Pathogenetic Correction of Neuroinflammation and Cerebral Edema Using Nimesulide in Severe Traumatic Brain Injury

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Abstract: Severe traumatic brain injury (sTBI) is characterized by accelerated neuroinflammation and progressive brain edema. This study evaluated the clinical efficacy of augmenting standard therapy with Nimesulide, a selective COX-2 inhibitor, in 150 patients. In the Nimesulide-treated group, Glasgow Coma Scale (GCS) scores were significantly higher by Day 10, the incidence of pneumonia and post-traumatic seizures decreased, and neuroimaging and ophthalmoscopy parameters normalized more rapidly.

Keywords: Severe traumatic brain injury, cerebral edema, neuroinflammation, Nimesulide, Glasgow Coma Scale.

Introduction. Severe traumatic brain injury (sTBI) remains a critical issue in neurointensive care due to high mortality and disability rates. Secondary neuroinflammation and the subsequent cytokine storm disrupt the integrity of the blood-brain barrier (BBB), triggering progressive vasogenic and cytotoxic cerebral edema, as well as intracranial hypertension. Conventional osmotic therapy fails to suppress the underlying molecular mechanisms of inflammation. Therefore, implementing targeted pathogenetic treatments that effectively dampen neuroinflammation is of paramount importance.

Study Objective. To evaluate the clinical, laboratory, and instrumental efficacy of incorporating Nimesulide, a selective COX-2 inhibitor, into the comprehensive intensive care protocol for patients with severe traumatic brain injury.

Material and Methods. The study was conducted in the neurointensive care unit of the Bukhara Branch of the Republican Research Center of Emergency Medicine (RRCEM), which serves as the primary clinical base for the Department of Anesthesiology and Intensive Care at the Bukhara State Medical Institute. A total of 150 patients diagnosed with sTBI (GCS score less than or equal to 8 points) were divided into two groups: the Main Group (n=115) received conventional intensive therapy combined with enteral Nimesulide at a dose of 100 mg twice daily for 7 days;

the Control Group (n=35) received standard treatment alone. Patient status was assessed on Days 1, 5, and 10 using the Glasgow Coma Scale (GCS), neutrophil-to-lymphocyte ratio (NLR), multi-spiral computed tomography (MSCT), and ophthalmoscopy.

Results. Upon admission on Day 1, GCS scores reflected an identical baseline severity in both groups (9.1±1.2 points; $p>0.05$). By Day 10 of treatment, consciousness recovered significantly faster in the Nimesulide-augmented group, with the GCS score reaching 13.1±1.7 points, compared to a significantly lower score in the control group (11.5±1.6 points; $p<0.05$).

Regarding laboratory parameters, the main group demonstrated a rapid normalization of the NLR to 1.9±0.2 by Day 10 (compared to 3.0±0.3 in the control group; $p<0.05$), indicating a substantial reduction in systemic immunological stress. MSCT scans revealed a more rapid resolution of brain edema mass-effect signs in the main group. Ophthalmoscopic evaluation showed that the retinal arteriole-to-vein (A:V) ratio improved from a pathological 1:2.2 on Day 1 to a physiological 1:1.5 by Day 10 in the main group, confirming the stabilization of intracranial pressure.

Conclusion. Augmenting the standard neurointensive care protocol with Nimesulide effectively blocks the neuroinflammatory cascade in severe traumatic brain injury. This acceleration reduces the volume of cerebral edema, enhances the rate of consciousness recovery, and minimizes life-threatening complications. The utilization of selective COX-2 inhibitors during the acute phase of sTBI is a safe and pathogenetically justified therapeutic approach.