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Commentary

Immunological Imbalance between Atopic Dermatitis and Asthma and its Subsequent Association

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DESCRIPTION

Atopic disease is a multifactorial chronic disorder that can evolve from one another and have overlapping etiological mechanisms. Atopic dermatitis is most often the first step in the development of atopic dermatitis and represents a major socio-economic burden in developed countries. Treatment of atopic dermatitis is often time consuming and in some cases less effective than expected.

Stratification of patient populations according to the clinical phenotype of the disease and specific measurable values (biomarkers) helps identify the most important etiologic mechanisms of the disease in these populations. This increases the predictive value of the evolution of the disease and allows the use and exploration of more targeted therapies to prevent this evolution and comorbidities.

Asthma is a long-term condition that affects both children and adults. Approximately 300 million people worldwide suffer from asthma, and it is estimated that another 100 million will be affected by 2025. Atopic asthma is the most common form of asthma, affecting 70-90% of children and 50% of adult patients.

AD is associated with Food Allergies (FA), asthma, and Allergic Rhinitis (AR), with or without elevated IgE levels. This gradual transition from one atopic disease to another over a nearly specific age range is called an atopic march. AD is considered to be the first step because disorders of the skin barrier, inflammation, and bacterial dysbiosis. Cause the sensitization required for the development of other atopic disorders. However, AD can follow asthma and AR. FA develops early in life, can occur before or after AD, and in some cases can be the first sign of an atopic march.

The actual pathogenesis of bronchial allergies and AD isn't always completely understood. Both illnesses are related to continual inflammation. In bronchial allergies sufferer's cytokines and different inflammatory mediators are observed in bronchial washings. Both illnesses may be IgE- mediated which shows genetic predisposition as atopy refers back to the familial tendency to supply Ig-E. Imbalance withinside the Th1/Th2 ratio is related to better manufacturing of IgE in atopic sufferers. By generating Il-2 and Il-13, Th2 cells sell the manufacturing of IgE with the aid of using B-cells in reaction to antigen trigger.

In addition to allergic comorbidities, AD and asthma are associated with non-allergic diseases. Comorbidities of non-allergic AD consist of skin and extra cutaneous infections, neuropsychiatric disorders, obesity, cardiovascular disease, and some cancers. Interventions to reduce the severity of childhood illness suggest that they may have protective functions against the development of these comorbidities.

Skin barrier

Disruption of the skin barrier is a major factor in the development of atopic diseases, as trans epidermal penetration of antigens leads to sensitization. For this reason, AD almost always precedes asthma. Allergic asthma is the most common type of asthma and is usually defined by the presence of sensitization to environmental allergens.

Microbiome

The microbial flora plays an important role in both health and disease because it is involved in the development of the immune system and the development of allergic diseases. Since the "Hygiene Hypothesis" in the 1980s, there has been increasing research linking the microbiota of the skin, gastrointestinal tract, and airways to allergic diseases.

Therapy

Many new treatment options focused on specific components of the immunological pathway of bronchial allergies and AD have already been evolved and are available, or are in system of development. Dupilumab, blockading IL-4 and Il-13 from binding to its receptors, become the primary biologic accredited for mild to intense AD, and Omalizumb, anti- IgE monoclonal

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antibody, become the primary biologic accredited for the remedy of bronchial allergies withinside the United States and the European Union.

CONCLUSION

There is a strong link between asthma and AD. Systemic factors such as genetics and Th2 immune disorders, as well as tissue-specific factors such as local immune response, barrier dysfunction, abnormal microbial flora, and environmental triggers, contribute to an increased risk of comorbid atopic disease.