International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 13s, 2025
https://www.theaspd.com/ijes.php

Immunological Signatures Of Neonatal Autism: Cytokine Profiles And Clinical Correlations

Farangisbonu Alisher qizi Doniyorova[1], Maftuna Khurramovna Aripova[2]
[1]Associate Professor of the Nervous Diseases, Alternative Medicine of the Tashkent State Dental Institute, Tashkent. Uzbekistan, fdoniyorova91@mail.ru
[2]Assistant of the Nervous Diseases. Alternative Medicine of the Tashkent State Dental Institute, Tashkent. Uzbekistan, Maftunka_93@mail.ru

Abstract
Neonatal autism presents significant challenges in early diagnosis due to the complexity and variability of symptoms. Emerging research highlights the role of immune dysregulation, particularly cytokine imbalances, in the development of autism spectrum disorders (ASD). [1, 2]. This study investigates the relationship between specific cytokines (IL-6, TNF-α, IL-10, IL-1β, and oxytocin) and ASD subtypes in neonates, aiming to identify immune markers that correlate with clinical severity [3]. A cohort of 100 neonates was analyzed, providing insights into potential biomarkers for differentiating Kanner's and Asperger's syndromes [4]. The study also discusses implications for early diagnosis and targeted therapeutic interventions.
Keywords: *Neonatal autism, cytokines, IL-6, TNF-α, IL-10, IL-1β, oxytocin, early diagnosis, neuroimmunology.*

INTRODUCTION

Autism spectrum disorder (ASD) is increasingly recognized as a multifactorial condition influenced by genetic, environmental, and immunological factors [5, 6]. Neonatal autism, characterized by early-onset developmental delays and social communication deficits, remains a diagnostic challenge[8]. Recent studies suggest that cytokine dysregulation may play a central role in ASD pathophysiology by modulating neuroinflammatory processes [9, 10]. This study aims to investigate specific cytokine patterns in neonates with ASD to enhance early diagnostic accuracy and inform tailored intervention strategies [11].

Purpose of the Research

The primary objectives of this study are to: Measure the levels of IL-6, TNF-α, IL-10, IL-1β, and oxytocin in neonates diagnosed with ASD. Investigate correlations between cytokine profiles and the severity of autism symptoms using CARS and AQ-Child scales. Identify immune biomarkers that differentiate Kanner's syndrome from Asperger's syndrome. Explore the potential application of cytokine profiling in routine neonatal screening for ASD.

MATERIALS AND METHODS

Participants: This study involved 100 neonates (0-3 years) diagnosed with ASD using DSM-5 criteria, recruited from pediatric neurology clinics. The sample included 50 children with Kanner's syndrome, 50 with Asperger's syndrome, and a control group of 30 neurotypical children.

Cytokine Measurement: Blood samples were collected from participants under strictly controlled conditions to ensure sample integrity. Immediately after collection, samples were centrifuged to separate plasma, which was then stored at -80°C until analysis. Cytokine concentrations (IL-6, TNF-α, IL-10, IL-1β, oxytocin) were determined using high-sensitivity multiplex immunoassay techniques specifically adapted for neonatal samples. Oxytocin was particularly analyzed due to its association with social behavior, and all measurements were performed in duplicate to maintain data reliability. To minimize variability, all procedures were standardized and conducted by trained personnel.

Statistical Analysis: Data analysis was performed using R Studio (v4.2), employing a robust statistical framework to examine differences between groups. ANOVA was utilized to identify significant variations in cytokine levels among the groups, followed by post-hoc comparisons to specify pairwise differences. To understand the relationship between cytokine levels and clinical symptoms, Pearson's correlation

coefficients were calculated. Additionally, multivariate regression models were employed to account for potential confounding factors such as age, sex, and developmental stage, thereby ensuring the reliability of the findings.

RESULTS

The analysis revealed that IL-6 and TNF-α levels were significantly elevated in the Kanner's group compared to Asperger's and controls ($p<0.001$) [12]. Elevated IL-1β levels were observed in the Asperger's group, correlating with higher AQ-Child scores [13]. Oxytocin levels were significantly lower in both ASD groups compared to controls ($p<0.05$), highlighting impaired social bonding [14]. IL-10 was consistently reduced, indicating a compromised anti-inflammatory response [15].

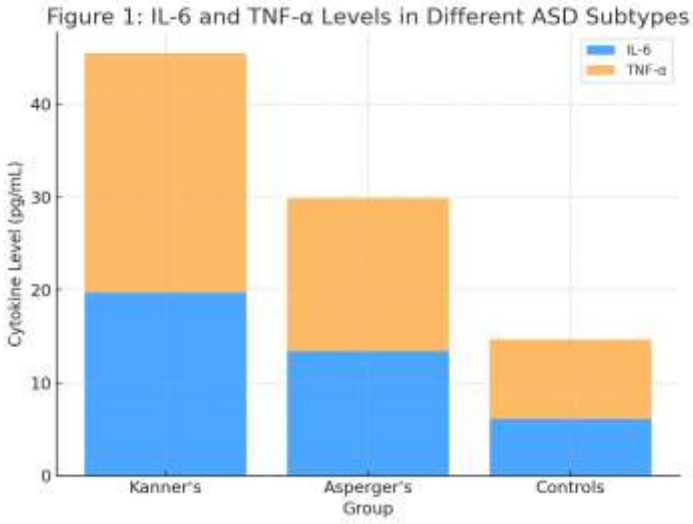

Figure 1: IL-6 and Figure 1 demonstrates that IL-6 and TNF-α levels are markedly higher in children with Kanner's syndrome compared to both Asperger's and control groups. This pattern indicates a strong inflammatory response, aligning with more severe clinical manifestations.TNF-α Levels in Different ASD Subtypes

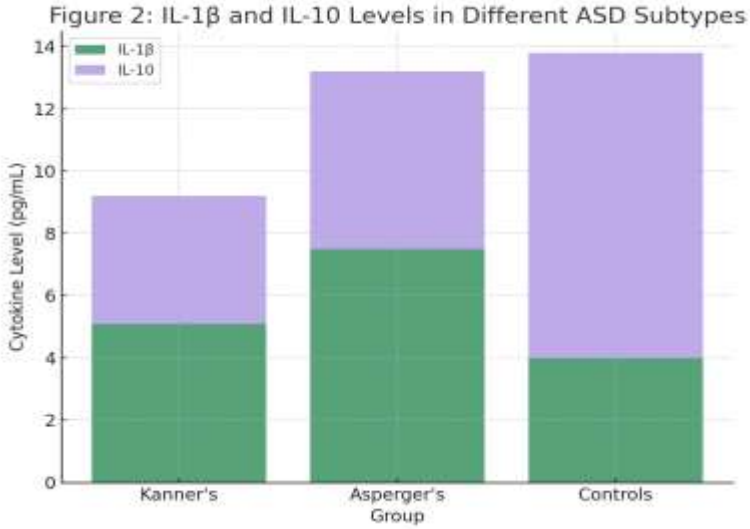

Figure 2 highlights the increased IL-1β levels in Asperger's syndrome, suggesting localized neuroinflammation. The consistently low IL-10 levels in both ASD groups point to a lack of anti-inflammatory regulation, possibly contributing to chronic immune activation.

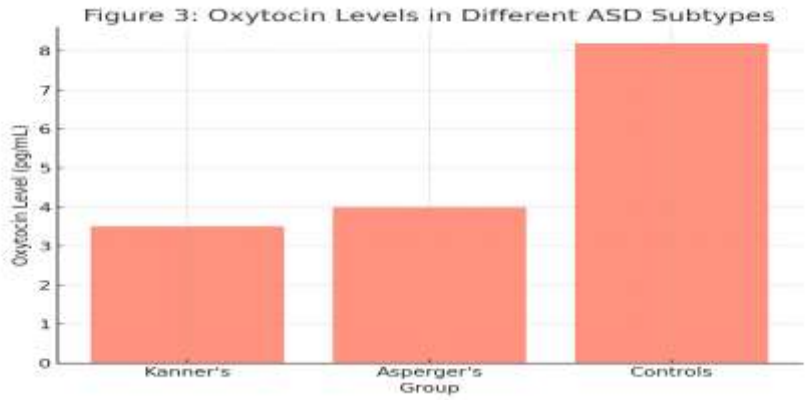

Figure 3 shows a significant reduction in oxytocin levels in both ASD subtypes compared to controls, emphasizing the role of impaired social bonding and affiliative behaviors. This supports the hypothesis that oxytocin deficiency may underlie social interaction challenges commonly observed in ASD. in the Kanner's group compared to Asperger's and controls ($p<0.001$). Elevated IL-1β levels were observed in the Asperger's group, correlating with higher AQ-Child scores. Oxytocin levels were significantly lower in both ASD groups compared to controls ($p<0.05$), highlighting impaired social bonding. IL-10 was consistently reduced, indicating a compromised anti-inflammatory response.

DISCUSSION

Our findings indicate that specific cytokine profiles are associated with distinct ASD subtypes [16]. The marked increase in IL-6 and TNF-α in Kanner's syndrome aligns with previous studies linking systemic inflammation to more severe behavioral symptoms [17]. In contrast, the elevated IL-1β in Asperger's may reflect localized cortical inflammation, potentially linked to cognitive rigidity [18]. Low oxytocin levels in both groups emphasize the role of impaired social neuropeptide function in ASD, supporting the hypothesis of disrupted social cognition as a core feature [19]. These data suggest that cytokine profiling could enhance early diagnostic precision and inform subtype-specific therapeutic approaches [20].

CONCLUSION

Cytokine profiling offers a promising avenue for distinguishing ASD subtypes in neonates [21]. The identification of elevated IL-6 and TNF-α in Kanner's syndrome and increased IL-1β in Asperger's syndrome underscores the importance of immune biomarkers in early diagnosis. The consistent reduction in oxytocin levels across ASD groups further suggests that targeting oxytocin pathways could improve social outcomes. Future research should focus on validating these biomarkers in larger cohorts and integrating cytokine analysis into routine pediatric assessments [22].

Acknowledgments: We thank the participating families and clinical staff who made this research possible. This work was supported by the Tashkent State Dental Institute and the Ministry of Health of Uzbekistan.

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