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Exploring Cerebrospinal Fluid Protein Dynamics in Chronic HIV-1 Infection

Mia Elizabeth*

Department of Pathology, Arcadia University, USA

DESCRIPTION

Human immunodeficiency virus type 1 (HIV-1) infection remains a significant global health challenge, with approximately 37.7 million people living with HIV worldwide. While effective antiretroviral therapy (ART) has transformed HIV from a fatal to a chronic disease, neurological complications still persist, affecting around 30-50% of individuals with HIV-1 infection. Among these complications, HIV-associated neurocognitive disorders (HAND) are prevalent, encompassing a spectrum of cognitive, motor, and behavioral impairments. Understanding the pathogenesis of HAND is crucial for developing targeted therapeutic interventions. Cerebrospinal Fluid (CSF) is a vital medium for studying the Central Nervous System (CNS) in HIV-1 infection. It provides insights into the molecular and cellular changes occurring within the CNS during the course of infection. Recent studies have focused on investigating alterations in CSF proteins across different stages of HIV-1 infection, both untreated and treated, to elucidate their role in neuro-inflammation and neurodegeneration. In untreated chronic HIV-1 infection, dysregulation of CSF proteins reflects the inflammatory milieu within the CNS. Elevated levels of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) are commonly observed, indicating ongoing neuro-inflammation. Additionally, markers of neuronal injury, such as neuro-filament light chain (NFL), are increased, suggesting neuronal damage and loss. These findings underscore the detrimental effects of untreated HIV-1 infection on CNS integrity and function. With the advent of ART, there has been a significant reduction in HIV-related morbidity and mortality. However, the impact of ART on CNS pathology remains incompletely understood. Studies examining CSF protein profiles in treated HIV-infected individuals reveal a complex interplay between viral suppression and persistent neuro-inflammation. While ART effectively suppresses viral replication in the CNS, some individuals exhibit residual immune activation and inflammation. This is evidenced by continued elevation of inflammatory markers like IL-6 and soluble CD14 (sCD14), indicating ongoing immune activation despite viral suppression. Moreover, alterations in proteins associated with synaptic function and neuroplasticity, such as Brain-derived Neurotrophic Factor (BDNF), suggest potential mechanisms underlying cognitive dysfunction in treated HIV infection. Interestingly, longitudinal studies have demonstrated dynamic changes in CSF protein levels over the course of HIV infection and treatment. Early initiation of ART appears to mitigate neuro-inflammatory processes, leading to normalization of certain CSF biomarkers. Conversely, late initiation of treatment or treatment interruptions may exacerbate neuro-inflammation and neuronal injury, underscoring the importance of timely and sustained ART adherence in preserving CNS health. Beyond conventional biomarkers, advances in proteomic technologies offer a comprehensive view of the CSF proteome in HIV-1 infection. High-throughput mass spectrometry-based approaches enable the identification of novel protein signatures associated with disease progression and treatment response. Integration of multi-omics data further enhances our understanding of the molecular pathways involved in HIV-associated neurodegeneration, paving the way for the development of precision medicine approaches targeting specific CNS mechanisms. In conclusion, alterations in CSF protein composition reflect the dynamic interplay between HIV-1 infection and CNS pathology. Untreated HIV infection is characterized by neuroinflammation and neuronal injury, whereas ART-mediated viral suppression partially attenuates these processes but may not fully restore CNS homeostasis.

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CONFLICT OF INTEREST

The author declares there is no conflict of interest in publishing this article.

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Corresponding author Mia Elizabeth, Department of Pathology, Arcadia University, USA, E-mail: MiaElizabeth992@yahoo.com

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