Elite Journal of Immunology. Volume 2 Issue 6(2024), Pp. 15-25
https://epjournals.com/journals/EJI

An update on Early Immunological Markers in HIV-Exposed Infants

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Abstract

Early immunological markers in HIV-exposed infants play a critical role in understanding immune development, assessing HIV infection risk, and guiding therapeutic interventions. This review synthesizes current knowledge on innate and adaptive immune responses, markers of immune activation, and their implications for HIV-exposed infants' health outcomes. Insights into these markers offer opportunities for early diagnosis, initiation of antiretroviral therapy (ART), and interventions to optimize immune function in this vulnerable population. The innate immune responses of HIV-exposed infants are shaped by the interplay between maternal HIV exposure and immune system maturation. Natural killer (NK) cells, monocytes, and dendritic cells (DCs) undergo developmental changes that affect their ability to recognize and respond to viral infections. Altered NK cell function and reduced innate immune surveillance may predispose exposed infants to heightened infection risk and influence HIV pathogenesis. Understanding these early innate immune markers is crucial for identifying vulnerabilities and designing targeted interventions to enhance immune defenses. Adaptive immune responses in HIV-exposed infants are characterized by variations in CD4+ T cell counts, T cell activation markers, and cytokine profiles. These factors influence the efficacy of immune responses against HIV and impact disease progression. Elevated levels of immune activation markers, such as soluble CD14 and CD38 expression on T cells, are associated with increased susceptibility to HIV infection and disease progression in exposed infants. Early identification of these markers informs clinical decision-making, facilitates early intervention strategies, and monitors immune reconstitution during ART, thereby improving long-term health outcomes in HIV-exposed infants.

Keywords: *HIV-exposed infants, early immunological markers, immune development, HIV infection, pediatric HIV/AIDS*

Introduction

Citation: Obeagu EI, Obeagu GU. An update on Early Immunological Markers in HIV-Exposed Infants**.** Elite Journal of Immunology, 2024; 2(6): 15-25

The perinatal transmission of HIV remains a significant global health challenge, particularly affecting infants born to HIV-positive mothers in resource-limited settings. Despite substantial progress in prevention of mother-to-child transmission (PMTCT) programs, approximately 150,000 infants acquire HIV annually, highlighting the critical need for effective management strategies from early infancy onwards.[1] HIV-exposed infants face unique immunological challenges due to their exposure to HIV antigens in utero, during delivery, and through breastfeeding. These exposures shape the infant's immune system development from the earliest stages of life, influencing innate and adaptive immune responses. The timing and duration of HIV exposure, maternal viral load, and maternal ART use during pregnancy profoundly impact the infant's immune environment and subsequent susceptibility to HIV infection and disease progression. Therefore, investigating early immunological markers in HIV-exposed infants is essential for elucidating immune pathways involved in HIV pathogenesis and for identifying predictive biomarkers of infection risk and disease progression.[2-3] The innate immune system serves as the first line of defense against pathogens, playing a crucial role in early immune responses in HIV-exposed infants. Natural killer (NK) cells, monocytes, macrophages, and dendritic cells (DCs) are integral components of innate immunity that undergo developmental changes influenced by maternal HIV exposure. Altered NK cell function, characterized by reduced cytotoxicity and impaired cytokine production, has been observed in HIV-exposed infants, potentially compromising their ability to control viral replication and mount effective immune responses.[4]

Adaptive immune responses in HIV-exposed infants are characterized by variations in CD4+ T cell counts, T cell activation markers, and cytokine profiles. CD4+ T cells play a central role in orchestrating immune responses against HIV, and their depletion is a hallmark of disease progression in untreated infants. Early immune activation, evidenced by elevated levels of activation markers such as CD38 on T cells and soluble immune factors like IL-6 and TNF-α, is associated with increased susceptibility to HIV infection and rapid disease progression in exposed infants. These immunological markers provide critical insights into the immune dysregulation and potential targets for therapeutic interventions aimed at restoring immune homeostasis and improving clinical outcomes.[5-6] Markers of immune activation also reflect systemic inflammation and immune dysregulation observed in HIV-exposed infants, contributing to long-term health implications beyond immediate HIV infection. Persistent immune activation is linked to accelerated immune senescence, premature aging of the immune system, and increased susceptibility to non-AIDS-related morbidities, such as cardiovascular disease and neurocognitive impairment. [7-8] Furthermore, the impact of maternal factors, including maternal HIV viral load, CD4+ T cell counts, and use of ART during pregnancy, influences the infant's immune environment and subsequent risk of HIV transmission. Maternal ART reduces maternal viral load and decreases the risk of vertical transmission but may also influence fetal immune development and the establishment of immune tolerance or activation in exposed infants. Investigating the interplay between maternal and infant immune responses provides critical insights into the factors shaping immune development and disease susceptibility in HIV-exposed infants.[9-10] The developmental trajectory of immune responses in HIV-exposed infants is further influenced by factors such as breastfeeding practices and exposure to other pathogens and environmental factors.

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Breastfeeding, while beneficial for overall infant health, presents a risk of postnatal HIV transmission and may modulate infant immune responses and the gut microbiome, impacting immune development and infection risk. Environmental factors, including nutritional status, socioeconomic conditions, and co-infections such as tuberculosis and malaria, also contribute to immune modulation and disease susceptibility in exposed infants. [11-12]

Innate Immune Responses

Innate immune responses play a crucial role in the early defense against pathogens, including HIV, in HIV-exposed infants. This initial line of immune defense is particularly important in the context of perinatal HIV transmission, where the timing and quality of innate immune responses can influence the establishment of infection and subsequent disease progression. [13-14] NK cells are critical components of innate immunity that contribute to early antiviral defense through their ability to directly kill infected cells and produce cytokines such as interferon-gamma (IFN-γ). In HIV-exposed infants, alterations in NK cell function have been observed, characterized by reduced cytotoxicity and impaired cytokine production, which may compromise their ability to control viral replication and contribute to increased susceptibility to infections. The developmental immaturity of NK cells in neonates further complicates their response to HIV exposure, highlighting the need to better understand factors influencing NK cell development and function in early life.[15-16] Monocytes and macrophages are crucial in innate immune responses against HIV, serving as reservoirs for viral replication and contributing to immune activation and inflammation. In HIV-exposed infants, monocytes/macrophages may exhibit altered phenotypes and functions, including enhanced inflammatory responses and increased susceptibility to HIV infection due to the expression of HIV co-receptors. These cells also play roles in antigen presentation and cytokine production, influencing the adaptive immune responses and shaping the overall immune environment in exposed infants.[17-18]

Dendritic cells (DCs) are key antigen-presenting cells that bridge innate and adaptive immunity by capturing and presenting antigens to T cells, initiating adaptive immune responses against HIV. DCs in HIV-exposed infants may exhibit dysregulated functions, characterized by impaired maturation, reduced ability to stimulate T cells, and altered cytokine secretion profiles. Dysfunctional DC responses may compromise the generation of effective adaptive immune responses against HIV and other pathogens, contributing to immune evasion strategies employed by the virus.[19-20] Furthermore, innate immune responses in HIV-exposed infants are influenced by maternal factors, including maternal HIV viral load, maternal ART use, and breastfeeding practices. Maternal viral load directly impacts the level of viral exposure to the infant in utero and during delivery, influencing the early immune activation and immune development in exposed infants. Maternal ART, while essential for reducing perinatal transmission risk, may also modulate infant immune responses through direct drug exposure or indirect effects on maternal immune function. Breastfeeding, a common practice with benefits for infant nutrition and immunity, introduces additional factors influencing infant immune development and exposure to HIV and other pathogens.[21-22]

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Adaptive Immune Responses

Adaptive immune responses in HIV-exposed infants are critical in shaping the course of infection and influencing long-term immune outcomes. These responses involve a complex interplay of cellular and humoral components aimed at recognizing and mounting specific immune responses against HIV antigens. Central to adaptive immunity are CD4+ T cells, which orchestrate immune responses by coordinating cellular and humoral immunity. In HIV-exposed infants, the functionality and numerical distribution of CD4+ T cells are influenced by various factors, including the timing of HIV exposure, maternal viral load, and maternal antiretroviral therapy (ART) use. Early depletion of CD4+ T cells is a hallmark of HIV infection progression in infants, compromising immune responses and increasing susceptibility to opportunistic infections. Monitoring CD4+ T cell counts and their dynamics provides critical insights into immune status and guides decisions regarding ART initiation and management in exposed infants.[23-25] CD8+ T cells play a crucial role in immune surveillance and the elimination of HIV-infected cells through cytotoxicity and the release of antiviral cytokines. Effective CD8+ T cell responses are essential for controlling viral replication and reducing HIV reservoirs in exposed infants. However, HIV-specific CD8+ T cell responses may be impaired or delayed in HIV-exposed infants due to immune immaturity and ongoing viral exposure. Strategies aimed at enhancing CD8+ T cell function and promoting the generation of broadly neutralizing antibodies are under investigation to improve immune control and potentially achieve viral remission in infected infants.[26-27]

B cell responses in HIV-exposed infants are crucial for the production of HIV-specific antibodies and the development of humoral immunity. B cell dysfunction, characterized by impaired antibody production and reduced affinity maturation, may compromise vaccine responses and limit the effectiveness of antibody-mediated immune responses against HIV. Early identification of B cell dysfunction and strategies to enhance B cell function are essential for improving vaccine-induced immunity and immune surveillance in exposed infants.[28-29] Markers of immune activation, such as CD38 expression on T cells and elevated levels of pro-inflammatory cytokines, provide insights into the degree of immune dysregulation and disease progression in HIV-exposed infants. Persistent immune activation contributes to immune exhaustion, accelerated immune senescence, and increased susceptibility to non-AIDS-related comorbidities in later life. Strategies aimed at mitigating chronic immune activation and restoring immune homeostasis are crucial for improving long-term health outcomes and reducing the burden of HIV-related morbidity in exposed infants.[30-31] Moreover, the interplay between innate and adaptive immune responses in HIV-exposed infants is complex and interconnected, influencing the overall immune environment and susceptibility to infections. Dysregulated immune activation, immune exhaustion, and impaired immune reconstitution following ART initiation underscore the challenges in achieving durable viral remission or functional cure in exposed infants. Comprehensive immune monitoring and personalized therapeutic approaches are essential for optimizing ART management, enhancing immune recovery, and improving clinical outcomes in this vulnerable population.[32-33]

Markers of Immune Activation

Markers of immune activation in HIV-exposed infants serve as crucial indicators of immune dysregulation, disease progression, and potential therapeutic targets. These markers encompass a spectrum of immune activation pathways, including cellular activation markers on T cells, cytokine profiles, and systemic inflammation, which collectively influence the course of HIV infection and immune responses in exposed infants.[34-35] One prominent marker of immune activation is the expression of CD38 on T cells, particularly CD8+ T cells, which is upregulated in response to antigenic stimulation and reflects ongoing immune activation. Elevated CD38 expression on T cells has been consistently associated with HIV disease progression and poor clinical outcomes in both adults and pediatric populations. In HIV-exposed infants, increased CD38 expression on T cells correlates with higher viral loads and decreased CD4+ T cell counts, highlighting its utility as a prognostic marker for monitoring disease severity and guiding therapeutic interventions.[36-37] Soluble immune factors, such as soluble CD14 (sCD14) and markers of systemic inflammation (e.g., interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α)), provide additional insights into immune activation and inflammatory responses in HIV-exposed infants. sCD14, a biomarker of monocyte activation, is elevated in HIV-infected individuals and correlates with increased microbial translocation, systemic inflammation, and disease progression. In exposed infants, elevated sCD14 levels reflect heightened immune activation and may contribute to immune dysfunction and susceptibility to infections.[38-39]

Cytokine profiles in HIV-exposed infants also play a critical role in immune activation and regulation. Pro-inflammatory cytokines, such as IL-6, TNF-α, and interferon-gamma (IFN-γ), are elevated during HIV infection and contribute to chronic immune activation, T cell dysfunction, and accelerated disease progression. Dysregulated cytokine production in exposed infants may impair immune responses, compromise viral control, and contribute to the development of immune-mediated pathologies.[40-41] Markers of immune activation not only serve as diagnostic and prognostic indicators but also offer potential therapeutic targets for mitigating immune dysregulation and improving clinical outcomes in HIV-exposed infants. Strategies aimed at reducing immune activation, such as immune modulatory therapies and anti-inflammatory agents, hold promise for restoring immune homeostasis, enhancing immune responses to HIV, and reducing the risk of non-AIDS-related comorbidities. Combinatorial approaches targeting both viral replication and immune activation pathways may offer synergistic benefits in achieving durable viral remission and improving long-term health outcomes in this vulnerable population.[42-43] Moreover, the impact of maternal factors, including maternal HIV viral load, ART use during pregnancy, and breastfeeding practices, influences immune activation markers in exposed infants. Maternal ART reduces maternal viral load and decreases vertical transmission risk but may also modulate fetal immune development and alter the immune activation profile in exposed infants.[44]

Clinical Implications

The clinical implications of immune activation markers in HIV-exposed infants are profound, guiding both diagnostic strategies and therapeutic interventions aimed at optimizing health outcomes in this vulnerable population.[45] Firstly, immune activation markers, such as CD38 expression on T cells and levels of soluble immune factors like sCD14, serve as valuable tools for

early diagnosis of HIV infection in exposed infants. Elevated CD38 expression on CD8+ T cells is associated with increased viral replication and decreased CD4+ T cell counts, making it a sensitive marker for identifying infants at higher risk of HIV disease progression. Early detection allows for prompt initiation of antiretroviral therapy (ART), which is crucial for suppressing viral replication, preserving immune function, and improving long-term outcomes.[46-47] Monitoring immune activation markers during ART provides critical insights into treatment responses and immune reconstitution in HIV-exposed infants. ART effectively reduces viral load and decreases immune activation markers, such as CD38 expression and levels of pro-inflammatory cytokines, thereby mitigating chronic immune activation and inflammation. Regular monitoring of these markers informs clinicians about treatment efficacy, adherence to therapy, and the need for adjustments in ART regimens to achieve sustained viral suppression and optimize immune recovery.[48-49]

Furthermore, immune activation markers serve as prognostic indicators of disease progression and predictors of non-AIDS-related comorbidities in HIV-exposed infants. Elevated levels of sCD14 and pro-inflammatory cytokines are associated with increased risk of immune dysfunction, accelerated immune senescence, and heightened susceptibility to infections and chronic diseases later in life. Early identification of these markers allows for early intervention strategies aimed at reducing immune activation, preserving immune function, and improving overall health outcomes in exposed infants.[50-51] Incorporating immune activation markers into routine clinical care facilitates personalized medicine approaches tailored to individual patient needs. Comprehensive immune monitoring helps clinicians assess immune status, predict treatment responses, and identify infants at higher risk of HIV-related complications. This personalized approach optimizes clinical management strategies, improves patient outcomes, and reduces healthcare disparities in resource-limited settings where pediatric HIV/AIDS burden is disproportionately high.[52-55]

Moreover, research into novel therapeutic strategies targeting immune activation pathways holds promise for improving outcomes in HIV-exposed infants. Interventions aimed at modulating immune activation, such as immune modulatory therapies and anti-inflammatory agents, complement ART by addressing immune dysregulation and reducing the risk of immune-mediated pathologies. Combining these therapeutic approaches with early initiation of ART and comprehensive pediatric care enhances the potential for achieving durable viral remission and improving long-term health outcomes in this vulnerable population.[56-60]

Conclusion

The innate immune responses, mediated by NK cells, monocytes/macrophages, and dendritic cells, lay the foundation for early defense against HIV and other pathogens. Adaptive immune responses, particularly the dynamics of CD4+ and CD8+ T cells, along with B cell function and antibody responses, are crucial determinants of HIV disease progression and treatment outcomes. Markers of immune activation, such as CD38 expression on T cells and cytokine profiles, serve as sensitive indicators of immune dysregulation and predictors of disease severity in exposed infants. Early identification of these markers facilitates timely intervention with antiretroviral therapy and

personalized management strategies aimed at preserving immune function and reducing long-term morbidity. The clinical implications of immune activation markers extend beyond diagnosis to include treatment monitoring, prognostication, and the development of targeted therapies. Regular monitoring of immune activation markers guides therapeutic decisions, informs patient management strategies, and supports efforts to achieve durable viral remission in HIV-exposed infants.

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