ATOPIC DERMATITIS

Atopic dermatitis is a chronic pruritic inflammatory skin disease. It is predominantly a childhood disease, but its incidence in both adults and children is on the rise. Persistent atopic dermatitis occurs in approximately 50 percent of patients diagnosed with atopic dermatitis during childhood.

My approach to treatment may include any or all of the following strategies.

1. Improve skin barrier function with the application of ceramide containing moisturizers.

Application of ceramide enriched emollients aid in the restoration of permeability barrier function and improved antimicrobial defense in patients with atopic dermatitis. Moisturizers containing urea, alpha-hydroxy acids, or hyaluronic acids have also led to the improvement of the integrity of the stratum corneum.

2. Broad-spectrum multivitamin and antioxidant supplementation.

Atopic dermatitis patients are more prone to damage caused by reactive oxygen species when compared to normal healthy controls, as evidenced by an increase in serum malondialdehyde, a stable end product of fatty acid peroxidation. Other oxidative stress markers such as superoxide dismutase, catalase, glutathione peroxidase, and non-enzymatic antioxidants like reduced glutathione, vitamin A, vitamin E, and vitamin C, are decreased in atopic dermatitis patients, further demonstrating increased levels of oxidative stress in this population. Therefore, antioxidant supplementation may be of some benefit in the treatment of atopic dermatitis.

3. Diet improvement: A low glycemic, mostly plant-based diet with adequate protein is a given for any inflammatory disease. Also, increasing vitamin A intake, preferably through dietary manipulation, is advocated.

Deficiency of retinoids and retinoid signaling in the skin, or general vitamin A deficiency, has been associated with various symptoms of atopic dermatitis. Also, imbalanced T-cell immunity, altered apoptosis, altered skin differentiation and proliferation, and increased bacterial skin colonization have been associated with vitamin A deficiency or deletion of retinoid receptor-mediated signaling in transgenic skin-specific mouse models.

Consumption of carrot juice containing high concentrations of the provitamin A carotenoid beta-carotene results in significantly increased plasma concentrations of the active vitamin A metabolite all-trans retinoic acid.

Compared to a raw carrot meal without avocado, the addition of one avocado increased beta-carotene absorption 6.6 times, alpha-carotene absorption 4.8 times, and increased the conversion of provitamin A to vitamin A 12.6 times.

4. Food sensitivity testing

Sensitization to food allergens, while not a causative factor, maybe a contributory factor in a subgroup of patients with severe disease. Skin or in vitro (blood) testing for food allergies and sensitivities may be helpful for this group of atopic dermatitis patients.

5. Vitamin D supplementation based on serum 25OH-vitamin D3 levels.

6. Essential fatty acid supplementation, including DHA, EPA, and GLA.

Essential fatty acid supplementation will be based on blood tests to determine the patient's omega-3 index and blood levels of omega 3&6 fatty acids. While clinical trials have yielded mixed results, there is good evidence for beneficial effects, especially when there is documentation that improved blood levels of the appropriate fatty acids have been obtained.

7. Dilute bleach baths to decrease S. aureus colonization and superantigen production.

The effectiveness of bleach baths is controversial but may be useful in those with frequent and recurring flares.

8. Virgin coconut oil can be applied as a moisturizer and to reduce colonization with S. aureus. Indeed, coconut oil has shown efficacy in clinical trials for atopic dermatitis. Topical application of coconut oil and sunflower oil can improve barrier function and decrease transepidermal water loss in preterm infants.

9. Dust mite control measures may be helpful in patients with documented sensitivity to dust mites.

House dust mite proteins generate neolipid antigens presented by CD1a (present on Langerhans cells and cutaneous dendritic cells) to T cells and thereby directly activate T cells in the blood and skin lesions of affected individuals. CD1a-reactive T cell activation appears to be dependent on house dust mite derived phospholipase A2, which is inhibited by the skin barrier protein filaggrin.

Filaggrin stands for filament aggregating protein. Filaggrin binds to keratin fibers in epithelial cells and is important for skin barrier function and cutaneous pH regulation, hydration, and antimicrobial protection. Filaggrin is aberrantly expressed in many atopic dermatitis patients.

Filaggrin insufficiency is associated with atopic dermatitis. It follows that patients with atopic dermatitis cannot inhibit house dust mite derived phospholipase A2. Phospholipase A2 activates T cells, which contributes to the inflammation seen in atopic dermatitis patients.

10. Testing for intestinal dysbiosis with followup treatment as indicated, including the use of probiotics, are additional considerations.

11. Stress management is essential. Stress has a deleterious effect on the skin barrier function.

12. A prescription for a calcineurin inhibitor-containing ointment to be applied twice daily to affected areas can be quite helpful.

13. Crisaborole is a boron-based topical phosphodiesterase 4 inhibitor for the treatment of mild to moderate atopic dermatitis in adults and children three months of age and older. Crisaborole ointment can improve the clinical signs of atopic dermatitis, including pruritus, redness and skin thickening, and decrease excoriations.

A recent and exciting addition to the armamentarium in controlling atopic dermatitis symptoms is systemic medications that can be administered via injection or oral ingestion. These will be discussed in future blogs.

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