Severe Adult-Onset Atopic Dermatitis: Investigating the Pathogenic Role of Malassezia spp. and Anti-Fungal Treatment in Refractory Disease - A Case Report

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Abstract
Atopic Dermatitis (AD) is a common inflammatory dermatosis characterized by pruritus and a cyclic clinical course. It’s estimated prevalence in industrialized countries has tripled over the last 30 years, affecting 15-30% of children and up to 10% of adults1. AD is one part of the ‘Atopic Triad’, along with allergic rhinitis and asthma. The pathogenesis and clinical course of AD is likely multifactorial with defective barrier function (filagrin deficiency/mutation), allergic, and infectious processes implicated1,2,3. Specifically, Malassezia spp. yeasts have been demonstrated to have a pathogenic role in at least some cases of AD. Circulating anti-Malassezia antibodies are only seen in patients with AD3. Antibody titers have been correlated to severity in several studies1,2,3,4.

We report the case of an otherwise healthy 37 year old African American patient with severe, disfiguring AD of 19 years duration. The patient presented to our dermatologic clinic an intensely pruritic and cosmetically disfiguring dermatitis. Physical exam revealed a diffuse inflammatory dermatosis with lichenification, thickening, and disfiguration involving > 80% BSA and non-tender axillary/inguinal adenopathy. The psychological impact for this patient was severe; the disfigurement had made employment virtually impossible, and interpersonal relationships suffered greatly.

Various topical corticosteroids (triamcinolone 0.1% ointment, hydrocortisone 2.5% cream), as well as topical calcineurin inhibitors (tacrolimus 0.1% ointment) failed to improve the condition over a one year course, and oral cyclosporine therapy was initiated at 100mg twice daily, in addition to the topicals. Minimal improvement was seen over another year on this combination. At this point, other etiologies and therapeutic strategies for the dermatosis had to be considered. Laboratory testing was ordered

Clinical Images 10/2014
Prior to initiation of cyclosporine 100mg BID

Clinical Images 7/2015
After 12 months of cyclosporine 100mg qd and 1 month of terbinafine 250mg qd

Clinical Images 8/2015
2 months after initiation of terbinafine 250mg qd

Table 1
Selected Laboratory Values
IgE: 24311 (H)
Malassezia Mix-IgE: 11.30 kU/L (H)

- **Reference Ranges**
  - >50.00 - Very High Positive
  - >30.00 - High Positive
  - >10.00 - Moderate Positive
  - >0.34 - Low Positive
  - <0.35 - Evasive/Borderline
  - <0.10 - Negative

- **CBC**
  - WBC: 5.8
  - HGB: 12.3
  - HCT: 38.0
  - Na: 140
  - K: 3.7
  - Cl: 108
  - IgG: 641
  - IgM: 18
  - IgA: 122

- **CMP**
  - CRP: 0.1
  - ESR: 14
  - Blood Culture: Neg.
  - ANA: Neg.
  - IgG: 24311 (H)

ANA: Neg
Blood Culture: Neg.
ESR: 14
Lymph Node Biopsy: Reactive lymph node with paraclonal hyperplasia consistent with dermatopathic lymphadenopathy.

The possibility of an allergy-mediated process was supported by the elevated serum IgE level (in the 24,000kU/L range; ref. range <114kU/L), and the suspected role of Malassezia spp. commensurate yeast was confirmed by immunoCAP allergen-specific IgE testing (11.30; ref. range <0.35).

The regimen was then changed to oral terbinafine 250mg daily, cyclosporine 100mg daily, and the topicals previously mentioned. Within one month the patient reported significant improvement in pruritus, diffuse softening of the skin, and repigmentation.

Clinical improvement continues to be seen today.

References