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NEUROINFLAMMATION AND NEURODEGENERATIVE DISEASES: THEIR ROLE IN PATHOGENESIS AND CONTEMPORARY PERSPECTIVES

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Abstract:

This article analyzes the role of neuroinflammation in the pathogenesis of neurodegenerative diseases and reviews contemporary scientific perspectives on the subject. Neuroinflammation is a complex immune-inflammatory process that occurs within the central nervous system and develops through the involvement of microglial cells, astrocytes, and pro-inflammatory cytokines. Neurodegenerative diseases are currently among the most pressing challenges facing global healthcare systems. As the world's population continues to age, the prevalence of these disorders is increasing each year. At present, more than 55 million people worldwide are living with dementia, with approximately 10 million new cases diagnosed annually. In most of these disorders, neuroinflammation represents one of the principal pathogenic mechanisms. Recent studies have demonstrated that chronic neuroinflammation contributes to neuronal degeneration in diseases such as Alzheimer's disease, Parkinson's disease, Huntington's disease, and multiple sclerosis. This article highlights the significance of microglial activation, oxidative stress, inflammasome activation, disruption of the blood–brain barrier, and genetic factors in the development of these conditions. In addition, it provides an overview of contemporary therapeutic approaches, including anti-inflammatory therapy, immunotherapy, antioxidant agents, and gene therapy. A deeper understanding of neuroinflammation may enhance opportunities for the early diagnosis and effective treatment of neurodegenerative diseases.

Keywords: Neuroinflammation, neurodegenerative diseases, oxidative stress, multiple sclerosis, Alzheimer's disease, Parkinson's disease, microglial activation, diagnosis, effective treatment, central nervous system.

INTRODUCTION:

The relationship between neuroinflammation and neurodegenerative diseases has become one of the most significant areas of research in modern medicine. The incidence of neurodegenerative diseases continues to increase annually, with approximately 10 million new cases reported each year.

Alzheimer's disease is the most common form of dementia and is characterized by progressive impairment of memory, cognition, and activities of daily living.

Prevalence:

- Accounts for 60–70% of all dementia cases.
- More than 35–40 million people worldwide are affected.
- Millions of new cases are diagnosed annually.

Mortality:

- The average survival time following diagnosis is 4–8 years, although some patients may survive for up to 20 years.

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- Death most commonly results from complications such as pneumonia, infections, and swallowing or respiratory dysfunction.

Parkinson's disease is a progressive neurodegenerative disorder characterized by bradykinesia, tremor of the hands, legs, or head, muscular rigidity, and impaired balance.

Prevalence:

- More than 10 million people worldwide are affected.
- Approximately 90,000 new cases are reported each year.

Mortality:

- Mortality is primarily associated with disease-related complications rather than the disease itself.
- The average life expectancy after diagnosis ranges from 10 to 20 years or longer.

Neurodegenerative diseases represent some of the most complex, multifactorial pathological conditions in modern medicine and neurology, for which no definitive cure has yet been developed. These disorders are characterized by progressive and irreversible neuronal loss within the central nervous system, disruption of synaptic connections, and gradual functional deterioration of brain structures. Consequently, cognitive, motor, and psycho-emotional functions progressively decline, significantly affecting patients' daily activities, social functioning, and overall quality of life.

The most common neurodegenerative diseases include Alzheimer's disease, Parkinson's disease, Huntington's disease, and multiple sclerosis. Although these disorders present with distinct clinical manifestations, they share common pathogenic mechanisms, including neuronal injury, inflammatory processes, and programmed cell death.

Neuroinflammation and Alzheimer's Disease

Pathogenesis:

- Accumulation of β-amyloid peptides.
- Formation of neurofibrillary tangles due to hyperphosphorylation of the tau protein.
- Chronic activation of microglia.
- Impaired synaptic transmission and alterations in neurotransmitter levels.

Changes in neurotransmitters:

- Acetylcholine ↓
- Glutamate ↑
- Serotonin ↓
- Noradrenaline ↓

A reduction in acetylcholine is considered one of the primary causes of memory impairment and learning deficits.

Neuroinflammation and Parkinson's Disease

Pathogenesis:

- Aggregation of α-synuclein (Lewy bodies).
- Degeneration of dopaminergic neurons in the substantia nigra.
- Microglial activation.
- Mitochondrial dysfunction.

Changes in neurotransmitters:

- Dopamine ↓
- Acetylcholine (relative increase)
- Noradrenaline ↓
- Serotonin ↓

Dopamine deficiency results in the characteristic clinical manifestations of Parkinson's disease, including tremor, rigidity, and bradykinesia.

Neuroinflammation and Multiple Sclerosis

Pathogenesis:

Activation of the immune system

T lymphocytes and B lymphocytes become activated by unknown factors, including genetic susceptibility, viral infections, and environmental influences.

Disruption of the blood–brain barrier

Activated immune cells cross the blood–brain barrier and infiltrate the central nervous system.

Development of neuroinflammation

Activated T lymphocytes, B lymphocytes, and microglial cells release numerous inflammatory mediators, including:

- TNF-α
- IL-1β
- IL-6
- IFN-γ
- IL-17

Inflammation leads to demyelination, resulting in slowed or blocked nerve impulse conduction.

Axonal injury

As the disease progresses, not only the myelin sheath but also axons become damaged, leading to irreversible neurological deficits.

Formation of sclerotic plaques

Scar-like lesions (sclerotic plaques) develop within affected regions of the central nervous system, giving rise to the term *multiple sclerosis*.

Role of neurotransmitters and inflammatory mediators

- Glutamate ↑ — induces excitotoxicity.
- GABA ↓ — reduces inhibitory neurotransmission.
- TNF-α, IL-1β, IL-6, and IL-17 — amplify the inflammatory response.
- Reactive oxygen species and nitric oxide damage oligodendrocytes.

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Over the past several decades, both fundamental and clinical studies have demonstrated that neuroinflammation plays a central and decisive role in the development of neurodegenerative diseases. Neuroinflammation represents the innate immune response of the central nervous system and is primarily mediated by microglia and astrocytes. Under physiological conditions, these cells protect neurons, maintain tissue homeostasis, and remove damaged cellular components. However, under pathological conditions, their excessive activation creates a pro-inflammatory environment that becomes detrimental to neuronal survival.

Upon activation, microglial cells secrete numerous biologically active substances, including pro-inflammatory cytokines (IL-1β, IL-6, and TNF-α), chemokines, and reactive oxygen species. Excessive production of these mediators exerts toxic effects on neuronal membranes, synaptic structures, and intracellular organelles. Consequently, oxidative stress is intensified, mitochondrial function becomes impaired, and cellular energy metabolism is disrupted.

Astrocytes also play a crucial role in neuroinflammation. Under normal physiological conditions, they regulate ionic homeostasis, recycle neurotransmitters, and maintain the integrity of the blood–brain barrier. During neuroinflammation, however, astrocytes become reactive, leading to excessive glutamate accumulation and excitotoxicity. This process results in calcium overload within neurons and activation of apoptotic pathways.

Disruption of the blood–brain barrier is another critical component of neuroinflammation. Increased permeability of the blood–brain barrier allows peripheral immune cells to infiltrate the central nervous system. These infiltrating cells further amplify local inflammation, promoting the transition of the pathological process into a chronic state. In addition, genetic factors play an important role in the development of neurodegenerative diseases. Certain genetic mutations may enhance microglial activation or reduce neuronal resistance to cellular stress.

Current scientific approaches are increasingly focused on controlling neuroinflammation as a strategy to halt or slow the progression of neurodegeneration.

Molecular Mechanisms

Research indicates that the accumulation of pathological proteins, including amyloid-β, tau protein, and α-synuclein, is recognized by microglia as inflammatory stimuli, thereby initiating and sustaining neuroinflammatory responses.

Anti-inflammatory Therapy

Anti-inflammatory therapy aims to protect neurons by reducing neuroinflammation.

Mechanism of action

In neurodegenerative diseases, activated microglia release inflammatory mediators such as:

- IL-1β
- IL-6
- TNF-α
- IFN-γ

These cytokines exacerbate neuronal injury. Anti-inflammatory therapy suppresses their production, thereby limiting inflammation and neuronal damage.

Major therapeutic strategies

1. Cytokine blockade
    - TNF-α inhibitors.
    - IL-1β-targeted therapies.
2. NLRP3 inflammasome inhibitors
    - Reduce microglia-mediated inflammation.
    - Decrease the release of IL-1β and IL-18.
3. Microglial modulators
    - Limit excessive and harmful microglial activation.
    - Provide neuroprotective effects.
4. Antioxidant therapy
    - Reduces the formation of free radicals.
    - Attenuates oxidative stress.

Therapeutic objectives

- Reduce neuroinflammation.
- Protect neurons from progressive damage.
- Slow disease progression.
- Preserve cognitive and motor function for as long as possible.

Gene Therapy

Gene therapy is a treatment approach that aims to correct genetic defects responsible for disease progression or introduce healthy genes into damaged cells. In neurodegenerative diseases, this strategy is designed to restore neuronal function and slow disease progression.

According to contemporary scientific perspectives, neuroinflammation functions not only as a protective mechanism but, when prolonged, can evolve into a pathological and destructive process. This condition is characterized by impaired synaptic plasticity, reduced neuronal signal transmission, and enhanced programmed cell death (apoptosis). In particular, activation of inflammasome complexes, especially the NLRP3 inflammasome, is increasingly recognized as a key factor accelerating neurodegenerative processes.

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Therefore, an in-depth understanding of the mechanisms underlying neuroinflammation is of considerable scientific and clinical importance. Such knowledge not only contributes to a more comprehensive understanding of disease pathogenesis but also facilitates the identification of early diagnostic biomarkers and the development of novel therapeutic strategies. Currently, several promising treatment approaches are being actively investigated, including anti-inflammatory therapy, immunomodulatory interventions, antioxidant therapies, monoclonal antibody-based treatments, and gene therapy.

CONCLUSION

This article examined the role of neuroinflammation within the central nervous system and its significance in the pathogenesis of neurodegenerative diseases. The findings indicate that although neuroinflammation initially serves as a protective response, its chronic activation transforms it into a pathological process that is detrimental to neuronal survival. Excessive activation of microglia and astrocytes results in the release of pro-inflammatory cytokines, oxidative stress mediators, and other neurotoxic factors, leading to progressive damage of brain tissue.

Furthermore, disruption of the blood–brain barrier facilitates the infiltration of peripheral immune cells into the central nervous system, thereby amplifying the inflammatory response. These processes ultimately promote neuronal apoptosis, synaptic dysfunction, and mitochondrial impairment. Multiple sclerosis, in particular, provides a clear example of the close relationship between neuroinflammation and demyelination.

Current scientific evidence increasingly recognizes neuroinflammation not merely as a consequence of neurodegenerative diseases but as one of their principal pathogenic mechanisms. Consequently, therapeutic strategies aimed at regulating microglial activation, reducing oxidative stress, and modulating immune responses are considered promising avenues for disease management.

Overall, a deeper understanding of the molecular and cellular mechanisms underlying neuroinflammation provides an essential scientific foundation for improving the early diagnosis, prognosis, and development of more effective therapeutic strategies for neurodegenerative diseases. Continued advances in this field are expected to enhance the long-term management of neurodegenerative disorders and contribute to slowing their progression.

REFERENCES:

1. Axmedov, A. A. (2022). Neyrodegenerativ kasalliklarda yallig‘lanish jarayonlarining o‘rni. Tibbiyot va Zamon, 4(2), 45–52.
2. Karimov, B. R., & Rasulov, S. M. (2023). Neyroimmunologiyaning zamonaviy yo‘nalishlari. O‘zbekiston Tibbiyot Jurnali, 1(3), 18–26.
3. Usmonov, X. X. (2024). Neyroyallig‘lanish va markaziy nerv tizimi patologiyalari. Nevrologiya va Neyroxirurgiya Axborotnomasi, 2(1), 30–39
4. Bloem, B. R., Okun, M. S., & Klein, C. (2021). Parkinson's disease. The Lancet, 397(10291), 2284–2303.
5. Ransohoff Richard M.. How neuroinflammation contributes to neurodegeneration. Science. 2016;353(6301):777–783
6. Leng Fei, Edison Paul. Neuroinflammation and microglial activation in Alzheimer's disease. Nature Reviews Neurology. 2021.
7. Biswas Kaushiki. Microglia mediated neuroinflammation in neurodegenerative diseases: A review on the cell signaling pathways involved in microglial activation. Journal of Neuroimmunology. 2023.
8. Gao Chao va boshq. Microglia in neurodegenerative diseases: mechanism and potential therapeutic targets. Signal Transduction and Targeted Therapy. 2023.