

NEUROINFLAMMATION AND NEURODEGENERATION IN NEUROLOGICAL DISORDERS: MOLECULAR BIOMARKERS, NEURONAL EXCITABILITY, AND IMPLICATIONS FOR SEIZURE ASSESSMENT

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ABSTRACT

Background: Neuroinflammation and neurodegeneration are interconnected biological processes associated with structural and functional disturbances of the central nervous system. Alterations in inflammatory signaling, neuronal excitability, synaptic transmission, and protein metabolism may contribute to neurological dysfunction and potentially influence susceptibility to epileptic seizures.

Objective: To analyze the relationships between neuroinflammation, neurodegeneration, molecular biomarkers, and neuronal excitability, with emphasis on their potential clinical relevance to seizure assessment.

Materials and Methods: A narrative review was conducted using 33 bibliographic publications addressing Parkinson's disease, Parkinson-plus syndromes, cerebrovascular pathology, neuroinflammatory processes, neurological rehabilitation, and molecular biomarkers. Selected studies of monocyte chemoattractant protein-1 (MCP-1), alpha-synuclein, and neuronal injury markers were examined in relation to established concepts of neuronal excitability.

Results: The literature indicates that neuroinflammatory responses involve complex interactions among neurons, microglia, astrocytes, endothelial cells, and immune mediators. These mechanisms may affect neurotransmitter balance, membrane excitability, synaptic function, and neuronal network stability. MCP-1 has been investigated as a marker of inflammatory activity, whereas alpha-

synuclein is closely associated with neurodegenerative processes in Parkinson's disease and related syndromes. Research on neuroaxonal injury markers provides additional perspectives on neurological damage and disease progression. Nevertheless, the reviewed studies do not establish these markers as validated diagnostic or prognostic indicators of epilepsy.

Discussion: Neuroinflammation may contribute to neuronal hyperexcitability in specific pathological settings; however, biomarker associations observed in neurodegenerative diseases cannot automatically be extrapolated to seizure disorders. Disease-specific studies with standardized clinical outcomes, electroencephalographic evaluation, and appropriate control groups are required.

Conclusion: Understanding interactions among neuroinflammation, neurodegeneration, and neuronal excitability may support future biomarker research and improve mechanistic assessment of neurological disorders. Further prospective investigations are necessary before inflammatory or neurodegenerative biomarkers can be recommended for seizure prediction or diagnosis.

Keywords: neuroinflammation; neurodegeneration; neuronal excitability; MCP-1; alpha-synuclein; biomarkers; epileptogenesis; neurological disorders.