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Severe Atopic Dermatitis in an Adult Exacerbated by *Staphylococcus intermedius* and Improvement with IVIG

Varaz Bozoghlanian*, Ravi Gutta and Donald Levy
Department of Basic and Clinical Immunology, UC Irvine School of Medicine, USA

***Address for Correspondence**
Varaz Bozoghlanian, Department of Basic and Clinical Immunology, UC Irvine School of Medicine, Medical Sciences I, C-249, Irvine, CA 92697, Tel: 949-824-5818; Fax: 949-824-4362; E-Mail: bouzoghv@uci.edu

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Reviewed & Approved by: Dr. Shasa Hu, Assistant Professor, Department of Dermatology and Cutaneous Surgery, University of Miami, USA

Introduction

Staphylococcus aureus is known to exacerbate atopic dermatitis with production of bacterial enterotoxins and superinfection of lesions [1-3]. We report the first case of severe atopic dermatitis in a human exacerbated by *Staphylococcus intermedius*, which has been associated with exacerbation of atopic dermatitis in dogs. After failing first and second-line therapies, our patient had significant improvement with high-dose IVIG treatment.

Case Description

A 26 year-old Hispanic man presented with a 6-year history of extremely pruritic diffuse rash consistent with atopic dermatitis. He was referred for total IgE >16,000 IU/ml and failure to respond to high-dose topical and oral corticosteroids, methotrexate, cyclosporine, and phototherapy. In our clinic, an immunodeficiency evaluation was normal. Workup for hyper-IgE syndrome, including STAT-3 and Dock-8 gene mutations, was normal. Skin biopsies were negative for malignancy, fungal staining, or vasculitis, and suggestive of severe atopic dermatitis with normal immunofluorescence and immunophenotyping. Workup for a 70-pound unintentional weight loss was performed, with negative serum and urine electrophoresis, HIV, HTLV, bone marrow biopsy, flow cytometry, CT chest/abdomen/pelvis, endoscopies, and autoimmune evaluation. The patient had multiple severe exacerbations requiring several hospitalizations. Aggressive skin care measures (including wet/dry wraps), high-dose antihistamines, gabapentin, oral antibiotics, a one-month trial of terbinafine, and a five-month trial of omalizumab were not efficacious.

Multiple skin cultures identified highly-resistant *Staphylococcus intermedius*. The patient developed malignant otitis externa treated with daptomycin and meropenem. Cultures from the ear also grew *Staphylococcus intermedius*. His skin lesions improved on the IV antibiotics, but he continued to have a relapsing-remitting course when the antibiotics were withdrawn. He was started on high-dose IVIG 2g/kg every 4 weeks, with significant improvement of his symptoms and no exacerbations over 4 months.

Discussion

Staphyloccocus intermedius has been associated with exacerbation of atopic dermatitis in dogs, and several studies report increased *Staphylococcus intermedius* colonization and adherence to the keratinocytes of atopic dogs compared to healthy controls [4-8]. Dogs may be a reservoir for antimicrobial-resistant bacteria, including *Staphylococcus intermedius* [9,10]. *Staphylococcus intermedius* may be transmitted to humans with close contact to dogs [11,12], and our patient did have a history of dog exposure when living with his parents. This case suggests that in patients with severe atopic dermatitis refractory to first and second-line therapies, it may be prudent to investigate for *Staphyloccocus intermedius* colonization and/or infection, and if identified, should be treated with tailored antibiotic therapy based on drug sensitivity.

The management of some moderate to severe forms of atopic dermatitis (SCORAD 18–40) resistant to topical treatment as well as that of severe forms (SCORAD >40) requires the use of systemic drug application [13]. Treatment with IVIG has been reported to be beneficial in carefully selected patients with severe atopic dermatitis who have failed first and second-line therapies [14-19]. These studies include many case reports and case series in adults and children, but there are few large randomized double-blind placebo controlled trials. Some studies have not shown benefit [20]. Based on our patient, there may be a role for IVIG in treating the phenotype of severe recalcitrant atopic dermatitis with multi drug-resistant bacterial colonization or superinfection. The mechanism of action, duration of treatment and dosing guidelines of IVIG in treatment of atopic dermatitis remains to be elucidated. Further randomized controlled studies are needed to evaluate the utility of IVIG in severe atopic dermatitis.

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