

Bacterial colonization of psoriasis plaques. Is it relevant?

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Abstract

Bacterial colonization was investigated retrospectively in patients with plaque psoriasis (n=98 inpatient treatments, n=73 patients). At least one pathogen was found in 46% of all cases. *Staphylococcus aureus* was the most frequent bacterium. Bacterial colonization of psoriasis plaques could be relevant in individual cases.

The exacerbation of Psoriasis vulgaris can be caused by various triggers.¹ While bacterial infiltration of afflicted skin is known to be a trigger factor in chronic inflammatory dermatoses such as atopic dermatitis,² the bacterial flora of psoriasis plaques and their possible importance have received only little attention thus far.

The bacterial smears of lesional skin performed routinely on admission to our clinic were examined retrospectively for all psoriasis patients. The observation period was 16 months. The data of n=98 inpatient treatments (n=73 patients) were assessed, whereby n=56 patients were treated once, n=12 patients twice and n=5 patients underwent repeated inpatient treatments. Overall, n=45 (46% of all psoriasis cases) had colonization of the psoriasis plaques with pathogenic bacteria, whereby gram-positive bacteria were found most frequently (Figure 1). Of these patients with pathogenic colonization, n=22 had one pathogen, while n=18 of those examined had two bacteria and n=5 three or four different bacteria. *Staphylococcus aureus*, *Streptococcus pyogenes*, *Streptococcus agalactiae*, *Enterococcus faecalis*, *Klebsiella pneumoniae* and *Oxytoca*, *Acinetobacter baumannii*, *Proteus mirabilis*, *Stenotrophomonas maltophilia*, *Serratia marcescens*, *Acinetobacter lwoffii*, *Pseudomonas aeruginosa* and *Escherichia coli* were identified as pathogenic bacteria. The most prevalent bacterium was *Staphylococcus aureus* (n=31). Clinically, the plaques covered with pathological bacterial flora were in part erosive. The PASI on admission for patients with pathological colonization was 16.1 versus 14.0 in patients without proof of bacterial colonization or resident flora (not significant (t-Test)). The patients admitted two or more times showed a change in bacterial coloniza-

tion in eleven cases without any regular pattern. There was a change between gram-positive and gram-negative colonization, as well as new colonization of previously-sterile plaques with various pathogenic bacteria. However, in three cases, colonization with *Staphylococcus aureus* was no longer present on re-admission (sterile finding in each case). These three patients did not receive any antimicrobial treatment between their in-house treatments. One woman developed erysipelas of the abdominal wall out of a navel plaque with colonization by *Staphylococcus aureus* (Figure 2).

Nearly half of the patients examined had colonization of the psoriasis plaques with (potentially) pathogenic bacteria. Examinations to date of the bacterial flora in psoriasis have concentrated on the proof of *Staphylococcus aureus* and found a prevalence of up to 64% in lesional skin of patients with plaque psoriasis.^{3,4} Corresponding to this, *Staphylococcus aureus* has also been found as the most prevalent bacterium in patients with superinfected pustular psoriasis.⁵ The frequency of 46% bacterial colonization in the group we examined was lower than that reported in the literature, but it showed a considerably broader spectrum of pathogens, especially including gram-negative bacteria.

In most patients, the pathogens appear as (transient) colonization or superinfection. This is supported by the lack of difference in the PASI scores on admission and the changing flora in patients admitted more than once. Nonetheless, bacterial colonization may have systemic and local effects. For example a superantigen effect has been described in psoriasis especially for *Staphylococcus aureus*, the most-frequently found bacterium.^{3,4} Enterotoxins seem to play an important role in this superantigen activity.⁴ Moreover, triggering of psoriasis by Streptococci is known.^{1,6} On the other hand, local effects on single colonized plaques might be possible, since the clinical picture with crusting and erosive surface indicates an immunoresponse. Local infections of psoriasis plaques are usually prevented by high concentrations of antimicrobial peptides.⁷ Nonetheless this barrier seems to be breached in single cases. In the group of patients we examined, one woman developed erysipelas which began clinically in the area of a psoriasis plaque. This shows that systemic infections from local bacterial colonization could not be prevented in individual cases in spite of an overexpressed innate immune system in psoriasis.

Additional investigations are needed to better estimate the clinical relevance of colonization and potential breaches of the innate immune system in psoriasis patients.

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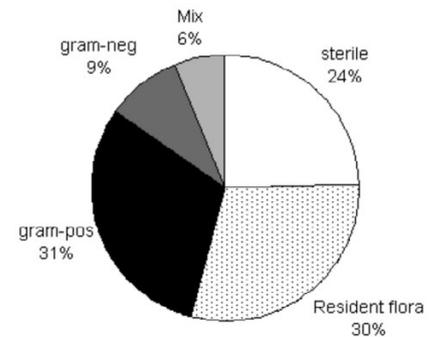


Figure 1. Distribution of bacterial infection of psoriasis plaques. Mix=Combination of gram-positive and gram-negative pathogens.



Figure 2. Erysipelas of the abdominal wall starting from an umbilical psoriasis plaque with proof of *Staphylococcus aureus*.

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