Detection of Secondary Bacterial Contaminants in Patients with Eczema

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Abstract:

Background: Eczema is an inflammatory skin reaction in which a lot of external and internal factors acting singly or in combination to induce the condition. Eczema leads to damage of the normal defense mechanisms of the skin. Therefore, eczematous skin becomes favorable site for bacterial contamination.

Objectives: The study aims at identifying the types of bacteria that commonly infect eczematous skin. It is also aimed at assessment of age factor and its relation to the type of bacteria that commonly infect various eczematous skins. Thirdly, it is aimed at identifying the bacterial sensitivity to antibiotics to determine the appropriate treatment.

Patients & methods: The study subjects were patients suffering from various types of eczema involved in cross-sectional study. Eighty-eight patients were collected. The patients were attending the dermatology clinics at Al-Yarmook Teaching Hospital and Al-Kindy Teaching Hospital. The study period was from September 2001 till February 2002. Identification of bacterial isolates is performed by 3 ways: morphological identification, biochemical tests and Api tests. Bacterial sensitivity to antibiotics was performed by Kirby-Bauer diffusion method. Statistical analysis was performed by utilizing Pearson Chi-Square test.

Results: One hundred sixty bacterial specimens were taken from the study subjects. Statistical analysis proved a significant association between the types of eczema and the types of bacterial isolates (Pearson $\chi^2 = 38.728$, df = 18, P value = 0.003).Staphylococcus aureus was mainly sensitive to neomycin (100%). Staphylococcus epidermidis was mainly sensitive to neomycin (94.9%) and vancomycin (100%). Escherichia coli was mainly sensitive to streptomycin (86.7%) and amikacin (86.7%). Klebsiella pneumoniae was mainly sensitive to neomycin (83.3%) and amikacin (86.7%). Enterobactor cloacae was mainly sensitive to amikacin (100%). Proteus mirabilis was mainly sensitive to neomycin (100%) and amikacin (100%). Pseudomonas aeruginosa was mainly sensitive to neomycin (85%), streptomycin (85%) and amikacin (85%). All bacterial types were highly resistant to penicillin G, ampicillin, amoxicillin and cloxacillin.

Conclusions: Gram-positive bacteria especially staphylococcus aureus and staphylococcus epidermidis commonly infect eczematous skin more than Gram-negative bacteria. Staphylococcus aureus is a common invader of atopic eczema and discoid eczema, this phenomenon stresses its pathogenic role in these 2 types of eczema and to a lesser extent in all damaged eczematous skin. All bacterial types have shown high resistance to penicillins whilst they are vulnerable to aminoglycosides especially neomycin and amikacin.

Key words: Eczema, Bacteria, Contaminants.

Introduction

Eczema is an inflammatory skin reaction characterized histologically by spongiosis with varying degrees of acanthosis, and a superficial perivascular lymphohistiocytic infiltrate. The clinical features of eczema include itching, redness, scaling, and clustered papulovesicles. A wide range of external and internal factors acting singly or in combination can induce the condition.[1]

An intact stratum corneum and its continuous desquamation prevents invasion of the skin by normal skin flora or pathogenic microorganisms so acts as mechanical barrier.[2] The skin also has chemical barriers in which sebaceous lipids have been reported to possess antibacterial properties.[3] Glycophospholipids and free fatty acids of the stratum corneum have bacteriostatic effects selective for pathogenic microorganisms.[4] Antimicrobial peptides (AMPs) are a diverse group of peptides that are present on the epidermis and its appendages is the first line of immune defense.[5,6] AMPs directly kill a broad spectrum of microbes, including Gram-positive and Gram-negative bacteria, fungi and certain viruses.

However, skin diseases like eczema can disrupt the above mentioned defense mechanisms and provide portals of entry to microorganisms particularly staphylococci or streptococci. This may lead to serious local and systemic complications as a result of skin and soft-tissue infections.[2]

The study is aimed at identifying the types of bacteria that commonly infect eczematous skin. Secondly, it is aimed at assessment of age factor and its relation to the type of bacteria that commonly infect various eczematous skins. Thirdly, it is aimed at identifying the bacterial sensitivity to antibiotics to determine the appropriate treatment.

Patients & Methods

Eighty-eight study subjects were involved in a cross-sectional study. The study subjects were patients suffering from various types of eczema and collected from the dermatology clinics at Al-YarmookTeaching Hospital and Al-Kindy Teaching Hospital. The study period was from September 2001 till February 2002.

Sterile swabs are used to swab the eczematous skin. The swabs are put in Nutrient broth. Cultures are used to identify the types of bacteria. Bacterial cultures are performed on Nutrient Agar and MacConkey Agar. The cultures are incubated for 24-48 hours in 37 C˚.

Identification of bacterial isolates is performed by 3 ways. Morphological identification,

biochemical tests and Api tests. Bacterial sensitivity to antibiotics is performed by standard Kirby-Bauer diffusion method.

Statistical analysis is performed by utilizing Pearson Chi-Square test. A statistical result with P values of less than 0.05 is considered significant.

Results

Eighty-eight patients with various types of eczema were involved in the study (mean age= 30.7±17.7 years). The types of eczema are contact dermatitis (39 patients comprising 44.3%), atopic dermatitis (18 patients comprising 20.5%), discoid eczema (20 patients comprising 22.7%) and asteatotic eczema (11 patients comprising 12.5%). One hundred sixty bacterial specimens are taken from the study subjects (where in some cases more than one bacterial specimen is taken).

Table I shows the association between the types of eczema and the types of bacterial isolates.

Statistical analysis proved a significant association (Pearson $\chi^2 = 38.728$, df = 18, P value = 0.003).

Table I: The distribution of the 160 bacterial isolates among different types of eczema.

Type of bacterial isolate	Type of Eczem*a*							
	Contact dermatitis		Atopic dermatitis		Discoid eczema		Asteatotic eczema	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Staphylococcus aureus	8	13.3%	17	41.5%	13	32.5%	2	10.5%
Staphylococcus epidermidis	21	35%	5	12.2%	6	15%	7	36.8%
Escherichia coli	6	10%	2	4.9%	3	7.5%	4	21.1%
Klebsiella pneumoniae	15	25%	6	14.6%	6	15%	3	15.8%
Enterobactor cloacae	2	3.3%	5	12.2%	2	5%	0	
Proteus mirabilis	0		2	4.9%	5	12.5%	0	
Pseudomonas aeruginosa	8	13.3%	4	9.8%	5	12.5%	3	15.8%
Total	60	100%	41	100%	40	100%	19	100%
Pearson $\chi^2 = 38.728$, df = 18, P value = 0.003								

The distribution of bacterial isolates according to age groups for all types of eczema is shown in table II. Examination of the associations between the types of bacterial isolates and age groups did not show any significant associations for all types of eczema

Table III shows the detailed bacterial sensitivity to antibiotics and their related statistical significance. Staphylococcus aureus was mainly sensitive to neomycin (100%). Staphylococcus epidermidis was mainly sensitive to neomycin (94.9%) and vancomycin (100%). Escherichia coli was mainly sensitive to streptomycin (86.7%) and amikacin (86.7%). Klebsiella pneumoniae was mainly sensitive to neomycin (83.3%) and amikacin (86.7%). Enterobactor cloacae was mainly sensitive to amikacin (100%). Proteus mirabilis was mainly sensitive to neomycin (100%) and amikacin (100%). Pseudomonas aeruginosa was mainly sensitive to neomycin (85%), streptomycin (85%) and amikacin

Table II. The distribution of 160 bacterial isolates according to age categories for patients with eczema

Age category	Type of bacterial isolate No(%)						
	Staphylococcus aureus	**Staphylococcus epidermidis**	**Escherichia coli**	**Klebsiella pneumoniae**	**Enterobactor cloacae**	**Proteus mirabilis**	**Pseudomonas aeruginosa**
< 10 years	9 22.5%	4 10.3%	1 6.7%	5 16.7%	3 33.3%	0	5 25%
10-19 years	8 20%	4 10.3%	4 26.7%	4 13.3%	3 33.3%	2 28.6%	1 5%
20-29 years	6 15%	6 15.4%	1 6.7%	4 13.3%	2 22.2%	1 14.3%	4 20%
30-39 years	5 12.5%	4 10.3%	1 6.7%	7 23.3%	0	2 28.6%	2 10%
40-49 years	9 22.5%	14 35.9%	4 26.7%	7 23.3%	1 11.1%	1 14.3%	5 25%
>50 years	3 7.5%	7 17.9%	4 26.7%	3 10%	0	1 14.3%	3 15%
Total	40 100%	39 100%	15 100%	30 100%	9 100%	7 100%	20 100%
Pearson Chi-Square = 27.55, df = 30, P value = 0.954							

Table III: The bacterial sensitivity to antibiotics for different bacterial isolates and their related statistical significance.

Isolate Type (No.)	Antibiotics sensitivity No.(%)										Total sensitive isolates (%)
	Penicillin G	Ampicillin	Amoxicillin	Cloxacillin	Neomycin	Gentamycin	Streptomycin	Amikacin	Tetracycline	Vancomycin	
Staphylococcus aureus (40)	4 (10%)	5 (12.5%)	14 (35%)	4 (10%)	40 (100%)	28 (70%)	33 (82.5%)	35 (87.5%)	34 (85%)	35 (87.5%)	232 (58%)
Staphylococcus epidermidis (39)	7 (17.9%)	6 (15.4%)	9 (23.1%)	2 (5.1%)	37 94.9%)	30 (76.9%)	34 (87.2%)	36 (92.3%)	32 (82.1%)	39 (100%)	232 (59.5%)
Escherichia Coli (15)	2 (13.3%)	4 (26.7%)	7 (46.7%)	2 (13.3%)	5 (33.3%)	10 (66.7%)	13 (86.7%)	13 (86.7%)	10 (66.7%)	2 (13.3%)	68 (45.3%)
Klebsiella pneumoniae (30)	2 (6.7%)	1 (3.3%)	4 (13.3%)	0	25 83.3%)	22 (73.3%)	18 (60%)	26 (86.7%)	22 (73.3%)	1 (3.3%)	121 (40.3%)
Enterobactor cloacae (9)	2 (22.2%)	3 (33.3%)	2 (22.2%)	1 (11.1%)	5 (55.6%)	6 (66.7%)	5 (55.6%)	9 (100%)	7 (77.8%)	2 (22.2%)	42 (46.7%)
Proteus Mirabilis (7)	0	2 (28.6%)	2 (28.6%)	0	7 (100%)	5 (71.4%)	3 (42.9%)	7 (100%)	5 (71.4%)	0	31 (44.3%)
Pseudomonas aeruginosa (20)	1 (5%)	6 (30%)	11 (55%)	10 (50%)	17 (85%)	15 (75%)	17 (85%)	17 (85%)	15 (75%)	0	109 (54.5%)

A…(Pearson Chi-Square = 200.082, df = 9, P value = 0.0007). B (Pearson Chi-Square = 218.492, df = 9, P value = 0.0007).
C…(Pearson Chi-Square = 47.776, df = 9, P value = 0.0009). D (Pearson Chi-Square = 159.412, df = 9, P value = 0.0006).
E…(Pearson Chi-Square = 27.5, df = 9, P value = 0.001). F (Pearson Chi-Square = 39.892, df = 9, P value = 0.0005).
G…(Pearson Chi-Square = 78.012, df = 9, P value = 0.0003).

Discussion

This study highlights the diversity of the microbiology of secondarily infected eczematous skin lesions. The isolation of multiple organisms from more than half of the patients illustrate the polymicrobial nature of secondarily infected skin lesions and the potential for bacterial synergy between the different microbial isolates. Several studies have documented the synergic effect of mixtures of bacteria in experimental infections.[7,8] Various hypotheses have been proposed to explain such microbial synergy. It may be the result of protection from phagocytosis and intracellular killing,[9] production of essential growth factors[10] or lowering of oxidation-reduction potentials in host tissue.[11]

Organisms that reside in the mucous membranes close to the lesions predominated in infections next to these membranes. In our fashion, enteric Gram-negative bacilli were found most often in the buttock and leg lesions. The probable sources of these organisms are the rectum and vagina, where they normally reside.[12]

The exact pathogenic role of all organisms isolated from secondarily infected eczematous skin lesions has not yet been determined. The isolation of different types of organisms from infected eczematous skin lesions raises questions about their pathogenic role. However, these organisms are known pathogens in certain types of skin and soft tissue infections. These include diabetic foot infection,[13] decubitus ulcers[14] and bite wound infections.[15] Therefore, it is possible that they may have a pathogenic role in infected eczematous skin lesions.

As with this study, staphylococcus aureus commonly colonizes eczematous lesions of children and adults with atopic dermatitis, whereas it is found on skin of healthy subjects in minority of cases.[16] More specifically, staphylococcus aureus has a major role in the pathogenesis of atopic dermatitis. It has been found that some staphylococcal exotoxins are superantigens,[17] which activate a greater number of lymphocytes than those stimulated by specific antigen, resulting in a superstimulation. One manifestation is an excessive synthesis of IgE.[18] Therefore, this organism may induce purulent superinfection as well as enhance the inflammatory process by this superantigen-mediated T-cell activation.[19]

In discoid eczema, the role of bacteria has been stressed in its pathogenesis. As in other forms of eczema, heavy colonization of the lesions by staphylococci may increase their severity, even in the absence of clinical evidence of infection.[20,21] However, allergic sensitivity to staphylococci or micrococci may be responsible at least for secondary dissemination.[22]

Staphylococcus aureus is isolated from many skin lesions. It seems that colonization with staphylococcus aureus is not a characteristic feature for atopic dermatitis but is a frequent event in damaged skin;[23] significantly elevated values were also observed in non-atopic eczema. The degree of colonization may depend on the severity and duration of the eczematous lesions.[24]

The effect of local steroid therapy on the initiation and progression of secondary bacterial infection in many secondarily infected eczematous skin lesions is uncertain. Steroids may mask inflammation, and may suppress some of the immune response.[25]

Although local application of specific corticosteroid therapies is the mainstay of treatment of eczema, management of secondary bacterial infections is essential. Local application of antibacterial agents is important component of the treatment of these complications. Treatment of widespread skin infection should include systemic antimicrobial therapy. This could be important in the event of poor response to corticosteroid therapy or spread of the infection.[26]

The data presented demonstrate the presence of β-lactamase-producing organisms in many secondarily infected eczematous skin lesions. These organisms not only survive penicillin therapy but also can protect susceptible bacteria from penicillins by releasing the enzyme into the adjacent tissues.[27]

The improper and non-rational use of penicillins led to the emergence of resistant bacterial strains.[28] Generally, penicillins have a weak effect against staphylococcus species.[29] Staphylococcus species have the capability to produce beta-lactamase enzyme which destroy the penicillins extracellularly.[30] They also can obtain resistance by modifying their penicillin binding protein (PBP) targets.[31] Gram-negative bacteria have the ability to alter their cellular permeability to penicillins or to produce beta-lactamase enzyme to destroy penicillins intracellularly by these ways they can obtain resistance.[32]

Generally, aminoglycosides (like neomycin, gentamycin, streptomycin and amikacin in this study) are used against Gram-negative bacteria. Only some Gram-positive bacteria and a minority of Gram-negative bacteria can resist aminoglycosides by producing aminoglycoside transferase enzyme.[33] Tetracycline is bacteriostatic against Gram-positive and Gram-negative bacteria. Although bacterial resistance is less than previously thought, their use is limited in dermatology as much more effective agents are available.[34] Resistance to tetracycline is mediated by bacterial plasmid.[35] Vancomycin is

useless against Gram-negative bacteria whilst it is highly effective against Gram-positive bacteria specially staphylococcus species.[36]Age was not a significant factor statistically and clinically in the colonization of bacteria among various types of eczema. For instance, a clinical study demonstrated such non-significance as the prevalence of staphylococcal colonization in AD children is comparable to that in adults.[37

In conclusion, Gram-positive bacteria especially staphylococcus aureus and staphylococcus epidermidis commonly infect eczematous skin more than Gram-negative bacteria. Staphylococcus aureus is a common invader of atopic eczema and discoid eczema, this phenomenon stresses its pathogenic role in these 2 types of eczema and to a lesser extent in all damaged eczematous skin. All bacterial types have shown high resistance to penicillins whilst they are vulnerable to aminoglycosides especially neomycin and amikacin.

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