

Post-stroke Cognitive Impairment: Mechanisms and Interventions

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Introduction

Stroke is not only a leading cause of mortality and physical disability but also a major contributor to cognitive decline worldwide. Post-stroke cognitive impairment (PSCI) is a frequent and debilitating complication that affects up to one-third of stroke survivors, with presentations ranging from mild cognitive deficits to severe vascular dementia. Unlike motor or sensory deficits, cognitive impairment is often underrecognized, yet it profoundly impacts functional independence, quality of life and long-term prognosis. Despite its prevalence, there are currently limited therapeutic strategies specifically tailored for cognitive recovery after stroke. Conventional rehabilitation approaches often emphasize motor and physical recovery, leaving cognitive domains insufficiently addressed. Understanding the mechanisms and interventions for PSCI is crucial for improving outcomes in stroke survivors. By integrating insights from vascular biology, neurodegeneration research and rehabilitation science, clinicians and researchers aim to develop more holistic, patient-centered approaches. This article reviews the underlying mechanisms of post-stroke cognitive impairment and discusses emerging therapeutic interventions, highlighting opportunities to reduce its burden and enhance long-term recovery [1].

Description

Post-stroke cognitive impairment (PSCI) is increasingly recognized as one of the most significant long-term consequences of stroke, affecting up to 30–40% of survivors. It encompasses a wide spectrum of deficits, ranging from subtle executive dysfunction and slowed processing speed to severe global impairment resembling dementia. The risk of PSCI varies according to stroke type, location and severity, with cortical and subcortical strokes showing distinct patterns of cognitive dysfunction. Hemorrhagic strokes, although less frequent, are strongly associated with early cognitive impairment due to mass effect, perihematomal injury and disruption of neural networks. Beyond lesion characteristics, patient-related factors such as age, educational background, vascular risk factors and pre-existing neurodegenerative changes shape the trajectory of cognitive

decline. Importantly, PSCI is not a static condition but may evolve dynamically over months to years, reflecting the combined influence of primary brain injury and ongoing secondary processes. This dynamic nature underscores the need for longitudinal monitoring and adaptive management strategies tailored to individual patient profiles [2].

The mechanisms underlying PSCI are multifactorial and extend beyond the immediate effects of ischemia or hemorrhage. Stroke triggers a cascade of secondary injury processes, including oxidative stress, excitotoxicity, mitochondrial dysfunction and neuroinflammation, all of which contribute to progressive neuronal injury. Activated microglia and astrocytes release pro-inflammatory cytokines that exacerbate synaptic dysfunction and neuronal loss in regions connected to the initial lesion. Disruption of the blood–brain barrier further facilitates infiltration of peripheral immune cells, amplifying inflammatory damage. Cerebral hypoperfusion and impaired autoregulation following stroke also contribute to chronic ischemia, particularly in watershed regions, leading to ongoing white matter degeneration. Structural and functional connectivity of neural networks, particularly within the default mode and executive control networks, is disrupted, impairing higher-order cognitive processes. Together, these mechanisms highlight PSCI as a product of complex interactions between vascular injury, neurodegeneration and maladaptive neuroinflammation [3].

The clinical manifestations of PSCI are diverse and depend on both lesion characteristics and systemic factors. Commonly affected domains include attention, memory, executive function, visuospatial ability and language. Cognitive deficits often coexist with post-stroke depression, apathy and fatigue, creating a multidimensional burden that complicates rehabilitation. Neuropsychological assessment remains the gold standard for diagnosis, but cognitive screening tools such as the Montreal Cognitive Assessment (MoCA) and Mini-Mental State Examination (MMSE) are widely used for initial evaluation. Neuroimaging studies have provided valuable insights into structural correlates of PSCI, showing that white matter hyperintensities, cortical atrophy and microbleeds are strong predictors of decline. Functional MRI and diffusion tensor

imaging further reveal disruptions in connectivity across large-scale brain networks. Importantly, PSCI is not limited to the elderly; Recognizing the clinical heterogeneity of PSCI is essential for tailoring management strategies to individual patient needs [4].

Interventions for PSCI are currently evolving, with promising advances in both pharmacological and non-pharmacological domains. Pharmacological approaches have included the use of cholinesterase inhibitors, memantine and nootropic agents, though results have been mixed and large-scale trials are limited. Cerebrolysin, citicoline and other neuroprotective compounds are being investigated for their potential to enhance neuroplasticity and cognitive recovery. Lifestyle modifications, including management of vascular risk factors, healthy diet and social engagement, are also critical in preventing further decline. Emerging technologies such as virtual reality, brain-computer interfaces and digital health platforms are creating new opportunities for personalized rehabilitation. Ultimately, a multimodal approach that combines pharmacological, behavioral and technological strategies appears most promising. The future of PSCI management lies in precision medicine approaches that account for individual risk profiles, lesion characteristics and genetic predispositions [5].

Conclusion

Post-stroke cognitive impairment represents one of the most challenging and often underrecognized consequences of stroke, with significant implications for long-term recovery and quality of life. Its mechanisms are complex, involving a combination of direct vascular injury, secondary neuroinflammatory and oxidative processes and interactions with pre-existing neurodegenerative pathology. The clinical presentation is heterogeneous, ranging from mild cognitive deficits to severe dementia, underscoring the need for early identification and comprehensive assessment. Technological innovations, including virtual reality and digital health tools, offer promising adjuncts to traditional therapies, paving the way for more personalized and accessible interventions. Ultimately, addressing PSCI requires a multidisciplinary approach that integrates vascular risk

management, targeted cognitive therapies and psychosocial support. Continued research into mechanisms and interventions will be essential to reduce its burden and improve the long-term outcomes of stroke survivors.

Acknowledgment

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Conflict of Interest

None.

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