPH XYLOSUS , 4 : STAPH , 5 : STAPH CAPITIS , 6 : STAPH HOMINIS , 7 : STREPT PYOGENES , 8 : STREPT FAECALIS , 9 : STREPT MUTANS , 10: E.COLI , 11 : ENTEROBACTER SP. , 12 : KLEBSIELLA SP. , 13 : ACINETOBACTER SP. , 14: PROTEUS SP. , 15: PS AERUGINOSA , 16: PR ACNES , 17: PR GRANULOSUM , 18: H INFLUENZAE , 19: BACTEROID SP. , 20 : CORYNEBACTERIUM SP.

ISOLATION MODES : (AS SHOW IN FIGURES)
 DEPEND ON ABOVE NUMBERS- 1 : 1 , 2 : 2 , 3 : 1&2 , 4 : 1&2&7 , 5 : 1&2&8 , 6 : 1&7 , 7 : 1&9 , 8 : 2&8 , 9 : 2&4 , 10 : 2&6 , 11 : 2&7 , 12 : 2&8 , 13 : 2&9 , 14 : 2&10 , 15 : 2&10&11&17 , 16 : 1&10&17&19&20 , 17 : 7&11&13&16&20 , 18 : 4&6&9&10&14&18&20 , 19 : 3&5&8 , 20 : 6&9&10&13&14 , 21 : 1&6&8&15&16&18 , 22 : 10&15&18&20 , 23 : 12&17&19&20 , 24 : NO GROWTH .

Figure 4

Figure 2: Illustrated means of bacterial numbers measured for eczematous lesion and healthy area of ad patients . (p < 0.001)



DISCUSSION

Our results revealed that Staph. aureus was the predominant bacterial agent isolated from (60.48%) of eczematous lesion, while Staph. epidermidis was predominantly from (57.34%) of healthy area of AD patients. And we evidenced highly prevalence /or occurrence of bacterial types in each studied areas.

Our knowledge concerning the complex interaction between microbes ad skin inflammation of atopic dermatitis has improved dramatically and today the Gram-positive bacterium, Staph. aureus is recognized as an important triggering factors for the maintenance of skin inflammation and acute exacerbations of the genetically determined skin disease atopic dermatitis(12,13).

Recent studies confirmed results of our study such as Brook, et al. (1996) that found 36%, 20%, and 44% of aerobic, an aerobic, and mixed respectively isolated from AD patients, these bacteria were Staph aureus, group A-streptococci, E.coli, Peptostreptococcus, Prevotella, Porphyromonas sp.

And *Fusobacterium*(₁₄).

Others demonstrated that the colonization density of eczematous lesions can reach 10^7 CFU/cm²(₁₅). And the skin of 100% of AD patients may be colonized with *Staph. aureus*, up to 65% of all *Staph. aureus* strains isolated from lesional skin have been shown to produce exotoxins with superantigenic properties(₁₆).

Also Heaton, et al. (2003) illustrated that the incidence of *Staph. aureus* colonization on the skin of AD patient is approximately 90%(₁₇).

Many modern studies illustrate the factors whose relevance to the increased colonization of atopic dermatitis skin with bacteria -in general- and with *Staph. aureus* -in especially- such as production of exotoxins(_{4,18}), ability of bacterial types to adhere with host cells of atopic skin(₁₉), pH values were shifting toward alkalinity with adherence of *Staph. aureus* to human keratinocytes being highest at pH= 7-8(₂₀). And extracellular lipids of stratum corneum of epidermal layer of skin, the quantitative and qualitative changes in lipid composition could result in diminished antibacterial activity(₂₁).

CONCLUSION

There is a high occurrence and variation of bacterial types in eczematous lesions and healthy areas of skin of AD patients, with high numbers of bacteria in the first above area in comparison with the second that indicate presence of highly infections in the lesional skin. Also *Staph. aureus* was the predominant bacterial types in eczematous lesions of AD skin.

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References

1. Habif, T.P. Clinical Dermatology: A color guide to diagnosis and therapy. 4th ed. Ch 5: Atopic dermatitis. Mosby co. London. 2004. pp:105-128.
2. Jordaan, H.F. Atopic dermatitis. available from ; <http://www.xtraMSNHealth.Net.2005>.
3. Stanway, A. Atopic dermatitis. available from ; <http://www.DermNetNZ.bookstore.Net.2005>.
4. Leung, D.Y.M. Role of *Staphylococcus aureus* in atopic dermatitis. In: Bieber, T. and Leung, D.Y.M. Atopic dermatitis. Marcel Dekker, Inc. New York. 2002. pp:401-418.
5. Hauser, C., Prins, C. and Lacour, M. The role of infectious agents in atopic dermatitis. in: Leung, D.Y.M. atopic dermatitis: from pathogenesis to treatment. Springer Verlag, New York. 1996. pp:67-112.
6. Hauser, C., Wuetrich, B., Matter, L. *Staphylococcus aureus* skin colonization in atopic dermatitis patients. *Dermatologica*, 1985. 170: 35-39.
7. Gloor, M., Peters, G. and Stoika, D. On the resident aerobic bacterial skin flora in unaffected skin of patients with atopic dermatitis and in healthy controls. *Dermaologica*, 1982., 164: 258-265.
8. Hanifin, J.M. and Rajka, G. Diagnostic features of atopic dermatitis. *Acta. Derm. Venereol. (Stockh)*. 1980., 92(suppl.):44-47.
9. Spergel, J.M. and Schneider, L.C. Atopic dermatitis. *Inter. J. Asthma Allergy Immunol.* 1999., 1(1):1-16.
10. Stanway, A. Atopic dermatitis. Available from ; <http://www.DermNetNZ.bookstore.Net.2005>.
11. Forbes, B.A., Sahm, D.F. and Weissfeld, A.S. Baily & Scotts Diagnostic Microbiology. 10th ed. Vol.1, Mosby Co., St. Louis, 1998., pp: 68-280, 282-418, 606-618.
12. Leyden, J.L., Marples, R. and Kligman, A.M. *Staphylococcus aureus* in the lesions of atopic dermatitis. *Br. J. Dermatol.*, 1993., 90: 525-530.
13. Strange, P., Skov, L., Lisby, S. Staphylococcal enterotoxin B applied on intact normal and intact atopic skin induces dermatitis. *Arch. Dermatol.*, 1996., 132:27-33.
14. Brook, I., Frazee, E.H. and Yeager, J.K. Microbiology of infected atopic dermatitis. *Int J. Dermatol.*, 1996.35(11): 791-793.
15. Breuer, K., Wittmann, M., Bosche, B., Kapp, A. and Werfel, T. Severe atopic dermatitis is associated with sensitization to staphylococcal enterotoxin B (SEB). *Allergy*, 2000., 55(6):551-555.
16. Breuer, K., Haussler, S., Kapp, A., and Werfel, T. *Staphylococcus aureus*: colonizing features and influence of an antibacterial treatment in adult with atopic dermatitis. *Br. J. Dermatol.*, 2002., 147(1):55.
17. Heaton, T., Mallan, D., Venaille, T. and Holt, P. Staphylococcal enterotoxin induced IL-8 stimulation as a cofactor in the pathogenesis of atopic disease: the hygiene hypothesis in reverse? . *Allergy*, 2003., 58(3):252-256.
18. Ramirez, H.M., Kang, K., Stevens, S.R. and Cooper, K.D. Cellular aspects of atopic dermatitis: overview. In: Bieber, J., and Leung, D.Y.M. Atopic dermatitis . Marcel Dekker, Inc. New York, ch. 1, 2002., pp:217-230.
19. Abeck, D. and Mempel, M. *Staphylococcus aureus* colonization in atopic dermatitis and its therapeutic implication. *Br.J. Dermatol.*, 1998., 139:13-16.
20. Seidenari, S.T. and Giusti, G. Objective assessment of the skin of children affected by atopic dermatitis: a study of pH, capacitance and TEWL in eczematous and clinically uninvolved skin. *Acta. Derm. Venereol. (Stockh)*, 1995., 75: 429-433.
21. Ohnishi, Y., Okino, N., Ito, M. and Imayama, S. Ceramidase activity in bacterial skin flora as a possible cause of ceramide deficiency in atopic dermatitis. *Clin. Diag. Lab. Immunol.*, 1999., 6(1): 101-104.

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