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CLINICAL REPORT

Microorganisms in Intertriginous Psoriasis: No Evidence of *Candida*

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Infection can be a trigger and an aggravating factor in psoriasis. Antibacterial and/or antifungal agents are commonly used in the treatment of intertriginous psoriasis, because it is believed that flexures in psoriasis are often colonized by *Candida* species and *Staphylococcus aureus*. Bacterial and fungal cultures were studied from 32 psoriatic patients with no topical treatment in the intertriginous areas, from 13 psoriatic patients treated with topical steroids and from 19 patients with no psoriasis or other affections of the skinfolds. Untreated psoriatic patients were colonized by *S. aureus* significantly more often than the control group but infection seemed to be unlikely. *Candida* was not found in any of the groups. It is proposed that intertriginous psoriasis be treated with topical steroids alone and that the routine use of antimycotic and antibacterial combinations should be avoided. *Key words: bacteria; dermatophytes; Staphylococcus aureus; topical steroids.*

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Psoriasis vulgaris is characterized by hyperproliferation of the epidermis and a persistent inflammatory infiltrate. Flexural psoriasis is estimated to occur in 2–6% of psoriatic patients (1). The body folds most affected are the axillary, inguinal, submammary, umbilical and intergluteal folds. The psoriasis plaques in intertriginous areas are characterized by an oozing, red inflammation without scaling. Earlier studies propose that infection can be a trigger and an aggravating factor in psoriasis. Streptococcal infection has been suspected as a triggering factor for many decades, especially in children and guttate forms of psoriasis (2–4). In several studies it has been suggested that *Staphylococcus aureus* and *Candida albicans* can act as superantigens in psoriasis, producing selective expansion of a population of T lymphocytes bearing a particular T-cell receptor variable region (VB) specificity (5–8).

Flexural psoriasis is believed to be frequently colonized by microorganisms, especially *Candida* species and *S. aureus* (9). Treatment with combinations of topical corticosteroids with antibacterial and/or antifungal agents is commonly used (9). However, if the role of microoganisms in psoriasis could be clarified, we would know if and what kind of antimicrobial treatment is needed. No microbial studies have been undertaken with special attention to the intertriginous areas in psoriasis.

The aim of this study was to investigate the occurrence of microoganisms in skinfolds affected by psoriasis and the influence of topical treatment.

MATERIALS AND METHODS

Study population

Thirty-seven patients with intertriginous psoriasis were selected consecutively. Both in- and outpatients were included. Inpatients were sampled on the day of admission to avoid hospital acquisition of *S. aureus*. Patients were excluded from the study if they had received systemic or topical antimicrobial or UV-light treatment during the preceding two weeks. Intertriginous areas sampled were the inguinal ($n=43$), axillary ($n=4$), submammary ($n=4$) and intergluteal ($n=16$) folds and the umbilicus ($n=3$).

Three groups of patients were studied:

I. Untreated psoriasis patients. This group comprised 32 patients, 18 males and 14 females (age range 32–81, mean age 61 years), with no topical treatment in the intertriginous areas for at least 2 weeks prior to the study.

II. Treated psoriasis patients. Thirteen patients, 5 males and 8 females (age range 23–81, mean age 60 years), treated with topical steroids in the intertriginous area were included. Ten patients were treated with clobetasone butyrate and 3 patients with mometasone furoase. Eight of the patients in this group had been included in the group of untreated patients first and were then given clobetasone butyrate for 2 weeks.

III. Control subjects. Nineteen control subjects, 8 males and 11 females (age range 24–84, mean age 62 years) were also studied. They were mainly patients with leg ulcers and skin tumours, with no affection of the skinfolds. Six patients were examined in two locations.

Microbiological techniques

Bacterial culture. Samples for bacterial cultures were taken from intertriginous areas with a cotton swab. The swab was inoculated on Colombia agar medium containing 5% horse blood and on selective agar medium for Gram-negative rods, streptococci and staphylococci. The selective agar medium for streptococci was incubated at 37°C in 95% air and 5% CO_2 and the other plates were incubated in air. The agar plates were examined after 24 and 48 h. Qualitative analyses were

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performed. Bacteria were identified using the conventional techniques.

Fungal culture. Samples for fungal cultures were taken as above. Two different media were used; Sabouraud's glucose medium without any antimicrobial additives and Casein medium containing thiamin. The plates were incubated at 32°C and examined every week for 3 weeks. Fungi were identified using standard procedures.

Statistical methods. Statistical analysis was performed using Fisher's exact test (two-tailed) for comparison of independent samples.

RESULTS

Bacterial cultures

The first 32 of the 70 samples were taken before and after cleaning the skin with saline. No difference was found in bacterial growth (data not shown) and the following samples were therefore taken without previous cleaning of the skin.

Significantly more of the untreated psoriatic patients had *S. aureus* compared with the controls ($p<0.005$). Thirty-eight percent of the samples showed *S. aureus* in the untreated group and 4% in the control group (Table I). In the group treated with clobetasone/mometasone, *S. aureus* was found in 54% of the samples. However, this difference was not statistically significant when compared with the untreated group. In the control group, the normal skin flora coagulase-negative staphylococci predominated. *Streptococcus haemolyticus group G (Str. Gr. G)* was found in 19% of the untreated psoriatic patients and in none of the controls ($p<0.05$) or treated patients. No significant difference in qualitative bacterial growth in different intertriginous areas was found.

Table I. *Distribution of microorganisms in intertriginous areas*

	Positive sites		
Culture findings	Untreated psoriasis $n=32$ % (n)	Treated psoriasis[a] $n=13$ % (n)	Controls $n=19$[b] % (n)
Staph. aureus	38 (12)	54 (7)	4 (1)
Coag.-neg. staph.	59 (19)	46 (6)	92 (23)
Diphtheroids	13 (4)	0 (0)	28 (7)
Str. haemolyt Gr A	0 (0)	8 (1)	0 (0)
Str. haemolyt Gr B	13 (4)	15 (2)	0 (0)
Str. haemolyt Gr C	0 (0)	0 (0)	4 (1)
Str. haemolyt Gr G	19 (6)	0 (0)	0 (0)
Enterococc.	6 (2)	0 (0)	0 (0)
Proteus	3 (1)	0 (0)	0 (0)
Gram-neg. flora	16 (5)	8 (1)	0 (0)
Candida	0 (0)	0 (0)	0 (0)
Dermatophytes	0 (0)	8 (1)	0 (0)

[a]Clobetasone butyrate ($n=10$); mometasone furoase ($n=3$).
[b]19 patients, 25 sampling sites.

Fungal cultures

Not a single case of *Candida* was detected in any of the groups. The only dermatophyte found was one case of *Trichophyton rubrum* in a patient with inguinal psoriasis and tinea pedis.

DISCUSSION

In this study, special attention has been directed to the bacteria and fungi found in intertriginous psoriasis, which has not been done in earlier studies. Intertriginous psoriasis is characterized by red, oozing inflammation, and cutaneous candidiasis is often suspected (9). The microflora of 297 patients with psoriasis has been extensively examined by Noah (10). She found more than 30 different microorganisms in lesional skin. In the intertriginous areas, the following microorganisms were found: streptococci, Gram-negative rods, *S. aureus*, *C. albicans* and dermatophytes. No comparison with controls was made. In a retrospective study, Rosenberg et al. (11) found associations between *Malassezia* and scalp, ear and face psoriasis, and between bacteria and bodyfold, nailfold, gluteal and rectal psoriasis. The microorganisms found in the skinfolds were streptococci, Gram-negative rods and, in a few cases, *Candida*.

Some authors have paid special attention to *S. aureus*, without focus on the intertriginous areas. Marples et al. (12) found that nearly half of the psoriatic lesions were colonized by *S. aureus* and that uninvolved skin showed significantly less *S. aureus*. However, in several reports (13–15) no significant difference in the colonization of *S. aureus* in psoriatic plaques compared to adjacent normal skin was found. Nyström et al. (16) found that, compared with other dermatoses, staphylococcal colonization in psoriatic lesions was not more common and the intensity of growth was less. Healing of dermatoses, including psoriasis, seemed uninfluenced by *S. aureus* colonization (16). Total eradication of staphylococci was extremely difficult to achieve and no subjective or objective improvement was seen during treatment (16). The main problem is to differentiate infection from simple colonization. Few of our cultures showed a monoculture of *S. aureus*, and it is therefore highly likely that *S. aureus* does not act as a pathogen. Using topical antibiotics with the intention of eradicating *S. aureus* does not seem necessary or even appropriate.

Our finding of *Str. Gr. G* in 19% of the intertriginous psoriatic lesion is very interesting. Nohlgård et al. (17) studied positive streptococcal skin cultures in inpatients in a dermatological ward. Samples were taken from ulcers or infected skin areas, the throat or intertriginous areas. They found *Str. Gr. G* in half of the patients with positive cultures. Patients with no clinical infection were not treated but some later developed erysipelas. These authors believe that *Str. Gr. G* skin infections must be treated with the same clinical vigilance as *Str. Gr. A* infections, and therefore treated even if an

infection is not clinically observed. Smith & Waterworth (18) investigated some cases of intertrigo in different diseases and found that streptococci were present in all the fissured or eroded lesions. Nine out of 15 strains isolated were haemolytic. Smith & Waterworth suggest using topical antibiotics to treat fissured or eroded areas of intertrigo.

Candida was not detected in our study. No comparable study can be found. The files of 3,006 patients with psoriasis and 1,808 patients with atopic dermatitis were analysed at the Department of Dermatology in Kiel. *Candida* infections of the skin were seen in psoriatic patients more often than in controls, but no connection between *Candida* infection in skin and psoriasis activity could be detected (19). No information on the sampling sites of the skin was given. The results in our study do not warrant recommending general local antimycotic treatment for patients with intertriginous psoriasis.

One case of *T. rubrum* in the inguinal fold was found in our study. This patient was culture negative when he was included in the group of untreated psoriatic patients. Two weeks later after treatment with clobetasone butyrate, he was culture positive for *T. rubrum*. It is possible that a topical corticosteroid cream used to treat the patient could have activated a tinea infection. The patient also had tinea pedis. The presence of dermatophytes in psoriasis has been studied by Henseler & Tausch (19), who even found that patients with psoriasis presented a decreased rate of tinea, but the decrease was only significant for tinea corporis.

Whether antibiotics should be combined with topical steroids is a difficult question to answer. Marples et al. discussed the issue of topical steroid-antibiotic combinations elsewhere (20). They induced *S. aureus* and *C. albicans* infections in healthy individuals. The corticosteroid alone (triamcinolone) clearly moderated the lesions even though *S. aureus* was seen to multiply. The addition of antibiotics (neomycin and gramicidin) to the corticosteroid almost completely suppressed the signs of infection. Marples et al. (20) suggest that a steroid-antibiotic combination would be justified because the dermatologist cannot undertake a bacteriological analysis of every case, and that he must seek security in broad coverage. Chan et al. (21) found no significant quantitative or qualitative changes in the microbial flora when topical triamcinolone alone was applied to normal skin, when compared with controls. When a skin infection is treated with corticosteroids, the lessening of the serous exudate can be expected to aid the restoration of the normal microflora. This is one reason why corticosteroids used alone can be beneficial in infected dermatoses. There is no evidence to show that corticosteroids potentiate topical infections (20). The main problem in intertriginous psoriasis is whether there is an infection or not. Our findings tend to favour colonization.

In conclusion, we recommend that intertriginous psoriasis be treated with topical steroids alone and the routine use of antimycotic and antibacterial combinations be avoided. When fissures or erosions are present, more frequent sampling is recommended and when pathogens such as streptococcus haemolyticus are found, treatment should be given.

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