

Case report

A novel finding in atopic dermatitis: film-producing *Staphylococcus epidermidis* as an etiology

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Recently, we surveyed our inner city practice regarding the forms of atopic dermatitis we see here, including facial-extendsor, flexural, nummular, lichen planus-like (seen only in darkly pigmented skin), and pityriasis alba.¹ Many factors triggering this disease have been proposed, such as xerosis and sweating,² but factors causing pruritus, especially in flexural eczema, have not been elucidated. We believe the patient presented herein shows findings pointing to an explanation for such pruritus. We believe that subclinical miliaria caused by film-producing *Staphylococcus epidermidis* blocks the eccrine ducts and causes pruritus similar to that seen in overt miliaria rubra.

A 19-year-old Caucasian woman with a family history of maternal asthma presented with a very pruritic, pink-red, papulovesicular eruption on the antecubital and popliteal fossas. She habitually bathed twice daily with a body wash and did not use a moisturizer. We diagnosed atopic dermatitis and prescribed twice-daily application of a midpotency corticosteroid cream; skin care advice consisted of using a moisturizer daily and decreasing the frequency of bathing. We recommended that she use a milder soap and restrict its use to certain body parts. At her three-week follow-up visit, the pruritus had abated and the rash had cleared.

At the follow-up visit, a skin scraping was taken from the right antecubital fossa in a manner similar to a potassium hydroxide preparation for fungus. The sample was cultured for bacteria and was examined under confocal microscopy. The culture showed normal skin flora (predominantly

S. epidermidis) without any *Staphylococcus aureus*. Cocci with biofilms similar to those of film-producing *S. epidermidis* were noted on confocal microscopy and appeared as a syncytium as opposed to ordinary single bacterial cells (Fig. 1).

This patient demonstrated typical flexural atopic dermatitis that responded well to treatment. The skin scraping showing film-producing *S. epidermidis* is of interest. We believe subclinical miliaria causes the itching in this form of eczema, which has been termed “the itch that

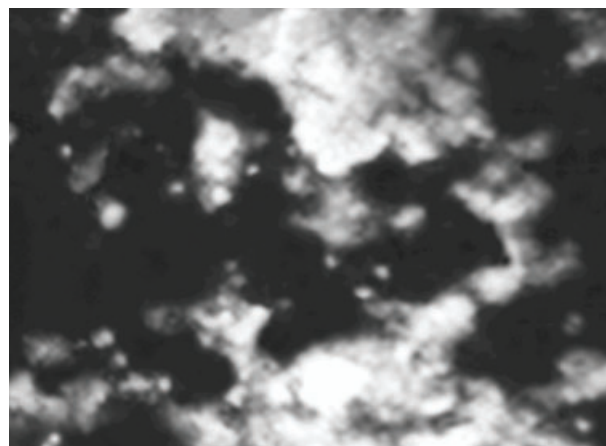


Figure 1 Confocal photomicrograph (original magnification $\times 600$) shows the thick syncytia of cocci and biofilms typical of *Staphylococcus epidermidis*. Those of *Staphylococcus aureus* are much thinner

rashes".³ Sulzberger *et al.*⁴ first alluded to this when they found blockage of the sweat ducts in a biopsy specimen from an eczema patient. Tuft *et al.*⁵ later showed that eczema occurred in areas of sweating, even in areas where patients did not realize they perspired. Dahl,² in his review of flare factors, discussed sweating and sweat retention as important factors in the induction of atopic dermatitis. Recently, it was shown in patients keeping diaries that sweating was a significant factor in exacerbating eczema.⁶

Miliaria arises from blockage of the eccrine sweat ducts. The material causing this blockage is a periodic acid Schiff-positive substance that contains many gram-positive bacteria.⁷ This material has been identified as extracellular polysaccharide substance, so-called slime or film, and the bacterial organism has been identified as *S. epidermidis*.⁸ The genes responsible for the film production in *S. epidermidis* have been elucidated.⁹ Our finding of film-producing *S. epidermidis* (the syncytium seen on confocal microscopy) in flexural eczema extends Sulzberger *et al.*'s concept⁴ and couples it with the observations of both Holze and Kligman⁷ and Mowad *et al.*⁸ The finding is also consistent with Tuft *et al.*'s observations that eczema occurs in areas where insensible (unrecognized) sweating has occurred.⁵ The finding also supports the supposition that the organism is ever-present: theoretically, it should be present before, during, and after the rash.

Filaggrin, the gene that regulates epidermal barrier function, has recently been identified as a major predisposing factor in atopic dermatitis.¹⁰ Our concept of subclinical miliaria provoked by film-producing *S. epidermidis* as part of a "double-hit" phenomenon¹¹ fits well in the pathogenesis of atopic dermatitis. Consequently, the patient would experience subclinical miliaria as a result of insensible sweating and sweat duct occlusion with film-producing *S. epidermidis*, itch profusely, damage an already predisposed, filaggrin-deficient stratum corneum, and create clinically apparent disease. In our patient with clinical flexural atopic dermatitis, we have demonstrated the presence of the organism that, we believe, starts this cascade.

Conflicts of interest

None.

References

- 1 Allen HB, Jones NP, Bowen SE. Lichenoid and other clinical presentations of atopic dermatitis in an inner city practice. *J Am Acad Dermatol* 2008; 58: 503–504.
- 2 Dahl MV. Atopic dermatitis: the concept of flare factors. *S Med J* 1977; 70: 453–455.
- 3 Boguniewicz M. Atopic dermatitis: beyond the itch that rashes. *Immunol Allergy Clin North Am* 2005; 25: 333–351.
- 4 Sulzberger MB, Herman F, Zak FG. Studies of sweating. I. Preliminary report with particular emphasis on a sweat retention syndrome. *J Invest Dermatol* 1947; 9: 221–242.
- 5 Tuft L, Tuft HS, Heck M. Atopic dermatitis. II. Role of the sweating mechanism. *J Invest Dermatol* 1950; 15: 333–337.
- 6 Langan SM, Bourke JF, Silcocks P, Williams HC. An exploratory prospective study of environmental factors exacerbating atopic eczema in children. *Br J Dermatol* 2006; 154: 979–980.
- 7 Holze E, Kligman AM. The pathogenesis of miliaria rubra: role of the resident microflora. *Br J Dermatol* 1978; 99: 117–137.
- 8 Mowad CM, McGinley KJ, Foglia A, Leyden JJ. The role of extracellular polysaccharide substance produced by *Staphylococcus epidermidis* in miliaria. *J Am Acad Dermatol* 1995; 33: 729–733.
- 9 Arciola CR, Baldassarri L, Montanaro L. Presence of icaA and icaD genes and slime production in a collection of staphylococcal strains from catheter-associated infections. *J Clin Microbiol* 2001; 39: 2151–2156.
- 10 Palmer CNA, Irvine AD, Terron-Kwiatkowski A, *et al.* Common loss-of-function variants of the epidermal barrier protein filaggrin are a major predisposing factor for atopic dermatitis. *Nat Genet* 2006; 38: 441–446.
- 11 Serra E, Puig S, Otero D, *et al.* Confirmation of a double-hit model for the NF1 gene in benign neurofibromas. *Am J Hum Genet* 1997; 61: 512–519.