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IMMUNE STATUS CHARACTERISTICS IN CHILDREN WITH CHRONIC TONSILLITIS ASSOCIATED WITH CYTOMEGALOVIRUS INFECTION

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Abstract
The elevated incidence of cytomegalovirus (CMV) infection in the pediatric population and its role in sustaining chronic inflammatory processes represent significant challenges in modern pediatrics and otorhinolaryngology. This study presents clinical and laboratory evidence supporting CMV as an important etiological factor in the chronification and recurrent course of tonsillar disease. Given the established capacity of this viral infection to induce autoimmune reactions and modify immune system function, we systematically evaluated the immunological characteristics of chronic tonsillitis in school-aged children with active CMV, with particular focus on cytokine profiles and disturbances in humoral immunity. The results provide a foundation for improving prognostic approaches and designing targeted therapeutic interventions.

Keywords: Chronic tonsillitis, children, cytomegalovirus infection, immunological status, cytokines, humoral immunity, autoimmune processes.

Introduction

Chronic tonsillitis (CT) represents a persistent inflammatory condition of the palatine tonsils, constituting one of the most prevalent pathologies in pediatric otorhinolaryngology practice. Epidemiological data indicate that CT accounts for approximately 23.7% to 54% of all pharyngeal inflammatory diseases, with the highest incidence observed in children aged 2–15 years . The condition is characterized by recurrent exacerbations, potential for systemic complications involving the cardiovascular, renal, and musculoskeletal systems, and a complex multifactorial etiology.

The etiological landscape of CT has evolved considerably with advances in molecular diagnostics. Although group A beta-hemolytic Streptococcus (GABHS) was historically considered the predominant causative agent, it is now recognized that viral pathogens - particularly from the Herpesviridae family - play an equally significant role. Viral infections, including herpesviruses, are responsible for up to 71% of pediatric ENT pathologies [3]. Among herpesviruses, cytomegalovirus (CMV) has attracted increasing scientific attention due to its immunomodulatory properties and its capacity for long-term latent persistence in host tissues.

CMV is a ubiquitous double-stranded DNA virus that infects individuals of all age groups. Following primary infection, the virus establishes lifelong latency and may periodically reactivate, particularly

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under conditions of immune suppression. CMV exerts profound effects on both innate and adaptive immune responses: it inhibits interferon-alpha (IFN-α) production, impairs T- and B-lymphocyte function, promotes incomplete phagocytosis, and disrupts the cytokine balance. These immunosuppressive effects may predispose affected individuals to secondary bacterial infections, including CT, and may impair the resolution of existing inflammatory processes [4, 5].

CMV employs multiple strategies to evade host immunity. At the cellular level, it downregulates MHC class I expression, impairing cytotoxic T-lymphocyte recognition. It also suppresses natural killer (NK) cell activity and modulates dendritic cell maturation, thereby attenuating adaptive immune responses. At the humoral level, CMV infection is associated with elevated IgM titers during active or reactivated infection, along with decreased IgA, a critical secretory antibody for mucosal immunity [4].

The cytokine consequences of CMV infection are particularly relevant in the context of CT. CMV promotes a pro-inflammatory state characterized by elevated interleukins IL-1β, IL-6, and IL-8 - key mediators of the acute-phase response and neutrophil recruitment. Persistent elevation of these cytokines contributes to chronicity of tonsillar inflammation and may perpetuate a cycle of microbial colonization and immune dysregulation [9, 10].

The study group comprised 55 children aged 4 to 14 years with a confirmed diagnosis of simple-form CMV-associated chronic tonsillitis. Diagnosis was established on the basis of clinical criteria, pharyngoscopic findings, serological confirmation of CMV infection (ELISA for anti-CMV IgM and IgG), and PCR where applicable. A control group of 10 healthy children of comparable age and sex was enrolled.

Age distribution of the study group was as follows: 4–6 years: 23 children (41.8%); 7–10 years: 22 children (40%); 11–14 years: 10 children (18.2%). Female sex predominated in younger children (4–6 years), while male sex was more common in the 7–10 year group.

The following investigations were performed: (1) complete blood count (CBC) with leukocyte differential; (2) ELISA for anti-CMV IgM and IgG; (3) quantification of serum immunoglobulins IgM, IgG, and IgE; (4) measurement of serum C-reactive protein (CRP); (5) cytokine profiling - IL-1β, IL-6, IL-8 by ELISA before and after standard treatment; (6) microbiological culture of tonsillar surface swabs (performed in 45 patients; 10 were excluded due to concurrent antibiotic use). All assays employed internationally certified test systems with reference ranges validated for the pediatric population.

The present study contributes to a growing body of evidence implicating CMV as a pathogenically relevant co-factor in pediatric CT. Our findings demonstrate that CMV-CT is not simply a coincidental co-infection but represents a distinct clinical entity with characteristic immunological perturbations that differ from CT of purely bacterial etiology.

The high prevalence of leukopenia and neutropenia in our cohort - affecting more than half of participants - reflects the well-documented myelosuppressive effects of CMV. By contrast with bacterial CT, which typically elicits leukocytosis, the lymphocytic-monocytic pattern observed in CMV-CT may mislead clinicians toward underestimating the severity of tonsillar inflammation or toward non-targeted antibiotic prescription [4, 12].

The humoral immune findings - elevated IgM across all age groups with compensatory decline in IgG in older children - indicate progressive exhaustion of B-lymphocyte reserves with chronic CMV

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stimulation. The concurrent IgA deficit is of particular concern given its role in mucosal defense: tonsillar IgA secretion is critical for opsonization and neutralization of pathogens in the crypts, and its reduction creates a permissive environment for S. aureus and S. pneumoniae colonization [13]. This mechanistic link between CMV-induced IgA deficiency and bacterial co-colonization is directly supported by our microbiological data.

The cytokine profile observed in our patients - with approximately 2-fold elevations of IL-1β, IL-6, and IL-8 - is consistent with the "cytokine storm" pathophysiology of viral tonsillar infections. IL-1β and IL-6 drive the hepatic acute-phase response (explaining the elevated CRP), promote fever and tissue catabolism, and sustain the pro-inflammatory microenvironment that underlies tonsillar chronicity. IL-8, a potent neutrophil chemoattractant, recruits inflammatory cells to the tonsillar tissue, contributing to further structural damage despite paradoxical peripheral neutropenia [9, 10]. The significant reduction of all three cytokines post-treatment confirms the reversibility of CMV-driven inflammation with appropriate therapy but also underscores that without antiviral targeting, cytokine-driven chronicity is likely to persist.

The progressive increase in CRP with age is particularly noteworthy. In older children (11–14 years), CRP exceeded normal limits by nearly 7-fold, suggesting cumulative systemic inflammatory exposure with prolonged CMV latency. This observation has implications for the risk of tonsillogeniccomplications - including cardiac, renal, and rheumatic - which are known to be mediated by chronic systemic inflammation [14].

From a pathogenic standpoint, maternal CMV seropositivity (69.7%) in our cohort highlights vertical transmission as a key risk pathway for postnatal CMV-associated immune dysregulation. Antenatal CMV exposure programs fetal immune responses in ways that may predispose the child to recurrent infections and impaired immune tolerance [5]. These findings support the recommendation to integrate prenatal CMV screening into antenatal care protocols in Central Asian healthcare settings.

Our study has several limitations. The relatively small control group (n=10) limits the statistical power of comparative analyses. Longitudinal follow-up data are not available, precluding assessment of long-term immunological trajectories. Additionally, viral load quantification (PCR-based CMV DNA copies/mL) was not performed, limiting our ability to correlate viremia severity with immune parameters. Future research should incorporate these methodological enhancements.

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