REVIEW ARTICLE

Environmental and Lifestyle Determinants of Early-Life Atopic Dermatitis: A Narrative Review of Cord Blood IgE as a Predictive Marker

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Abstract

Background and objective: Atopic dermatitis (AD) emerges in infancy and poses a considerable burden on families and early identification of relevant risk factors is essential, among which the cord blood immunoglobulin E (IgE) can be a potential biomarker. The present article reviews studies published between 2014 and 2024 on the relationship between cord blood IgE and the risk of developing AD during the first year of life.

Key Findings: Most studies show that neonates with elevated cord blood IgE demonstrate a higher likelihood of experiencing AD within their initial year. Overall findings highlight that elevated cord blood IgE may signal heightened immune activation in utero. Genetic predisposition and environmental exposures such as maternal smoking and pet contact frequently act as confounders or effect modifiers. But differences in IgE measurement and cutoff thresholds hamper direct comparisons among studies, necessitating greater standardization.

Conclusion: Elevated cord blood IgE indicates the possibility of using it as a marker to identify neonates at risk for atopic dermatitis. However, the clinical utility of cord blood IgE alone remains constrained by methodological heterogeneity and the multifactorial nature of AD. Carefully-refined studies may examine gene-environment interactions, and investigate preventative interventions. Also, cord blood IgE testing may become one component of a broader neonatal risk stratification to lessen early-onset allergic diseases.

Keywords: Atopic Dermatitis, Cord Blood Immunoglobulin E (IgE), Neonates, Allergic Diseases, Allergy Incidence, Predictive Biomarkers, First Year of Life.

1. Introduction

Atopic dermatitis (AD) is among the most prevalent allergic disorders presenting in the first year of life, often signaling as more complex atopic manifestations such as asthma or allergic rhinitis later on [1]. Its chronic, relapsing nature imposes significant personal, social, and economic burdens on affected infants and their families. In addition, there is a growing emphasis on identifying neonates at high risk for allergic conditions, with the goal of applying targeted preventive strategies or interventions.

Cord blood immunoglobulin E (IgE) has received attention as a potential early-life biomarker of allergic propensity. IgE lies at the center of type I hypersensitivity processes, playing a central role in numerous atopic conditions [2]. Detectable IgE in cord blood partly reflects fetal immune activation, and has been reported to forecast subsequent asthma, allergic rhinitis, and atopic dermatitis [3]. Such early sensitization might be influenced by maternal atopy, prenatal allergen exposure, and other environmental or immunologic factors [4]. Consequently, neonates with elevated cord blood IgE could be predisposed to severe immune responses upon postnatal encounters with common allergens.

Despite this intriguing possibility, the current literature reflects a spectrum of findings. Some studies have identified a strong, positive association between cord blood IgE and AD, whereas others have reported weaker or even null associations, highlighting methodological discrepancies and population-specific factors. The objective of the present review is to provide readers with a coherent synthesis of research published between 2014 and 2024 on the relationship between cord blood IgE levels and AD in infancy, concentrating on key insights, controversies, and implications for clinical practice and research in future.

2. Relevance of Atopic Dermatitis in Early Life

Atopic dermatitis often appears within months of birth and can significantly interfere with growth, sleep quality, and overall wellbeing [1]. Its incidence has risen almost all over the world, potentially due to changing global environmental exposures and shifts in microbiome diversity. In many children, AD serves as the initial stage in an “atopic march” that may evolve into allergic rhinitis or asthma over time [5]. Recognizing those at elevated risk is thus a priority for pediatric healthcare.

3. Cord Blood IgE as an Early Biomarker

In general, IgE plays an integral role in establishing allergic inflammatory pathways by binding allergens and initiating cell degranulation. Infants do not typically inherit large quantities of maternal IgE due to restricted transplacental transfer; that’s why we believe that the cord blood IgE is predominantly produced by the fetus [6,7]. Evidence suggests that prenatal exposures, ranging from maternal diet and environmental allergens to cytokine transfer, are likely to sensitize the developing immune system in utero [4]. Consequently, measurable IgE in cord blood might imply an immunologic “head start” toward allergic hypersensitivity.

4. Association of Elevated Cord Blood IgE with AD in the First Year

A substantial portion of studies (published from 2014 to 2024) report that neonates with above-threshold cord blood IgE (commonly 0.3–1.0 kU/L) carried significantly higher odds or relative risks of developing AD within the initial year of life. In many cohorts,

the likelihood of early AD was reported to be nearly doubled in infants classified as having elevated IgE. For instance, odds ratios ranging between 1.5 and 4.2 have been documented, suggesting variability in effect sizes across different populations [3,8].

However, not all reports consistently confirmed such a positive correlation. Studies that failed to find such significant associations often cited small sample sizes or heterogeneous IgE assay methods as limitations. In addition, differences in diagnostic criteria for AD (some employing clinical scoring systems, while others relied on parental self-report) complicate direct comparisons. This heterogeneity emphasizes the need for standardized assays and clearer diagnostic protocols.

5. Confounding Factors and Effect Modifiers

Multiple elements beyond cord blood IgE alone shape the onset of atopic dermatitis. Chief among them is parental atopy; infants with one or both atopic parents are more likely to show an amplified correlation between cord blood IgE and AD [9,10]. Maternal smoking, pet exposure, and mode of delivery (cesarean vs. vaginal birth) are other major confounders considered frequently in some studies. Several investigations also point to exclusive breastfeeding as a potential modifier of risk, though findings are somewhat mixed and diversly reported [11]. Some data further suggest that exclusive breastfeeding may underestimate the impact of elevated cord blood IgE, whereas others demonstrate some limited influence depending on genetic predisposition.

6. Biological Foundations

Although the precise mechanisms require further examination, fetal immune programming likely involves cytokine and chemokine mediators transferring from the maternal circulation to the fetus, in addition to direct fetal immunocyte stimulation [4,6]. High levels of interleukins such as IL-4 and IL-13 are particularly implicated in Th2-driven (allergy-associated) immune polarization [8,9]. The presence of elevated IgE at birth may thus represent an “initiated” Th2 bias, priming the neonate’s immune system toward skin inflammation and barrier dysfunction characteristic of AD. Further examination of epigenetic processes and genomic variants could elucidate our conceptions of why some high-IgE neonates never develop AD, while others proceed towards severe disease.

7. Measurement Variability and Cutoff Values

Despite the growing body of literature supporting the predictive value of cord blood IgE, consensus on the optimal cutoff still remains controversial. Assays such as ImmunoCAP, enzyme-linked immunosorbent assay (ELISA), and radioimmunoassay each yield slightly different calibration ranges and detection limits [3]. Reported thresholds for elevated cord blood IgE often fluctuate between 0.3 and 1.0 kU/L. This inconsistency in turn complicates comparisons between different studies and highlights a need for harmonizing measurement techniques. Such standardization may demand definitive meta-analyses and enhanced clarification regarding the role of IgE in neonatal allergy risk assessment.

8. Clinical and Research Implications

From a clinical perspective, cord blood IgE testing may find a place to serve as part of a broader perinatal risk profiling strategy for atopic dermatitis. By identifying neonates at higher risk, pediatricians might initiate proactive measures, including skincare

regimens aimed at preserving epidermal barrier function, specialized feeding protocols, and closer monitoring by allergists or dermatologists. However, we have further concerns about cost-effectiveness, as well as the potential for false negatives and false positives, because not all neonates with low IgE remain free of AD and not all with high IgE manifest disease.

We would like to recommend researchers to embark on refining the consensus IgE cutoff points, elucidating mechanistic pathways, and conducting larger cohort studies to supervise what may occur beyond the first12 months of life to capture the dynamic nature of atopic disorders longitudinally. Such efforts may examine how gene-environment interactions, microbiome profiles, and maternal health factors intersect with cord blood IgE to influence the onset and trajectory of atopic dermatitis.

9. Discussion

Evidence from the past decade reinforces the biological likelihood of cord blood IgE as an indicator of early immunologic priming and elevated risk of atopic dermatitis. For infants with additional risk factors (i.e., parental atopy or adverse environmental exposures), this association has been found to be especially pronounced. Nevertheless, both clinical and research spheres face persistent challenges. Heterogeneous IgE assays, variable AD definitions, and incomplete adjustment for confounders limit the comparability of existing study reports and results. These methodological gaps partly explain why study results differ, with effect sizes spanning from marginal to robust.

Given the multifactorial etiology of AD, we feel that it is yet unlikely that cord blood IgE alone can fully explain disease onset. Alternatively, it may interact with genetic predisposition and environmental stimuli in a broader atopic diathesis. In clinical practice, it earnestly calls for careful validation of IgE cutoffs, cost-effectiveness evaluations, and well-defined management pathways for infants testing “high” at birth. It seems that incorporating novel approaches (e.g., combining cord blood IgE with maternal biomarkers or early skin barrier measurements) may yield more accurate composite indices of neonatal atopic risk.

10. Conclusion

This narrative review highlights the potential significance of cord blood IgE levels as a contributing marker for atopic dermatitis risk in the first year of life. While most studies point to a positive correlation, methodological variability partly complicates assertive and definitive statements about its predictive accuracy. However, elevated cord blood IgE provides a biologically plausible window into fetal immune conditioning and neonatal susceptibility to allergic skin inflammation. Ongoing efforts to standardize assays, refine diagnostic criteria, and identify pertinent gene-environment interactions can be considered as a key to clarifying whether cord blood IgE can function as a reliable indicator in clinical settings or not. By planning individualized prevention strategies, better risk stratification has the potential to reduce the burden of atopic dermatitis and potentially help control its progression in later childhood.

Ethical Considerations

No primary data involving human or animal subjects were collected, and all references are duly cited.

Conflict of Interest

The authors declare no conflicts of interest.

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