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Commentary

A Short Note on Skin Allergy

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INTRODUCTION

Allergic skin diseases include urticaria, vascular edema, contact dermatitis, and atopic dermatitis, but the closest model to the systemic concept of allergies is Atopic Dermatitis (AD), the etiology of which is skin. It is associated with complex interactions between barrier disorders and environmental factors, as an allergen or microorganism. In particular, a significant advance was the demonstration that mutations in the skin barrier protein filaggrin are closely associated with allergen sensitization and the development of asthma in AD patients. Changes in skin barrier function caused by several factors cause allergens to pass through the skin and cause a systemic reaction. The central role of such a reaction is played by Langerhans cells, which capture allergens via immunoglobulin E (IgE) receptors and present them to T cells. Activation of T-helper type 2 (Th2) cells produces patterns of pro-inflammatory cytokines and chemokines that sustain inflammation. Known AD-related cytokines are interleukin (IL) 5, IL13, tumor necrosis factor (TNF) α, IL17, which is thought to cause airway inflammation after skin exposure to antigen, and IL31, which is predominantly expressed in skin homing Th2, is becoming more important. Skin homing is another important event in AD mediated by skin lymphocyte-related antigen (CLA) receptors that characterize T cell subpopulations that play different roles in AD and asthma.

SKIN DISORDERS

Contact dermatitis is usually topical and consists of skin inflammation at the site of contact with the hapten, whereas Urticaria/vascular edema (UA) and Atopic dermatitis (AD) are associated with specific allergens in the gastrointestinal tract. Appears systemically as an exposure to (as in both foods) UA and AD or the respiratory tract (as in the case of AD dustmite) can cause an allergic reaction to the skin. A typical skin lesion of urticaria is a wheal characterized by a central swelling surrounded by itchy erythema, which usually resolves after a few hours. In angioedema, the swelling is more pronounced, affects the dermis and subcutaneous tissue, is more painful than itching, subsides slowly, and lasts up to 72 hours. UA can be triggered by a variety of causes, including physicochemical stimuli, infections, autoimmunity, and vasculitis, and can manifest acutely (<6> lasting 6 weeks). Allergies are a relatively common cause of acute UA, but account for only 5-10% of chronic UA. Exposure to certain allergens causes the release of mast cell mediators, with histamine playing an important role. Local contact, such as that that occurs with latex, can cause urticaria (local urticaria) at the contact site, but the most common symptom is systemic urticaria caused by the ingestion of the causative food. In oral allergy syndrome, it is interesting that contact between food and the oral mucosa only causes local symptoms, but systemic symptoms are often with food intake, despite local disorders, most commonly urticaria.

ATOPIC DERMATITIS

The importance of systemic reactions is demonstrated by the natural history of atopic diseases. It is characterized by an early onset of AD followed by respiratory symptoms with asthma and rhinitis, summarized by the term “atopic march”.

Recent data on the role of skin barrier dysfunction focus on filaggrin, a filament-related protein bound to the keratin fibers of epidermal cells. Loss of function of the filaggrin gene null mutations have been found to predispose to AD, and the filaggrin gene is associated with sensitization to allergens, a more severe phenotype of eczema, and eczema-related asthma.

There are also serological markers of AD disease activity: in one study, Kakinumi et al. We have found that macrophage-derived chemokine CCL22 levels are closely associated with AD activity, as assessed by the commonly used scoring index SCORing AD (SCORAD). In another study, there is a significant correlation between AD activity and serum levels of eotaxin 3/CCL26. Both chemokines are suspected to be involved in the cause of AD.

CONCLUSION

In summary, AD has a complex pathophysiology and is recognized as a *primum movens* a skin barrier dysfunction associated with defective filaggrin expression. It promotes the passage of microorganisms and allergens through the skin, triggers an immune response involving many actors, and causes systemic symptoms. A better understanding of the subtle mechanisms that regulate this process can lead to significant advances in the diagnosis and treatment of AD.

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