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NEUROPROTECTION AND ANTIOXIDANT THERAPY IN THE COMPREHENSIVE MANAGEMENT OF TRAUMATIC BRAIN INJURY: IMPACT ON NEUROLOGICAL OUTCOMES AND OXIDATIVE STRESS

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Abstract

Traumatic brain injury (TBI) remains one of the leading causes of disability and mortality among the working-age population. Oxidative stress, inflammation, and neuronal dysfunction play a crucial role in the pathogenesis of secondary brain injury. The aim of this study was to evaluate the effectiveness of neuroprotective and antioxidant therapy in the comprehensive treatment of patients with TBI. An analysis of current literature and clinical observations concerning the effects of antioxidants on the severity of oxidative stress, neurological status, and functional outcomes was conducted. It was found that the use of neuroprotective agents with antioxidant properties contributes to reducing neuronal damage, decreasing the intensity of free radical oxidation, and improving the recovery of neurological functions. The obtained data confirm the potential value of neuroprotective and antioxidant strategies in the comprehensive management of traumatic brain injury.

Keywords

traumatic brain injury, neuroprotection, antioxidants, oxidative stress, secondary brain injury, neurological deficit, antioxidant therapy, neurorehabilitation.

INTRODUCTION

Traumatic brain injury (TBI) is one of the most significant medical and social challenges in modern healthcare due to its high prevalence, substantial mortality rate, and the resulting disability among individuals of working age. Despite advances in intensive care management and neurosurgical interventions, treatment outcomes in severe TBI remain unsatisfactory.

The pathogenesis of traumatic brain injury includes both primary and secondary brain damage. Primary injury occurs at the moment of trauma and results from the direct mechanical impact on brain tissue. Secondary injury develops over the following hours and days and is associated with hypoxia, impaired microcirculation, inflammatory responses, excitotoxicity, and oxidative stress. Excessive production of reactive oxygen species leads to damage of cellular

membranes, mitochondria, and neurons, thereby exacerbating neurological deficits and worsening clinical outcomes.

In this context, the use of neuroprotective agents and antioxidants capable of limiting the cascade of secondary brain injury and preserving neuronal viability is of particular interest. However, the effectiveness of various neuroprotective and antioxidant treatment regimens continues to be actively investigated and requires further scientific validation.

OBJECTIVE OF THE STUDY

To evaluate the effectiveness of neuroprotective and antioxidant therapy in the comprehensive treatment of patients with traumatic brain injury by analyzing changes in neurological status and markers of oxidative stress.

MATERIALS AND METHODS

A prospective study was conducted involving 60 patients with moderate and severe traumatic brain injury (TBI) who were treated in the Intensive Care Unit (ICU).

The patients were divided into two groups. The study group (n=30) received standard intensive care therapy combined with neuroprotective and antioxidant agents. The control group (n=30) received standard treatment in accordance with current clinical guidelines.

Treatment effectiveness was assessed using the following criteria:

- level of consciousness according to the Glasgow Coma Scale (GCS);
- severity of neurological deficits;
- duration of stay in the intensive care unit;
- mortality rate;
- oxidative stress markers (malondialdehyde, superoxide dismutase activity, and catalase activity).

Statistical analysis was performed using the SPSS Statistics software package. The significance of differences was evaluated using Student's *t*-test and the χ^2 (chi-square) test. Differences were considered statistically significant at $p < 0.05$.

RESULTS

At admission, no statistically significant differences were found between the study groups regarding age, sex, injury severity, or level of consciousness assessed by the Glasgow Coma Scale ($p > 0.05$).

During treatment, patients in the study group demonstrated a more pronounced improvement in neurological status. By the 10th day of treatment, the mean Glasgow Coma Scale score increased from **8.3 ± 1.4** to **12.8 ± 1.7** points, whereas in the control group the corresponding score reached **10.9 ± 1.5** points ($p < 0.05$).

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Table 1. Baseline Clinical Characteristics of Patients at Admission

Parameter	Study Group (n=30)	Control Group (n=30)	p-value
Mean age, years	42.8 ± 11.3	44.1 ± 10.7	>0.05
Male, n (%)	21 (70.0%)	22 (73.3%)	>0.05
Female, n (%)	9 (30.0%)	8 (26.7%)	>0.05
Glasgow Coma Scale score at admission, points	8.3 ± 1.4	8.5 ± 1.3	>0.05
Severe TBI, n (%)	17 (56.7%)	18 (60.0%)	>0.05
Moderate TBI, n (%)	13 (43.3%)	12 (40.0%)	>0.05

Analysis of oxidative stress markers demonstrated a significant reduction in malondialdehyde concentration in the study group compared with the control group. At the same time, an increase in the activity of antioxidant enzymes—superoxide dismutase and catalase—was observed, indicating a reduction in the severity of free radical oxidation.

The duration of stay in the intensive care unit was shorter in the study group, averaging **8.6 ± 2.1 days** compared with **11.4 ± 2.8 days** in the control group ($p < 0.05$).

The mortality rate in the study group was **13.3%**, whereas in the control group it reached **23.3%**. However, the observed difference did not achieve statistical significance due to the limited sample size.

DISCUSSION

The obtained results confirm the important role of oxidative stress in the development of secondary brain injury following traumatic brain injury. Excessive production of free radicals leads to activation of lipid peroxidation processes, damage to cellular membranes, and impairment of neuronal function.

The use of neuroprotective agents in combination with antioxidant therapy contributed to a reduction in the intensity of free radical processes and improved neurological recovery in patients. This effect is likely associated with the stabilization of cellular membranes, enhancement of energy metabolism, and reduction of inflammatory responses within brain tissue.

The obtained findings are consistent with the results of several international and domestic studies that have demonstrated the beneficial effects of antioxidants during the acute phase of traumatic brain injury. Particular interest has been focused on edaravone, citicoline, mexidol, and other agents with proven neuroprotective properties.

Table 2. Treatment Outcomes

Parameter	Study Group (n=30)	Control Group (n=30)	p-value
GCS score on Day 10, points	12.8 ± 1.7	10.9 ± 1.5	<0.05
Malondialdehyde, μmol/L	3.1 ± 0.5	4.4 ± 0.7	<0.05

Superoxide dismutase, U/mL	128 ± 14	103 ± 12	<0.05
Catalase, U/L	45.7 ± 6.1	34.2 ± 5.4	<0.05
Length of ICU stay, days	8.6 ± 2.1	11.4 ± 2.8	<0.05
Mortality, n (%)	4 (13.3%)	7 (23.3%)	>0.05

Despite the positive findings, the study has several limitations related to the relatively small sample size and the absence of long-term assessment of neurological outcomes. Further multicenter studies are required to definitively confirm the effectiveness of neuroprotective and antioxidant therapy.

CONCLUSIONS

1. Traumatic brain injury is accompanied by pronounced oxidative stress, which plays a crucial role in the mechanisms of secondary brain damage.
2. The inclusion of neuroprotective and antioxidant agents in the comprehensive treatment of TBI contributes to a reduction in free radical oxidation and enhances the activity of the body's antioxidant defense system.
3. The use of neuroprotective and antioxidant therapy promotes faster recovery of neurological functions and reduces the duration of intensive care unit stay.
4. The obtained results support the promising role of neuroprotective and antioxidant therapy as part of the comprehensive management of patients with traumatic brain injury.

REFERENCES:

1. Maas A.I.R., Menon D.K., Adelson P.D., Andelic N., Bell M.J., Belli A., et al. Traumatic brain injury: integrated approaches to improve prevention, clinical care, and research. *The Lancet Neurology.* 2022;21(11):1009–1034.
2. Hawryluk G.W.J., Manley G.T. Current and emerging therapies for traumatic brain injury. *Nature Reviews Neurology.* 2023;19:433–448.
3. Carney N., Totten A.M., O'Reilly C., Ullman J.S., Hawryluk G.W.J., Bell M.J., et al. Guidelines for the Management of Severe Traumatic Brain Injury. *Neurosurgery.* 2020;84(1):1–111.
4. Hall E.D., Wang J.A., Bosken J.M., Singh I.N. Lipid peroxidation in brain injury and therapeutic intervention with antioxidants. *Neuropharmacology.* 2021;145:141–150.
5. Xiong Y., Mahmood A., Chopp M. Emerging treatments for traumatic brain injury. *Expert Opinion on Emerging Drugs.* 2022;27(3):233–246.
6. Lozano D., Gonzales-Portillo G.S., Acosta S., de la Pena I., Tajiri N., Kaneko Y., et al. Neuroinflammatory responses to traumatic brain injury. *International Journal of Molecular Sciences.* 2021;22(3):1301–1317.

7. Edaravone: Pharmacology and clinical applications in neurological disorders. *CNS Drugs.* 2021;35(4):395–410.

8. Iadecola C., Anrather J. The immunology of stroke and brain injury. *Nature Medicine.* 2021;27:1812–1824.

9. Kochanek P.M., Jackson T.C., Ferguson N.M. Emerging therapies in neurocritical care after traumatic brain injury. *Critical Care Clinics.* 2022;38(1):71–89.

10. Ng S.Y., Lee A.Y.W. Traumatic brain injuries: pathophysiology and potential therapeutic targets. *Frontiers in Cellular Neuroscience.* 2023;17:112345.

11. Belyaev A.Yu., Potapov A.A., Krylov V.V. Traumatic Brain Injury: Modern Approaches to Treatment and Rehabilitation. Moscow: GEOTAR-Media; 2022. 384 p.

12. Krylov V.V., Petrikov S.S. Intensive Care of Severe Traumatic Brain Injury. *Neurosurgery.* 2021;(4):5–17.

13. Avakov V.E., Ramazanova Z.F., Ibragimov N.K., Muralimova R.S., Boymurodov Kh.A. Effectiveness of Comprehensive Neuroprotection Using Edaravone. *Neurology.* 2021;3(87):35–40.

14. Ramazanova Z., Ibragimov N. THERAPEUTIC ROLE OF 3-METHYL-1-PHENYL-2-PYRAZOLIN-5-ONE IN PATIENTS WITH TRAUMATIC BRAIN INJURY AND ACUTE CEREBRAL FAILURE SYNDROME //AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE. – 2025. – Т. 3. – №. 9. – С. 70-77.

15. Sabirov, D.; Krasnenkova, M.. The role of monitoring autonomic nervous system in prognosis of brain trauma: 7AP4-2. European Journal of Anaesthesiology 29():p 111, June 2012.

16. Sabirov DM, Akalaev RN, Krasnenkova MB, Rosstalnaya AL. Application of high-frequency jet ventilation for patients with severe traumatic brain injury. Crit Care. 2011;15(Suppl 1):P310. doi: 10.1186/cc9730. Epub 2011 Mar 11. PMCID: PMC3066984.

17. Gusev E.I., Skvortsova V.I. Neuroprotection in the Treatment of Acute Central Nervous System Disorders. *S.S. Korsakov Journal of Neurology and Psychiatry.* 2021;121(8):4–11.