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POST-STROKE COGNITIVE IMPAIRMENTS IN DIFFERENT PATHOGENETIC SUBTYPES OF STROKE: A REVIEW.

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Abstract: Post-stroke cognitive impairments (PSCI) are one of the most significant complications in modern neurology, substantially affecting patients' quality of life and recovery prognosis. The prevalence of cognitive deficits after stroke ranges from 30% to 80%, depending on stroke subtype, lesion location, and concomitant vascular pathology. This review discusses the mechanisms of PSCI in ischemic and hemorrhagic strokes, as well as in different pathogenetic subtypes of ischemic stroke: atherothrombotic, cardioembolic, lacunar, and mixed types. Special attention is given to structural and functional brain changes associated with impairments in memory, executive functions, attention, and language skills. The review emphasizes the importance of early diagnosis, individualized rehabilitation, and the potential use of neuroprotective therapies.

Keywords: stroke, cognitive impairment, ischemic stroke, hemorrhagic stroke, pathogenesis, rehabilitation.

Introduction. Stroke remains one of the leading causes of mortality and long-term disability worldwide, representing a major public health challenge. According to the World Health Organization, approximately 15 million people suffer a stroke each year, and of these, nearly 5 million die, while another 5 million are left permanently disabled. While the acute neurological deficits of stroke, such as motor weakness, sensory loss, and speech disturbances, are well-recognized, cognitive impairments following stroke are increasingly being acknowledged as a significant and often underdiagnosed consequence.

Post-stroke cognitive impairments (PSCI) encompass a wide spectrum of deficits affecting multiple domains, including memory, attention, executive function, language, visuospatial abilities, and processing speed. These impairments may be transient in some patients, improving within weeks or months, whereas in others, they persist or even progress to post-stroke dementia, contributing to long-term functional dependence and reduced quality of life. Cognitive deficits can hinder the patient's ability to adhere to rehabilitation programs, manage medications, perform daily activities, and reintegrate into social and occupational roles.

The severity and pattern of cognitive impairment are influenced by multiple factors, including the size, location, and type of the stroke, the patient's age, preexisting cognitive reserve, and comorbid vascular and neurodegenerative conditions. For instance, cortical strokes affecting frontal or temporal regions are more likely to result in executive dysfunction and memory deficits, whereas subcortical lacunar infarcts predominantly affect attention, processing speed, and subcortical cognitive functions. Hemorrhagic strokes, due to mass effect and secondary inflammation, may produce diffuse cognitive deficits and are often associated with poorer recovery trajectories.

Understanding the pathogenetic subtypes of stroke—such as atherothrombotic, cardioembolic, and small vessel (lacunar) ischemic strokes, as well as hemorrhagic strokes—and their specific impact on cognitive functions is essential for accurate prognosis, risk stratification, and the design of targeted rehabilitation strategies. Moreover, recognition of early cognitive changes allows clinicians to implement neuroprotective therapies, cognitive training, and supportive interventions aimed at minimizing long-term disability.

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Despite increasing research interest, post-stroke cognitive impairment remains underdiagnosed in routine clinical practice, partly due to the subtlety of early deficits and a historical focus on motor and sensory outcomes. Therefore, systematic assessment of cognitive function, integration of neuroimaging biomarkers, and consideration of individual patient characteristics are critical for the comprehensive management of stroke survivors. Addressing PSCI is not only important for improving individual patient outcomes but also has substantial socioeconomic implications, as cognitive deficits contribute to increased caregiver burden, healthcare costs, and decreased productivity.

Epidemiology of Post-Stroke Cognitive Impairments. According to recent studies, cognitive impairments are observed in 30–80% of patients after stroke. The prevalence and severity of deficits depend on:

- stroke type (ischemic or hemorrhagic),

- lesion location and volume,

- comorbid vascular conditions (hypertension, diabetes, atherosclerosis),

- age and premorbid cognitive reserve of the patient.

Patients with lesions in the frontoparietal and medial temporal regions are particularly vulnerable due to their key roles in executive functions and memory.

2. Mechanisms of Cognitive Impairments by Stroke Subtype. Ischemic Stroke. Ischemic stroke most often occurs due to thrombosis, embolism, or microcirculatory disturbances. The main pathogenetic subtypes are:

Atherothrombotic stroke:

- Typically involves large artery territories (e.g., middle cerebral artery).

- Affects frontoparietal and temporal regions, causing executive dysfunction and memory impairment.

- Chronic ischemia contributes to the development of vascular dementia.

Cardioembolic stroke:

- Associated with emboli originating from the heart (atrial fibrillation, valvular disease).

- Often produces large cortical lesions accompanied by apraxia and aphasia.

- High likelihood of early post-stroke cognitive deficits.

Lacunar stroke:

- Results from small vessel disease affecting deep brain structures (basal ganglia, thalamus, internal capsule).

- Commonly manifests as attention deficits, executive dysfunction, and psychomotor slowing.

- Risk of progressive subcortical dementia is significant.

Mixed subtypes:

- Combination of large and small vessel lesions exacerbates cognitive deficits.

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- Patients present with a mixed clinical picture, including both cortical and subcortical impairments.

Hemorrhagic Stroke

Hemorrhagic stroke is characterized by massive intracerebral bleeding leading to focal neurological deficits. Cognitive impairments include:

- pronounced amnesia,

- reduced attention,

- slowed psychomotor responses,

- potential development of post-hemorrhagic dementia.

Mechanisms involve direct neuronal damage, blood toxicity, and secondary inflammation.

Factors Exacerbating Cognitive Impairments

1. Age and cognitive reserve: older patients with low premorbid cognitive reserve are at higher risk of severe deficits.

2. Lesion volume and location: larger and cortical strokes are more likely to cause significant cognitive impairments.

3. Comorbid conditions: hypertension, diabetes, and atherosclerosis increase the risk of progression.

4. Rehabilitation timing: early cognitive rehabilitation enhances functional recovery.

Approaches to Diagnosis and Rehabilitation

Diagnosis

1. Clinical scales: MMSE, MoCA, FAB.

2. Neuropsychological testing: assessment of memory, attention, executive functions, and language.

3. Neuroimaging: MRI and CT to determine lesion location and volume, aiding in predicting cognitive outcomes.

Rehabilitation

- Individualized cognitive training (computer-based programs, speech therapy, psychological interventions).

- Pharmacotherapy (neuroprotective agents, vascular drugs, management of comorbidities).

- Comprehensive approaches, including physical rehabilitation and social support, improve recovery prognosis.

Future Research Directions

- Development of biomarkers for early identification of patients at high risk of PSCI.

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- Genetic and molecular studies to elucidate the mechanisms of cognitive impairment after stroke.

- Implementation of novel neurorehabilitation technologies (VR training, neurostimulation).

- Differentiated treatment strategies according to pathogenetic stroke subtype.

Conclusion. Post-stroke cognitive impairments are common and severe complications of stroke. Their type and severity depend on the pathogenetic subtype, lesion location and volume, and other vascular and neuronal factors. Early diagnosis and individualized rehabilitation can reduce the severity of deficits and improve quality of life. Future research should focus on personalized approaches to treatment and prognosis of cognitive outcomes.

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