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Exploring The Immune Mechanisms Behind Neurodegeneration: Insights

Patrono Salvatore*

Department of Neuroimmunology, University of Clinical Sciences, Berlin, Germany

Abstract

Neurodegenerative diseases (NDs) such as Alzheimer's, Parkinson's, and multiple sclerosis are characterized by progressive neuronal loss, often accompanied by an imbalance in immune responses. Recent research in the Journal of Clinical & Experimental Neuroimmunology (JCE Neuroimmunology) has expanded our understanding of the immune mechanisms that drive neurodegeneration. Immune system dysfunction, particularly in microglial activation, cytokine production, and T-cell responses, plays a central role in the pathophysiology of these diseases. This article reviews key findings from JCE Neuroimmunology, focusing on the molecular and cellular mechanisms of neuroinflammation, the contribution of innate and adaptive immune cells to disease progression, and the therapeutic potential of immune-modulating strategies.

Keywords: Neurodegeneration; Microglia; T-cell activation

Introduction

Neurodegenerative diseases (NDs) are a group of disorders characterized by the progressive degeneration of the central nervous system (CNS). These diseases include Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and multiple sclerosis (MS), which collectively represent a growing public health concern as the global population ages. The pathogenesis of NDs has long been thought to be driven primarily by genetic mutations and environmental factors; however, the role of immune system dysregulation is now emerging as a central player in disease development and progression. The Journal of Clinical & Experimental Neuroimmunology (JCE Neuroimmunology) has contributed significantly to advancing our understanding of the complex relationship between the immune system and neurodegeneration. In this article, we explore key insights derived from JCE Neuroimmunology that highlight immune mechanisms underpinning neurodegeneration. Specifically, we examine the role of neuroinflammation, the activation of microglia, and T-cell involvement in disease progression, while also considering emerging therapeutic strategies aimed at modulating the immune response in NDs [1].

Immune system involvement in neurodegeneration: Neurodegeneration is increasingly recognized as a process that is closely intertwined with immune system activation. In NDs, chronic neuroinflammation—driven by both innate and adaptive immune responses—plays a crucial role in disease progression. The immune system in the CNS differs from that in peripheral tissues, primarily due to the blood-brain barrier (BBB), which tightly regulates the entry of immune cells and inflammatory mediators. However, in the context of neurodegeneration, this regulation is disrupted, leading to local immune activation and subsequent neuronal damage.

Microglial activation: a central role in neuroinflammation: Microglia, the resident immune cells of the CNS, are the primary responders to injury or disease within the brain. Under normal conditions, microglia maintain homeostasis and support neuronal function. However, in response to injury or disease, microglia undergo activation, releasing a range of pro-inflammatory cytokines, chemokines, and reactive oxygen species (ROS). This response, while initially protective, can become chronic and maladaptive, exacerbating neurodegeneration. Recent research published in JCE Neuroimmunology has elucidated the dual nature of microglial activation in NDs. On one hand, microglia are essential for clearing cellular debris, plaques, and apoptotic cells. On the other hand, prolonged activation leads to the release of neurotoxic factors that contribute to neuronal death. Studies have highlighted the role of microglial polarization—specifically the M1 (pro-inflammatory) and M2 (anti-inflammatory) phenotypes—in determining the progression of neurodegenerative diseases. A shift toward M1 microglia and the dominance of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 have been associated with the exacerbation of diseases like Alzheimer's and Parkinson's.

Cytokine and chemokine dysregulation in neurodegeneration: Cytokines and chemokines are key mediators of neuroinflammation. In the context of neurodegenerative diseases, an imbalance between pro-inflammatory and anti-inflammatory cytokines contributes to the pathological environment in the brain [2]. Elevated levels of pro-inflammatory cytokines, such as IL-1β, TNF-α, and IL-6, have been detected in the brains and cerebrospinal fluid of individuals with AD, PD, and MS. These cytokines induce further activation of microglia and astrocytes, create a neurotoxic environment, and impair synaptic function.

Chemokines, such as CCL2 and CXCL10, play a crucial role in the recruitment of peripheral immune cells to the CNS. These immune cells, including T lymphocytes, macrophages, and neutrophils, further exacerbate neuroinflammation and contribute to neurodegeneration. Studies featured in JCE Neuroimmunology have shown that targeting specific cytokines and chemokines—through neutralizing antibodies or small molecule inhibitors—holds promise as a therapeutic strategy for controlling neuroinflammation in NDs [3].

Adaptive immune system in neurodegeneration: While the innate immune system, particularly microglia, has traditionally been seen as the primary mediator of neuroinflammation, emerging evidence

***Corresponding author:** Patrono Salvatore, Department of Neuroimmunology, University of Clinical Sciences, Berlin, Germany, E-mail: Salvatore78@gmail.com

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suggests that adaptive immune responses, especially T-cell activation, also play a significant role in neurodegeneration.

Discussion

T lymphocytes are central players in the adaptive immune response. In autoimmune neurodegenerative diseases such as MS, T cells play a well-established role in attacking the CNS, leading to demyelination and neuronal damage. In diseases like AD and PD, T cells also contribute to the inflammatory milieu, although the exact mechanisms remain less clear. JCE Neuroimmunology has published several studies examining the role of different T-cell subsets—particularly CD4+ helper T cells and CD8+ cytotoxic T cells—in NDs. T cells are activated by antigen-presenting cells (APCs) such as dendritic cells, which process and present antigens to initiate an immune response. In neurodegenerative diseases, these T cells can infiltrate the brain and contribute to tissue damage. In AD, for example, studies have shown that activated CD4+ T cells infiltrate the brain and exacerbate amyloid plaque deposition and neuronal loss. In PD, CD8+ T cells have been implicated in the destruction of dopaminergic neurons. Given the complex role of immune responses in neurodegeneration, researchers are increasingly focused on developing immune-modulating therapies to prevent or slow disease progression. Several promising approaches have emerged from research in JCE Neuroimmunology. Immunotherapy has shown promise in Alzheimer's disease, with several clinical trials targeting amyloid plaques or tau tangles—the hallmark pathological features of the disease. However, these therapies also modulate the immune response, either by enhancing or suppressing inflammation. For example, monoclonal antibodies targeting amyloid-beta (such as aducanumab) are thought to clear amyloid plaques but may also influence microglial activation and cytokine production [4-9]. In PD, therapies aimed at modulating the immune response are also in development. Studies have shown that reducing inflammation through the use of nonsteroidal anti-inflammatory drugs (NSAIDs) or immune-modulating agents may offer neuroprotective effects.

Conclusion

The immune system plays a pivotal role in the development and progression of neurodegenerative diseases. Chronic neuroinflammation, driven by activated microglia, cytokine dysregulation, and T-cell involvement, contributes to the pathology of diseases like Alzheimer's, Parkinson's, and multiple sclerosis.Future research should continue to explore how best to target immune mechanisms in the CNS, aiming to provide effective treatments that not only slow disease progression but also improve the quality of life for patients suffering from these devastating disorders.

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Conflict of Interest

None

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