Staphylococcus Aureus Carriage in Patients with Psoriasis

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Abstract

Background: The aggravating role of Staphylococcus aureus is well known in atopic dermatitis but has not yet been proven in psoriasis. The role of Staphylococcus aureus superantigens is emphasized in the initiation, maintenance and complications of psoriasis. We investigated the frequency of nasal, axillary, and perineal carriage of Staphylococcus aureus (SA) in patients with psoriasis and its possible influence on the severity of the disease.

Methods: one hundred patients with the clinical diagnosis of psoriasis participated in the study. Cultures of the bacterial flora were obtained from the right and left axilla and nasal nares and perineum, inoculated on standard bacterial medium (blood agar), and incubated at 37°C degrees for 48 h.

Results: one hundred patients with the clinical diagnosis of psoriasis (42% female and 58% male) comprised the study group. Mean age of the patients was 41.1±17.1 years. About 42 % of the patients carried *S. aureus*; of these, 32 % were from the nose, 13 % from axilla, and 11% from the perineum. Three patients were carriers in all 3 sites. There was no significant difference in the severity of the disease between the carriers and non-carriers measured by the psoriasis area and severity index (PASI) score.

Conclusions: According to our findings, S.aureus carriage in psoriasis had no significant influence on disease severity. It might be relevant for a subgroup of patients only when superantigen productions are found. *(Iran J Dermatol 2009;12: 1-3)*

Keywords: psoriasis, staphylococcus aureus, carrier, colonization

Introduction

Psoriasis is a chronic inflammatory skin disorder affecting 1-2% of the general population. The characteristic lesion of psoriasis is a sharply demarcated erythematous papule or plaque containing hyperproliferating keratinocytes as well as infiltrating neutrophils, monocytes, and T lymphocytes.[1] Although psoriasis is considered to be an autoimmune disease, increasing evidence suggests an important role for bacteria in its initiation and/or propagation.

Psoriasis can be provoked or exacerbated by a variety of different environmental factors, particularly infections and drugs. Strong evidence exists for the induction of guttate psoriasis by a preceding tonsillar Streptococcus pyogenes infection, and disease exacerbation has been linked with skin and/or gut colonization by Staphylococcus aureus, Malassezia, and Candida albicans.[2] Colonization of lesional psoriatic skin (and nares) by S. aureus has been demonstrated in approximately 60% of patients with psoriasis compared with 5% to 30% of individuals with a normal healthy skin.[3, 4] Isolation of S.aureus from the throat of 11 out of 22 patients with psoriasis has also been reported.[5] Furthermore, T cells specific for peptidoglycan from S.aureus have been cultured from psoriatic skin lesions. Macrophages carrying S. aureus-specific peptidoglycan were few in these lesions compared with the total number of peptidoglycan-containing macrophages detected; suggesting that specific activation of S. aureus peptidoglycan-specific T cells may not be involved in disease pathogenesis.[6]

In this study, we assessed the prevalence of S. aureus colonization in multiple body regions in patients with psoriasis and evaluated the correlation between S. aureus colonization and severity of the disease.

Patients and Methods

This cross-sectional study was conducted from June 2006 to December 2007. Psoriasis patients were selected randomly from all the cases who referred to our university dermatology clinic with no history of antibiotic administration in the past 2 weeks. These patients underwent a thorough clinical examination and the psoriasis area and severity index (PASI) was measured by a qualified inspector.[7] These patients were referred to the laboratory and the specimens were taken with a sterile cotton swab form nasal cavities, axilla and perineum of both sides of the body by a qualified laboratory technician. These specimens were immediately transferred to a sheep blood agar and were incubated at 37°C for 48 hours. The cultured colonies underwent coagulase test with rabbit plasma, manitol fermentation and DNase test. This study was designed as a quality-based assay of the S.aureus colonization; therefore, the specimens with positive results were diagnosed as staphylococcus aureus.

All patients were instructed about the study, and they signed informed consent forms. The study was performed according to the Declaration of Helsinki and the ethics committee of our university approved the study protocol. Collected data was analyzed with SPSS-11.5 software using independent t-test, Chi-square and Fischer's exact test. P-value less than 0.05 was considered significant.

Results

The study group consisted of one hundred patients with the clinical diagnosis of psoriasis (42% female and 58% male). Mean age of the patients was 41.1 ± 17.1 years. Forty two percent of the patients carried S.aureus at least in one site; 32 % in nose, 13 % in axilla, and 11% in perineum (Table 1). Six (6%) patients had positive cultures of the nose and perineum, six (6%) had positive cultures of the nose and axilla and 5 (5%) had positive cultures of axilla and perineum. Three patients were carriers of S.aureus in all 3 sites (i.e. nasal cavity, axilla and perineum). Mean PASI score was 21.4 ± 11.9 in carriers and 24 ± 11.2 in non-carriers which showed no significant difference (P>0.05). The mean period of psoriasis in carriers and non-carriers was 103.6 ± 100.5 and 113± 104 months, respectively, with no significant difference (P>0.05). We compared carriers with non-carrier patients regarding associated diseases including atopic state, sinusitis and furunculosis (Table 2). However, no significant difference was seen between carriers and non-carriers in this regard (P>0.05).

Table 1: *The site of staphylococcus colonization in carrier patients with psoriasis*

	Frequency	Relatie frequency
Right nose	30	30%
Left nose	29	29%
Right axilla	8	8%
Left axilla	9	9%
Perineum	11	11%

Table 2: *Associated conditions in carrier and non-carrier patients with psoriasis*

	Staph.aureus Carrier (42 cases)	Staph.aureus non-carrier (58 cases)
Atopic state	13(31%)	18(31%)
Sinusitis	12(28%)	17(29%)
Frunclosis	5 (12%)	6(10%)

Discussion

Bacteria can lead to recognized clinical conditions of the skin that are nowadays easily controlled using proper antibiotic therapy. It is likely that superantigens and proteolytic toxins can protract cutaneous inflammation via direct physical rupture of the normal skin barrier and via direct stimulation of activated skin-homing T lymphocytes. Although superantigens are likely to be one of the factors that can control disease development, they might be of primary importance only in few conditions.[8] Superantigen toxin could play a main role in initiation or exacerbation of some cutaneous diseases such as psoriasis. In a study by Tomi, et al., 36% of cultured species from psoriatic patients was toxinergic.[4] PASI score was significantly higher in psoriatic carriers of toxinergic S.aureus. This study suggested that bacterial toxins played a role in the exacerbation of psoriasis.[9] Moreover, it has been shown that topical use of staphylococcus toxin could cause more inflammatory reactions in psoriasis patients compared with controls or patients with atopic dermatitis or lichen planus.[10]

Nasal S.aureus carriage is frequent in patients with persistent allergic rhinitis. It seems that they are not only secondary bystanders, but actively alter the disease by promoting local IgE production.[11]

Moreover, S.aureus colonization is common in atopic dermatitis and can exacerbate the disease. Some patients with atopic dermatitis may act as a reservoir for S.aureus transmission to others. Atopic dermatitis patients have significantly greater presence of S.aureus on lesional and clinically normal skin, as well as the hand. The nares and

hands may be important reservoirs and vectors for autotransmission of S.aureus to the lesional skin and for transmission to patients with atopic dermatitis.[12]

In our study, 42% of psoriatic patients had positive cultures for S.aureus, at least in one anatomic site. The colonization rate of S.aureus in psoriasis has been reported to be between 27-33% in other studies that which is similar to our study.[13] The prevalence of the carrier state of S.aureus is estimated to be between 18-50% in healthy individuals.[14] It seems that the rate of S.aureus carriage shows no marked difference between psoriatic patients and healthy individuals. However, it would be better to perform a controlled study to reach more reliable results in this regard.

We found that a substantial percentage of psoriatic patients carried S.aureus. However, there was no significant difference in the PASI score between carriers and non-carriers in our study. Although a considerable percentage of psoriatic patients showed colonization of S.aureus, it had no significant impact on the severity of disease. On the other hand, the smears were not taken from active psoriatic lesions and we tried to determine nasal, axillary and perineum colonization of S.aureus in psoriasis patients. Further studies are required to assess the relationship between disease activity and lesional colonization to explain the role of S.aureus in psoriasis.

There are few studies which have determined the genes that code for S.aureus or Streptococcus toxins in patients with psoriasis. This fact might support the role of superantigens in the exacerbation of psoriasis.[15] However, further studies to determine the capacity of superantigen toxin production would be beneficial to elucidate the role of these toxins in the pathogenesis of psoriasis.

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