Immune-mediated mechanisms in neurological diseases: Advances in neuroimmunology.

Stanley Zaho*

Department of Neurology, Methodist Neurological Institute, Houston, USA

Introduction

The immune system plays a vital role not only in protecting the body against pathogens but also in maintaining the integrity and function of the Central Nervous System (CNS). In certain neurological diseases, immune-mediated mechanisms contribute to the pathogenesis and progression of the condition. This essay aims to explore the role of immune-mediated mechanisms in neurological diseases, focusing on autoimmune disorders and inflammatory processes. Understanding these mechanisms is crucial for the development of targeted therapies and interventions to mitigate the detrimental effects of immune dysregulation in neurological diseases [1].

Autoimmune neurological diseases

Autoimmune neurological diseases are characterized by an immune response directed against self-antigens within the CNS. These conditions involve immune-mediated inflammation and tissue damage within the nervous system [2]. Several examples of autoimmune neurological diseases include Multiple Sclerosis (MS), Guillain-Barré Syndrome (GBS), and Neuromyelitis Optica Spectrum Disorder (NMOSD).

Multiple Sclerosis (MS): MS is a chronic inflammatory disease characterized by the immune system's attack on myelin, the protective covering of nerve fibers in the CNS. Immune cells, primarily T cells, infiltrate the CNS and initiate an immune response against myelin, leading to demyelination, neuroinflammation, and subsequent neurological dysfunction. B cells and antibodies also contribute to MS pathogenesis by targeting myelin components and promoting immune cell activation [3].

Guillain-Barré Syndrome (GBS): GBS is an acute inflammatory disorder affecting the peripheral nervous system. It is primarily an immune-mediated attack on the myelin sheath surrounding peripheral nerves or the nerve roots. The immune response in GBS is often triggered by preceding infections, such as respiratory or gastrointestinal infections. Immune cells, including T cells and macrophages, infiltrate peripheral nerves and induce inflammation, resulting in demyelination and nerve damage.

Neuromyelitis Optica Spectrum Disorder (NMOSD): NMOSD is an autoimmune disorder characterized by inflammation and demyelination primarily affecting the optic nerves and spinal cord. In NMOSD, autoantibodies target the aquaporin-4 water channel protein located on astrocytes in the CNS. The binding of these autoantibodies leads to complement-mediated inflammation, astrocyte injury, and subsequent demyelination [4].

Inflammatory processes in neurological diseases

In addition to autoimmune diseases, immune-mediated inflammatory processes play a significant role in various neurological diseases. These inflammatory mechanisms can be triggered by infections, chronic inflammatory conditions, or dysregulated immune responses. Some examples of neurological diseases associated with immune-mediated inflammation include Alzheimer's disease, Parkinson's disease, and stroke.

Alzheimer's Disease (AD): Chronic inflammation within the brain contributes to the pathogenesis and progression of AD, the most common form of dementia. Activation of immune cells, such as microglia and astrocytes, leads to the release of pro-inflammatory cytokines and the accumulation of amyloid-beta plaques. This chronic inflammation exacerbates neuronal damage and contributes to cognitive decline.

Parkinson's Disease (PD): In PD, immune-mediated inflammation plays a crucial role in the neurodegenerative process. Activated microglia release pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-alpha) and interleukin-1 beta (IL-1beta), contributing to the degeneration of dopaminergic neurons in the substantia nigra. The presence of alpha-synuclein aggregates, a hallmark of PD, further activates immune responses, perpetuating the inflammatory cycle [5].

Stroke: Stroke involves the interruption of blood supply to the brain, leading to tissue damage and inflammation.

References

1. Zhao W, Beers DR, Appel SH. Immune-mediated mechanisms in the pathoprogression of amyotrophic lateral sclerosis. J NeuroImmune Pharmacol. 2013;8:888-99.
2. Tan JS, Chao YX, Rötzschke O, et al. New insights into immune-mediated mechanisms in Parkinson’s disease. Int J Mol Sci. 2020;21(23):9302.

***Correspondence to:** Stanley Zaho, Department of Neurology, Methodist Neurological Institute, Houston, USA, E-mail: Zaho45@staff.marburg.de

***Received:** 01-Jul-2023, Manuscript No. AACIR-23-104165; **Editor assigned:** 03-Jul-2023, Pre QC No. AACIR-23-104165(PQ); **Reviewed:** 17-Jul-2023, QC No. AACIR-23-104165; **Revised:** 20-Feb-2023, Manuscript No. AACIR-23-104165(R); **Published:** 24-Jul-2023, DOI: 10.35841/aacir-6.3.151*

***Citation:** Zaho S. Immune-mediated mechanisms in neurological diseases: Advances in neuroimmunology. 2023;6(3):151*

3. Amante FH, Haque A, Stanley AC, et al. Immune-mediated mechanisms of parasite tissue sequestration during experimental cerebral malaria. J Immunol. 2010;185(6):3632-42.
4. Levin MC, Krichavsky M, Berk J, et al. Neuronal molecular mimicry in immune-mediated neurologic disease. Ann Child Neurol. 1998;44(1):87-98.
5. Sanders DB. Lambert-Eaton myasthenic syndrome: Clinical diagnosis, immune-mediated mechanisms, and update on therapies. Ann Child Neurol. 1995;37(S1):63-73.

Citation: *Zaho S. Immune-mediated mechanisms in neurological diseases: Advances in neuroimmunology. 2023;6(3):151*