Commentary

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Journal of Stroke Research & Therapy

2024
Vol.8 No.4:221

Advanced Neuroprotective Agents for Ischemic Stroke Recovery

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Received date: November 22, 2024, Manuscript No. IPSRT-24-20085; **Editor assigned date**: November 25, 2024, PreQC No. IPSRT-24-20085 (PQ); **Reviewed date:** December 09, 2024, QC No. IPSRT-24-20085; **Revised date**: December 16, 2024, Manuscript No. IPSRT-24-20085 (R); **Published date**: Devember 23, 2024, DOI: 10.36648/ipsrt.8.4.221

Citation: Wu J (2024) Advanced Neuroprotective Agents for Ischemic Stroke Recovery. Stroke Res Ther Vol.8. No.4:221.

Description

Ischemic stroke, a leading cause of death and long-term disability worldwide, occurs when a blood clot obstructs blood flow to the brain, causing tissue ischemia and cellular death. This medical emergency triggers a cascade of molecular and biochemical processes, including oxidative stress, inflammation, excitotoxicity and apoptosis. These mechanisms exacerbate neuronal injury and hinder recovery. While the standard treatments like thrombolysis and mechanical thrombectomy aim to restore cerebral blood flow, their therapeutic window is narrow, leaving a significant proportion of patients untreated. Consequently, the focus has shifted towards neuroprotective agents that can limit neuronal damage, enhance recovery and expand therapeutic opportunities. Recent advancements in neuroprotective therapies offer potential to mitigate the impact of ischemic strokes. Neuroprotection involves strategies to preserve neural structure and function by intervening in the cascade of events triggered by ischemia. Effective neuroprotective agents target specific pathways involved in ischemic damage. Advanced neuroprotective agents represent a potential frontier in the management of ischemic stroke, addressing the unmet need for therapies that go beyond reperfusion. While significant challenges remain in translating these agents from bench to bedside, ongoing research and technological advancements are paving the way for a paradigm shift in stroke care. A comprehensive approach combining neuroprotection, reperfusion and rehabilitation will ultimately improve outcomes and quality of life for stroke survivors.

Neuroprotection in ischemic stroke

Oxidative stress mitigation: The ischemic brain generates excessive Reactive Oxygen Species (ROS), causing oxidative damage to lipids, proteins and DNA. Antioxidants like edaravone have demonstrated efficacy in scavenging free radicals and protecting neuronal integrity.

Anti-inflammatory action: Inflammation exacerbates brain injury post-ischemia. Neuroprotective agents like minocycline inhibit microglial activation and reduce pro-inflammatory cytokines, thereby limiting neuronal death.

Excitotoxicity reduction: Glutamate accumulation during ischemia overstimulates NMDA and AMPA receptors, leading to calcium overload and neuronal damage. NMDA receptor antagonists, such as memantine, prevent excitotoxicity and safeguard neurons.

Apoptosis inhibition: The ischemic cascade activates apoptotic pathways, culminating in programmed cell death. Caspase inhibitors and Bcl-2 modulators have shown potential in preclinical studies for reducing neuronal apoptosis.

Emerging neuroprotective agents

In recent years, research has unveiled a host of novel neuroprotective agents with multifaceted mechanisms of action, paving the way for better stroke management. Nicotinamide Adenine Dinucleotide (NAD^+) Precursors NAD^+ is a vital coenzyme involved in cellular energy metabolism and DNA repair. In ischemic stroke, NAD^+ levels are depleted, leading to mitochondrial dysfunction and neuronal death. NAD+ precursors, such as Nicotinamide Riboside (NR) and Nicotinamide Mononucleotide (NMN), have demonstrated efficacy in enhancing cellular resilience to ischemia by restoring mitochondrial function and reducing oxidative stress. Stem cell therapy Mesenchymal Stem Cells (MSCs) and Induced Pluripotent Stem Cells (iPSCs) are being described for their neuroprotective potential. These cells secrete trophic factors that modulate inflammation, promote neurogenesis and repair damaged tissues. Clinical trials have shown that MSC therapy improves functional recovery in stroke patients. Nrf2 Activators the Nuclear factor erythroid 2-related factor 2 (Nrf2) is a master regulator of antioxidant defense. Activating Nrf2 enhances the expression of antioxidant enzymes, countering oxidative stress in ischemic brain tissue. Sulforaphane and other Nrf2 activators have shown neuroprotective effects in preclinical studies. Sphingosine-1-Phosphate (S1P) receptor modulators S1P receptor modulators, such as fingolimod, have demonstrated efficacy in reducing post-stroke inflammation and protecting the blood-brain barrier. By preventing immune cell infiltration into the brain, these agents limit secondary damage and support functional recovery. Hypothermic therapies targeted temperature management reduces cerebral metabolism, attenuates inflammation and stabilizes the blood-brain barrier. While not a pharmacological agent, hypothermia synergizes with drug therapies, providing a potent neuroprotective strategy.