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Correlations Between Clinical and Laboratory Parameters in Virus-Associated Allergodermatoses in Children of Different Age Groups

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Abstract **Introduction.** Allergodermatoses, such as Atopic Dermatitis (AD), are chronic inflammatory skin diseases involving complex immune dysregulation. **Methods and Materials.** A clinical and laboratory study was performed in children with atopic dermatitis diagnosed by standardized criteria (e.g., Hanifin–Rajka and/or validated national guidelines) and clinical suspicion of virus-associated skin involvement. **Results.** In the 0–12 months group, IL-4 showed a moderate positive correlation with anti-herpesvirus IgM (r = 0.42) and was accompanied by reduced mucosal immunity (lysozyme decreased 2.5-fold, sIgA decreased 1.5-fold) and increased pro-inflammatory mediators (IL-4 increased 3.9-fold, IL-17A increased 215-fold, MCP-1 increased 6.4-fold) versus controls. **Conclusion.** Immune parameters in saliva and serum demonstrated age-dependent correlation patterns in children with virus-associated AD features. The marker combinations (IL-4/sIgA in infants, IgE in 1–3 years, and IgE/MCP-1 in 4–5 years) emerge as candidate diagnostic panels for further validation.

Keywords Virus-associated allergodermatoses, Atopic dermatitis, Children, Age groups, Immunological parameters, Correlation analysis, Cytomegalovirus (CMV), Herpes virus, Immunoglobulin E (IgE), Interleukin-4 (IL-4), Secretory IgA (sIgA), MCP-1, Diagnostic markers

1. Introduction

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by epidermal barrier impairment and immune dysregulation [2,5,8]. Barrier disruption may facilitate transepidermal sensitization and is also associated with increased susceptibility to cutaneous viral infections in children. However, age-related patterns of association between systemic/mucosal immune markers and herpesvirus /cytomegalovirus (CMV) serological parameters in children with AD remain insufficiently described [1,4,6,10]. This study presents an exploratory, correlational analysis of immune parameters in saliva and serum in children with AD and virus-associated clinical features to identify candidate age-specific diagnostic markers and generate hypotheses for future mechanistic studies [3,7,9].

Aim. To explore age-specific correlations between immunological parameters in saliva and serum and to identify candidate diagnostic markers for virus-associated AD across three age groups: 0–12 months, 1–3 years, and 4–5 years.

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2. Methods and Materials

A clinical and laboratory observational study with a cross-sectional, exploratory design was conducted in children diagnosed with atopic dermatitis (AD) according to standardized diagnostic criteria (Hanifin–Rajka criteria and/or validated national clinical guidelines). Only patients with clinically confirmed AD and signs suggestive of virus-associated skin involvement (e.g., recurrent herpetic eruptions, prolonged inflammatory exacerbations, or documented viral seropositivity) were included. Exclusion criteria comprised primary immunodeficiency, systemic autoimmune disease, chronic systemic infections, and recent vaccination within four weeks prior to sampling. Participants were stratified into three predefined age groups: infants (0–12 months), young children (1–3 years), and children aged 4–5 years, and were compared with an age- and sex-matched control group of clinically healthy children without allergic or infectious skin disease.

Clinical characterization of patients included a detailed dermatological examination and standardized assessment of disease severity using validated scoring systems such as SCORAD and/or EASI, depending on age and feasibility. Data on disease duration, frequency of exacerbations, family history of atopy, and comorbid allergic conditions were recorded. Current and recent treatments were documented in

detail, including the use of topical corticosteroids, topical calcineurin inhibitors, emollients, systemic therapies, and antiviral medications. To minimize treatment-related bias, biological sampling was performed during a clinically stable phase or prior to initiation or escalation of systemic therapy when possible.

Venous blood samples were collected under standardized conditions and processed according to manufacturer protocols. Serum immunological parameters included total immunoglobulin E (IgE), virus-related antibodies (IgM and IgG to cytomegalovirus and herpesviruses, with subtype specification where available), and cytokines associated with allergic and inflammatory responses (IL-4, IL-17A, and MCP-1). Quantification was performed using enzyme-linked immunosorbent assay (ELISA) or validated immunoassay techniques with commercially available kits, following strict quality control procedures. Given known limitations of viral IgM serology, results were interpreted as indicators of exposure or immune response rather than definitive evidence of active infection.

To assess mucosal immunity, unstimulated saliva samples were collected in the morning under fasting conditions. Levels of secretory immunoglobulin A (sIgA) and lysozyme were measured using standardized biochemical or immunoenzymatic methods. Saliva was chosen as a non-invasive biological matrix reflecting local immune defense, particularly relevant in pediatric populations. Samples with visible blood contamination or insufficient volume were excluded from analysis.

Statistical analysis was performed using validated statistical software. Data distribution was assessed using normality tests (e.g., Shapiro–Wilk). Depending on distribution characteristics, Pearson's or Spearman's correlation coefficients were applied to evaluate associations between immunological parameters. Between-group comparisons were conducted using appropriate parametric or non-parametric tests. To reduce the risk of false-positive findings, multiple comparison correction was applied using the Benjamini–Hochberg false discovery rate approach. Exploratory multivariable analyses were planned and/or conducted to adjust for potential confounders, including age, disease severity, and treatment status. A p-value <0.05 was considered statistically significant, with results interpreted in an exploratory, hypothesis-generating context.

3. Results

In the 0–12 months group, IL-4 showed a moderate positive correlation with anti-herpesvirus IgM ($r = 0.42$) and was accompanied by reduced mucosal immunity (lysozyme decreased 2.5-fold, sIgA decreased 1.5-fold) and increased pro-inflammatory mediators (IL-4 increased 3.9-fold, IL-17A increased 215-fold, MCP-1 increased 6.4-fold) versus controls. In the 1–3 years group, total IgE demonstrated a strong positive correlation with IL-4 ($r = 0.67$) and inverse correlations with anti-CMV IgM ($r = -0.69$) and anti-herpesvirus IgM ($r = -0.84$); this group also showed a marked reduction in sIgA (3.5-fold decrease) and elevated IL-17A (285-fold increase) versus controls. In the 4–5 years group, an association pattern more consistent with CMV serology was observed: IgE correlated negatively with anti-CMV IgM ($r = -0.98$) and positively with anti-CMV IgG ($r = 0.90$), along with higher MCP-1 (increase 7.3-fold) and reduced sIgA (3.0-fold decrease) compared with controls. These findings identify candidate age-linked marker sets: IL-4 and sIgA (0–12 months), total IgE (1–3 years), and IgE with MCP-1 (4–5 years), recognizing that these are exploratory associations.

4. Discussion

This exploratory correlation analysis suggests that the immune-marker association structure in virus-associated AD may vary by age. Across age strata, reduced mucosal immunity (sIgA/lysozyme) co-occurred with elevated cytokine signals (IL-4, IL-17A, MCP-1), and distinct patterns of association with viral serology were observed. Importantly, serological IgM results-especially for CMV-have known interpretive limitations (e.g., persistence, cross-reactivity, non-specific positivity), and therefore the observed correlations should be interpreted cautiously without assuming active infection. The extremely large fold changes for IL-17A warrant careful verification of assay units, detection limits, outliers, and analytical scaling (e.g., log-transformation), and should be replicated in independent cohorts. Overall, the data are hypothesis-generating and support future studies integrating virological confirmation (PCR and/or IgG avidity), disease severity metrics, and multivariable modeling.

5. Conclusions

Immune parameters in saliva and serum demonstrated age-dependent correlation patterns in children with virus-associated AD features. The marker combinations (IL-4/sIgA in infants, IgE in 1–3 years, and IgE/MCP-1 in 4–5 years) emerge as candidate diagnostic panels for further validation. Larger, well-characterized cohorts with standardized AD diagnosis, severity scoring, treatment stratification, and confirmatory viral testing are required to confirm these associations and assess clinical utility.

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