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COMPARATIVE ANALYSIS OF CLINICAL OUTCOMES BETWEEN REPERFUSION THERAPY AND CONSERVATIVE MANAGEMENT IN ACUTE ISCHEMIC STROKE: THE ROLE OF ISCHEMIA-REPERFUSION INJURY

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Background: Acute ischemic stroke (AIS) remains a leading global cause of mortality and long-term disability, with approximately 87% of all strokes being ischemic in nature. The pathophysiological hallmark of AIS is the "ischemic cascade," where the deprivation of oxygen triggers uncontrolled glutamate release, leading to a glutamate-calcium cascade and the eventual death of approximately 1.9 million neurons every minute. While reperfusion therapies like intravenous thrombolysis (IVT) are vital for salvaging the critically hypo-perfused ischemic penumbra, the restoration of blood flow paradoxically induces ischemia-reperfusion injury (IRI). This phenomenon, characterized by oxidative stress, reactive oxygen species (ROS) release, and blood-brain barrier (BBB) disruption, can exacerbate tissue damage and impede functional recovery.

Keywords: Acute Ischemic Stroke, Reperfusion Therapy, Thrombolysis, Ischemia-Reperfusion Injury (IRI), NIHSS, mRS, C-reactive protein, Ischemic Cascade, Penumbra.

Objectives: to perform a comprehensive comparative analysis of the efficacy and safety of reperfusion therapies—specifically intravenous thrombolysis—versus standard conservative medical management in patients with acute ischemic stroke.

Methods: This study involved a comparative analysis of 80 adult patients admitted to a neurological intensive care unit. The cohort was divided into the Reperfusion Group (n=40), who received IVT within the standard therapeutic window, and the Non-Reperfusion Group (n=40), who received conservative medical management. All patients underwent a comprehensive diagnostic protocol including baseline neurological assessment via the National Institutes of Health Stroke Scale (NIHSS). Laboratory screening focused on admission glucose, a full coagulogram (INR, fibrinogen), and inflammatory biomarkers including D-dimer and C-reactive protein (CRP). Neuroimaging included Computed Tomography (CT) to determine ASPECTS scores and real-time hemodynamic monitoring via Transcranial Doppler (TCD) using the Thrombolysis in Brain Ischemia (TIBI) grading system. Early Neurological Improvement (ENI) was defined as an NIHSS reduction of ≥ 4 points within 24 hours, while Early Neurological Deterioration (END) was marked by an increase of ≥ 4 points. Functional outcomes at 90 days were evaluated using the modified Rankin Scale (mRS). Statistical analysis utilized the Mann-Whitney U test for intergroup comparisons and Spearman's rho to correlate biochemical markers with clinical recovery trajectories.

Results: Statistical analysis revealed that the Reperfusion Group achieved significantly better early outcomes, with a mean NIHSS reduction of 7.8 ± 2.6 points compared to only 3.2 ± 1.4 points in the Non-Reperfusion Group (< 0.05). ENI was observed in 62.5% (n=25) of the Reperfusion Group, whereas the Non-Reperfusion Group demonstrated a significantly lower recovery rate of

15.0% ($p < 0.01$). Admission hyperglycemia (> 11.1 mmol/L) and elevated CRP were identified as independent risk factors for poor functional outcomes across both groups. Within the Reperfusion Group, 20% of patients (n=8) experienced Ischemia-Reperfusion Injury (IRI), manifesting as stagnant recovery or END despite successful recanalization (TIBI grades IV-V). Factors significantly triggering IRI included delayed onset-to-treatment time (OTT), admission hyperglycemia, and low post-IVT Platelet-to-Neutrophil Ratios (PNR). Specifically, the IRI subgroup had significantly higher 90-day mRS scores (3.4 ± 0.8) compared to those with successful reperfusion without injury (1.2 ± 0.5, $p < 0.01$). Furthermore, every 10% increase in successful reperfusion efficiency yielded an 11% increase in favorable outcomes (mRS 0–2), though this benefit was mitigated in patients with high baseline D-dimer and fibrinogen levels.

Conclusion: This study confirms that while intravenous thrombolysis is statistically superior to conservative management for restoring functional independence, its efficacy is frequently compromised by the occurrence of ischemia-reperfusion injury. The threat of IRI remains a formidable barrier, increasing the risk of 90-day mortality and functional dependency. To optimize the benefits of reperfusion therapy, it is imperative to move beyond simple recanalization toward a structured, multimodal assessment that addresses the biochemical triggers of IRI—such as hyperglycemia and oxidative stress. Developing neuroprotective strategies that run parallel to reperfusion is essential to mitigate secondary brain injury, minimize long-term disability, and improve the overall quality of care for stroke survivors.