Impact of Staphylococcus aureus colonisation on barrier function and cytokine profile in atopic dermatitis skin

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Atopic dermatitis (AD) is a widespread skin disease that is characterized by lesions at specific body sites, which are typically driven by a type 2 immune response. While the active state is well-researched and a broad variety of treatment options exists, lesions often recur in a matter of weeks after treatment cessation. Recent research suggests that clinically healed AD skin is still distinguishable from healthy skin through increased subclinical inflammation. To understand the pathophysiology of relapses in atopic dermatitis, a field for which hardly any research has been conducted, we conducted an exploratory study involving more than 20 patients with currently active (ex-lesional) AD, who underwent either topical therapy or phototherapy, and were biopsied before new lesional outbreaks. Using single-cell sequencing, we correlated the expression profile of the healed skin with the patients’ subsequent time until relapse. With the complementing multiplex analysis of blood cytokines we aimed to assess the relevance of the systemic immune response for the reappearance of flares. Our preliminary data suggest that certain AD associated molecular markers in blood and skin indeed precede clinical relapse. By sharing our first results of the study, we hope to pave the way to a deeper understanding of flare development and new therapeutic interventions to delay or even prevent AD relapses in a targeted manner.

Identifying immune targets for atopic dermatitis relapse prevention

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The autoimmune, blistering disease pemphigus vulgaris is caused by autoantibodies against the desmocollin cadherins, mainly desmoglein-3. Recently, it has been shown that blocking the neonatal Fc receptor (FcRn) can lead to a rapid decrease of pathogenic IgG and an improved cutaneous barrier function. Thus, FcRn has been proposed as a potential therapeutic target in pemphigus vulgaris. In this study, we aimed to investigate the potential of FcRn as a therapeutic target in pemphigus vulgaris.

We treated six symptomatic patients with FcRn blockade using the FcRn inhibitor efgartigimod, which is approved for the treatment of myasthenia gravis and is currently in development for pemphigus vulgaris. The patients were treated with either low-dose oral prednisone alone or in combination with FcRn blockade. During treatment, we monitored clinical parameters, serum levels of autoantibodies, and skin barrier function.

Our results showed a significant improvement in clinical parameters, including a reduction of bullous lesions and an increase in skin barrier function. Serum levels of autoantibodies also decreased significantly. These findings suggest that FcRn blockade has the potential to improve clinical outcomes in pemphigus vulgaris.

Thus, FcRn blockade may be a promising therapeutic approach for the treatment of pemphigus vulgaris.