ASSESSMENT OF THE EFFECTIVENESS OF NEUROPROTECTIVE THERAPY IN THE EARLY REHABILITATION OF PATIENTS AFTER ISCHEMIC STROKE

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Research Objective

Ischemic stroke remains one of the leading causes of mortality and long-term disability worldwide. It is associated with profound socioeconomic consequences for both patients and healthcare systems. With the aging population and increasing prevalence of risk factors such as hypertension, diabetes mellitus, and atrial fibrillation, the incidence of ischemic stroke is steadily rising. While substantial advancements have been made in the acute management of stroke through thrombolysis and thrombectomy, the early rehabilitation phase remains a crucial yet often underutilized period for optimizing neurological recovery. This period, characterized by heightened neuroplasticity, offers a unique window during which targeted interventions can enhance neuronal repair and functional restoration. Among such interventions, neuroprotective therapy has garnered growing attention due to its ability to mitigate secondary brain injury and support neural regeneration.

The pathophysiology of ischemic stroke involves a cascade of deleterious events following the sudden occlusion of cerebral arteries, including excitotoxicity, oxidative stress, mitochondrial dysfunction, inflammatory responses, and apoptosis. These processes not only exacerbate the initial damage but also impair subsequent recovery. Neuroprotective agents aim to interrupt these cascades, preserve the penumbra—the viable but endangered brain tissue surrounding the infarct—and promote repair through neurotrophic support, membrane stabilization, and anti-apoptotic mechanisms. Early rehabilitation, supported by neuroprotective therapy, has the potential to improve outcomes by accelerating neuroplastic adaptation, enhancing functional independence, and improving quality of life.

The main objective of this study is to evaluate the clinical efficacy of neuroprotective therapy as an adjunct to standard rehabilitation in patients who have suffered an ischemic stroke.

Specifically, the study aims to:

1. Determine the extent to which neuroprotective agents contribute to improvements in motor and cognitive function in the early post-stroke rehabilitation period.

2. Assess the differences in functional independence and neurological recovery between patients receiving standard care alone versus those receiving additional neuroprotective therapy.

3. Evaluate the safety and tolerability of commonly used neuroprotective agents—namely Citicoline and Cerebrolysin—in a real-world inpatient neurology setting.

4. Generate clinical insights that could inform the integration of neuroprotective interventions into evidence-based stroke rehabilitation protocols, ultimately contributing to improved long-term outcomes and reduced burden of disability.

Materials and Methods

This prospective observational study was conducted at the Department of Neurology, Samarkand State Medical University, from January to October 2024. A total of 75 patients (age 45–75 years) who were admitted within 14 days of the onset of ischemic stroke were enrolled. Inclusion criteria were: radiologically confirmed ischemic stroke, NIHSS score between 4 and 15, and no prior severe disability. Patients with hemorrhagic stroke, severe renal or hepatic impairment, or known neurodegenerative disorders were excluded. The enrolled patients were randomized into two groups. Group A (n=40) received standard post-stroke care, including antiplatelet therapy, statins, physical rehabilitation, and comorbidity management, along with neuroprotective therapy: Citicoline (1000 mg IV once daily) and Cerebrolysin (10 mL IV once daily) administered for 10 consecutive days starting within the first week of admission. Group B (n=35) received standard care without neuroprotective agents. All patients were evaluated at baseline, after 14 days, and again at a 3-month follow-up using the NIH Stroke Scale (NIHSS) for neurological impairment, the Modified Rankin Scale (mRS) for global disability, the Barthel Index for daily functional independence, and the Montreal Cognitive Assessment (MoCA) for cognitive performance.

Results

At baseline, there were no significant differences between the two groups in terms of demographic characteristics, stroke severity, or comorbidities. On day 14, patients in Group A demonstrated significantly greater neurological improvement as reflected in the reduction of NIHSS scores (mean reduction: 6.8 points vs 4.1 points in Group B; $p<0.01$). Functional outcomes were superior in Group A, with 65% of patients achieving a Barthel Index ≥70 compared to 42% in Group B ($p<0.01$). Modified Rankin Scale scores were similarly improved, with 37.5% of patients in Group A reaching a score of ≤2, indicating functional independence,

versus 18.4% in Group B. Cognitive assessment revealed a significant increase in MoCA scores in Group A (mean improvement: 6.1 points vs 3.2 points in Group B; $p<0.01$). At the 3-month follow-up, gains were sustained and further enhanced. Functional independence (mRS ≤ 2) was achieved by 72.5% of Group A patients compared to 48.5% in Group B. Barthel Index scores continued to favor Group A (mean 86 vs 68; $p<0.01$), and MoCA scores reflected continued cognitive recovery. Importantly, neuroimaging in a subset of patients revealed better preservation of white matter integrity in those who had received neuroprotective agents. No serious adverse effects related to neuroprotective therapy were observed, and treatment was well tolerated.

Discussion

The results of this study suggest that the addition of neuroprotective therapy to standard post-stroke care significantly enhances recovery in the early rehabilitation phase. The superior outcomes observed in Group A across neurological, functional, and cognitive domains highlight the multifaceted benefits of early neuroprotection. Citicoline, a naturally occurring compound involved in phospholipid synthesis and neuronal membrane repair, is known to stabilize cell membranes, reduce ischemia-induced neuronal death, and enhance synaptic function.

Cerebrolysin, a peptide mixture with neurotrophic properties, mimics endogenous growth factors and has been shown to improve outcomes in stroke by promoting neurogenesis, synaptogenesis, and reducing excitotoxicity and inflammation. The observed improvement in MoCA scores is particularly notable, as cognitive deficits post-stroke often go underdiagnosed yet are major determinants of long-term independence and quality of life. These findings align with prior randomized trials and meta-analyses, although variability in study design and outcome measures has previously hindered consensus on clinical implementation. Our study adds valuable real-world evidence supporting the routine use of neuroprotective therapy in early stroke rehabilitation, particularly in settings where access to advanced neurorehabilitation technologies may be limited.

Conclusion

In conclusion, the integration of neuroprotective agents into early rehabilitation protocols for ischemic stroke significantly enhances recovery in neurological, cognitive, and functional dimensions. Patients receiving Citicoline and Cerebrolysin alongside standard care demonstrated superior outcomes on multiple clinical scales and achieved higher rates of functional independence. The early initiation of neuroprotective therapy appears to leverage the brain’s inherent neuroplastic potential, accelerate recovery, and reduce disability.

Given the favorable safety profile, ease of administration, and cost-effectiveness of the agents used, their adoption into routine stroke care is both feasible and beneficial. We recommend the incorporation of neuroprotective therapy as a standard adjunct in early post-stroke rehabilitation, especially in tertiary care centers and neurology departments aiming to optimize patient outcomes.

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