Neurological consequences in the blood-brain barrier diseases.

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Introduction

The Blood-Brain Barrier (BBB) is the protective interface between the Central Nervous System [CNS] and circulating blood, and are critical in controlling the movement of ions, molecules, and cells to maintain CNS homeostasis. BBB disruption is a key event involved in the pathology of many neurological diseases and has recently been implicated in severe acute respiratory syndrome coronavirus infection. This review describes the cellular and molecular components that coordinate BBB formation and its maintenance across species. We discuss how this barrier can be modulated for efficient drug delivery to the brain and how BBB degradation is involved in neurological disease. Finally, we highlight a recent study identifying a possible mechanism by which may enter her CNS by crossing her BBB in a coronavirus disease [1].

Novel coronavirus disease is a respiratory illness. The virus has spread rapidly around the world and was declared a Public Health Emergency of International Concern by the World Health Organization (WHO). Genetic and phylogenetic analyses indicate that this virus has a strong relationship with severe acute respiratory syndrome and Middle East Respiratory Syndrome (MERS). Consistent with this, published epidemiological data indicate that this virus has a similar etiology and symptoms as pneumonia induced. Also, although less researched COVID-19 may be associated with the development of neurological symptoms along with these respiratory symptoms through effects on sensory, cerebrovascular, cognitive, or motor function. Approaches to study the effects of this new virus on brain function pose new and important challenges for neuroscience research. Because infections in the brain not only affect overall health, but can also create comorbidities and risk of death. Therefore, the aim of this review is to summarize the information reported so far in scientific publications on the neurological symptoms caused by SARS-CoV-2 infection and to explain the physiopathology of this new virus in the brain to date. It is to examine the underlying mechanisms [2].

The BBB is regulated by a number of physiological processes that limit vascular permeability and act as a barrier between the peripheral blood circulation and the CNS. Protects the central nervous system from pathogens, toxins and other harmful substances. A functional BBB is required to maintain certain physiological processes in the brain, such as nutrient supply, immune infiltration, and metabolism. The barrier properties of the BBB, maintained primarily by interactions between Endothelial Cells (ECs) and other Neuro Vascular Units (NVUs), are primarily paracellular endothelial Tight Junctions (TJs), influx and efflux transporters, and Determined by the metabolic properties of ECs. Disruption of the BBB underlies many neurodegenerative diseases, including Multiple Sclerosis (MS), Amyotrophic Lateral Sclerosis (ALS), Alzheimer's Disease (AD), Parkinson's Disease (PD), and Huntington's Disease (HD) accelerates degenerative changes in the CNS thought to occur in pathway may be involved in the process of SARS-CoV-2 infection into the central nervous system [3].

Stroke, a cerebrovascular disease, is a leading cause of death and severe long-term disability worldwide. It occurs when cerebral blood flow is locally interrupted or reduced, resulting in insufficient nutrients and oxygen supply, leading to brain cell death. It can be compromised and permeable, leading to various inflammatory reactions and secondary cerebral edema. For example, activated microglia in the infarct area phagocytize cell fragments and exhibit anti-inflammatory effects during the early stages of stroke. However, it then increases the release of ROS, cytokines, Tumor Necrosis Factor-α (TNF-α), and matrix metalloproteinases, leading to TJ degradation by degrading TJ proteins such as claudin-and occludin. On the other hand, activated protruding astrocytes cause physical separation of the astrocyte end feet from the EC, resulting in a leaky BBB. In addition, activated astrocytes release Vascular Endothelial Growth Factor (VEGF-A) to mediate protein degradation. Several other mechanisms, such as cytoskeletal reorganization, matrix metalloproteinase activation, oxidative stress, and inflammatory cytokines, have also been shown to contribute to her BBB damage [4].

These effects combine to cause BBB disruption and cerebral edema, which are associated with a poor clinical outcome in stroke. Cerebral edema is generally classified as either cytotoxic or angiogenic and is the leading cause of death in her of patients. After stroke, perturbations in cell metabolism lead to the early formation of cytotoxic edema, followed by disruption of the BBB, leakage of water and plasma proteins into the interstitial compartment of the brain, development of vasogenic edema, and finally lead to complete brain swelling, neurological dysfunction, and death.

Thus, disruption of the BBB exacerbates stroke damage, and restoring BBB integrity is critical to reducing brain swelling and stroke mortality. may be important. A major obstacle to this strategy is the limited understanding of the molecular

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***Received:** 31-Aug-2022, Manuscript No.AAJBN-22-77624; **Editor assigned:** 05-Sep-2022, PreQC No. AAJBN-22-77624(PQ); **Reviewed**: 20-Sep-2022, QC No. AAJBN-22-77624; **Revised**: 23-Sep-2022, Manuscript No. AAJBN-22-77624(R); **Published**: 28-Sep-2022, DOI: 10.35841/aajbn-5.5.125*

***Citation:** Zhang J. Neurological consequences in the blood-brain barrier diseases. J Brain Neurol. 2022;5(5):125*

mechanisms regulating her BBB dysfunction in stroke, which has been partially elucidated as described our recent studies showed that membranous claudin- redistribution with autophagy activation is essential for hypoxia-induced BBB damage after stroke. These results further strengthen the role of the BBB in stroke pathophysiology and facilitate the search for new therapeutics to combat BBB dysfunction [5].

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Citation: *Zhang J. Neurological consequences in the blood-brain barrier diseases. J Brain Neurol. 2022;5(5):125*