



CLINICAL OUTCOMES OF SEVERE TRAUMATIC BRAIN INJURY DEPENDING ON THE TYPE OF BRAIN DAMAGE AND THE SEVERITY OF TRAUMATIC DISLOCATION SYNDROME

Abdulkhakov Parvoz Vaxob ugli

The specialized scientific and practical center for neurosurgery and neurorehabilitation at Samarkand State Medical University

Abstract.

Background: Severe traumatic brain injury (sTBI) remains one of the leading causes of mortality and long-term disability worldwide. Clinical outcomes are largely determined by the morphological type of brain injury and the severity of traumatic dislocation syndrome (TDS), reflecting the extent of intracranial hypertension and brainstem involvement.

Objective: To evaluate treatment outcomes in patients with severe traumatic brain injury depending on the type of brain damage and the severity of traumatic dislocation syndrome.

Materials and Methods: A retrospective analysis of 216 patients with sTBI was conducted. Brain injury type was classified as diffuse brain injury or dislocation-type injury based on clinical course and computed tomography findings. The severity of TDS was staged according to the level of consciousness, brainstem reflex impairment, and vital function disturbances. Outcomes were assessed using the Glasgow Outcome Scale.

Results: Diffuse brain injury was associated with predominantly unfavorable outcomes, especially in patients with deep coma, with mortality reaching up to 69%. Early stages of TDS (I–II) were characterized by a significantly higher proportion of favorable outcomes, whereas advanced stages (III–V) demonstrated extremely high mortality rates, approaching 100% in terminal stages.

Conclusion: The type of brain damage and the severity of traumatic dislocation syndrome are decisive prognostic factors in severe traumatic brain injury. Early identification and management of dislocation processes are crucial for improving clinical outcomes.

Keywords: severe traumatic brain injury, diffuse brain injury, traumatic dislocation syndrome, intracranial hypertension, Glasgow Outcome Scale.

КЛИНИЧЕСКИЕ ИСХОДЫ ТЯЖЁЛОЙ ЧЕРЕПНО-МОЗГОВОЙ ТРАВМЫ В ЗАВИСИМОСТИ ОТ ВИДА ПОВРЕЖДЕНИЯ ГОЛОВНОГО МОЗГА И СТЕПЕНИ ТРАВМАТИЧЕСКОГО ДИСЛОКАЦИОННОГО СИНДРОМА

Абдулхакимов Парвоз Вахоб угли

Специализированный научно-практический центр нейрохирургии и нейрореабилитации при Самаркандском государственном медицинском университете

Аннотация.

Актуальность: Тяжёлая черепно-мозговая травма (ТЧМТ) остаётся одной из ведущих причин летальности и стойкой инвалидизации. Исходы заболевания в значительной степени определяются видом повреждения головного мозга и выраженностью травматического дислокационного синдрома (ТДС), отражающего степень внутричерепной гипертензии и поражения стволовых структур.

Цель исследования: Оценить исходы лечения больных ТЧМТ в зависимости от вида повреждения головного мозга и степени травматического дислокационного синдрома.

Материал и методы: Проведён ретроспективный анализ 216 больных с ТЧМТ. Вид поражения головного мозга классифицировали как диффузный или дислокационный на основании клинических данных и результатов компьютерной томографии. Стадии ТДС

определяли по глубине угнетения сознания, нарушению стволовых рефлексов и витальных функций. Исходы оценивали по шкале исходов Глазго.

Результаты: Диффузное повреждение головного мозга характеризовалось преимущественно неблагоприятными исходами, особенно при глубокой коме, с летальностью до 69%. Ранние стадии ТДС (I–II) ассоциировались с более благоприятным прогнозом, тогда как при поздних стадиях (III–V) летальность достигала почти 100%.

Заключение: Вид повреждения головного мозга и степень ТДС являются ключевыми прогностическими факторами при ТЧМТ. Ранняя диагностика и своевременная коррекция дислокационных процессов имеют решающее значение для улучшения исходов.

Ключевые слова: тяжёлая черепно-мозговая травма, диффузное повреждение мозга, травматический дислокационный синдром, внутричерепная гипертензия, шкала исходов Глазго.

OG‘IR BOSH MIYA JAROHATIDA DAVOLASH NATIJALARI: BOSH MIYANING SHIKASTLANISH TURI VA TRAVMATIK DISLOKATSION SINDROM DARAJASIGA BOG‘LIQLIGI

Abdulkhaimov Parvoz Vaxob o‘g‘li

Samarqand davlat tibbiyot universiteti huzuridagi
Ixtisoslashtirilgan ilmiy-amaliy neyroxirurgiya va neyroreabilitatsiya markazi

Annotatsiya.

Dolzarbli: Og‘ir bosh miya jarohati (OBMJ) butun dunyoda o‘lim va doimiy nogironlikning yetakchi sabablaridan biri bo‘lib qolmoqda. Klinik natijalar ko‘p jihatdan bosh miyaning shikastlanish turi va travmatik dislokatsion sindrom (TDS) darajasi bilan belgilanadi, bu esa intrakranial gipertenziya va miya ustuni zararlanishining og‘irligini aks ettiradi.

Tadqiqot maqsadi: Og‘ir bosh miya jarohati bilan og‘rigan bemorlarda davolash natijalarini bosh miyaning shikastlanish turi va travmatik dislokatsion sindrom darajasiga qarab baholash.

Material va usullar: 216 nafar OBMJ bilan og‘rigan bemorlarning davolash natijalari retrospektiv tahlil qilindi. Klinik kechish va kompyuter tomografiya ma’lumotlariga asoslanib, bosh miya shikastlanishi diffuz yoki dislokatsion turga ajratildi. TDS bosqichlari hushning pasayishi, miya ustuni reflekslari va hayotiy funksiyalar buzilishi asosida baholandi. Natijalar Glasgow Outcome Scale bo‘yicha tahlil qilindi.

Natijalar: Diffuz bosh miya shikastlanishi, ayniqsa chuqur koma bilan kechganda, noqulay klinik natijalar va yuqori o‘lim ko‘rsatkichi (69% gacha) bilan tavsiflandi. TDS ning erta bosqichlarida (I–II) nisbatan qulay prognoz kuzatildi, kech bosqichlarda (III–V) esa o‘lim darajasi deyarli 100% ga yetdi.

Xulosa: Bosh miyaning shikastlanish turi va travmatik dislokatsion sindrom darajasi OBMJ da asosiy prognostik omillar hisoblanadi. Dislokatsion jarayonlarni erta aniqlash va o‘z vaqtida davolash klinik natijalarni yaxshilashda muhim ahamiyatga ega.

Kalit so‘zlar: og‘ir bosh miya jarohati, diffuz bosh miya shikastlanishi, travmatik dislokatsion sindrom, intrakranial gipertenziya, Glasgow Outcome Scale

Introduction. Severe traumatic brain injury (sTBI) remains one of the most challenging and socially significant problems in modern neurosurgery, emergency medicine, and intensive care. Despite advances in neuroimaging, neurocritical care, and surgical techniques, sTBI continues to be associated with high mortality rates, substantial long-term disability, and severe impairment of quality of life among survivors. According to epidemiological data from the World Health Organization and large multicenter studies, traumatic brain injury represents a leading cause of death and disability in individuals of working age worldwide, with severe forms accounting for the majority of unfavorable outcomes.

The pathophysiology of sTBI is complex and multifactorial, involving both primary and secondary mechanisms of brain injury. Primary injury occurs at the moment of trauma and includes diffuse

axonal injury, focal contusions, intracranial hematomas, and brainstem damage. Secondary injury evolves over hours to days and is mediated by cerebral edema, ischemia, excitotoxicity, inflammatory cascades, mitochondrial dysfunction, and intracranial hypertension, ultimately leading to cerebral herniation and brainstem compression. Among the most critical determinants of outcome in sTBI are the type of brain damage and the severity of traumatic dislocation syndrome (TDS). Diffuse brain injury, particularly diffuse axonal injury involving subcortical, thalamic, and brainstem structures, is often associated with early and profound impairment of consciousness and poor neurological recovery. Conversely, focal mass lesions such as epidural, subdural, or intracerebral hematomas may be potentially reversible if timely surgical decompression is performed, although their progression frequently leads to intracranial hypertension and secondary brain displacement.

Traumatic dislocation syndrome reflects the degree of brain shift and herniation resulting from intracranial mass effect, edema, and cerebrospinal fluid circulation disturbances. Progressive stages of TDS are characterized by worsening depression of consciousness, brainstem reflex abnormalities, and failure of vital functions. Numerous clinical observations suggest that the stage of TDS at the time of treatment initiation is a crucial prognostic factor, often outweighing other demographic or clinical variables. Despite extensive literature on sTBI, there remains a need for integrated analyses that simultaneously evaluate outcomes in relation to both the morphological type of brain injury and the severity of traumatic dislocation syndrome. Such an approach allows for a more nuanced understanding of prognostic stratification and may contribute to improved clinical decision-making, optimization of treatment algorithms, and refinement of outcome prediction models. The aim of the present study was to assess the outcomes of treatment in patients with severe traumatic brain injury depending on the type of brain damage and the severity of traumatic dislocation syndrome, using standardized clinical and neuroimaging criteria and outcome assessment by the Glasgow Outcome Scale.

Materials and Methods. This retrospective observational study analyzed the treatment outcomes of 216 patients with severe traumatic brain injury who were managed in a specialized neurosurgical and intensive care setting. Severe TBI was defined according to accepted clinical criteria, including a Glasgow Coma Scale (GCS) score of 8 points or less upon admission or during the early post-traumatic period. The study population consisted of 159 men (73.6%) and 57 women (26.4%), with an age range from 17 to 74 years. All patients sustained isolated severe traumatic brain injury without concomitant life-threatening extracranial injuries that could independently influence survival or neurological outcomes. The type of brain injury was determined based on a comprehensive assessment of clinical presentation, dynamics of consciousness impairment, and findings from computed tomography (CT) of the brain. Neuroimaging studies were performed upon admission and repeated as clinically indicated to monitor lesion progression, mass effect, and signs of intracranial hypertension.

Diffuse brain damage was diagnosed in cases characterized by immediate depression of consciousness following trauma, in the absence of a dominant focal mass lesion, and with the presence of multiple contusional or hemorrhagic foci in subcortical, thalamic, and/or brainstem regions. These findings were interpreted as consistent with diffuse axonal injury and diffuse traumatic brain damage. A total of 41 patients (18.9%) met these criteria and were classified as having diffuse brain injury. Dislocation-type brain injury was diagnosed when CT imaging revealed intracranial hematomas and/or focal areas of brain contusion or crushing associated with compression of adjacent brain structures, midline shift, ventricular deformation, or signs of transtentorial or central herniation. This group comprised 175 patients (81.1%) and represented cases in which traumatic dislocation syndrome was a dominant pathophysiological component. The severity of traumatic dislocation syndrome was staged based on an integrated evaluation of the level of consciousness, brainstem reflexes, pupillary responses, respiratory patterns, and cardiovascular stability. This clinical-neurological staging reflected progressive involvement of brainstem structures and increasing failure of vital regulatory mechanisms. TDS was categorized into five stages, from early (I–II) to terminal (V), corresponding to increasing severity of brain displacement and herniation. Treatment strategies included standardized neurointensive care

measures aimed at controlling intracranial pressure, maintaining adequate cerebral perfusion pressure, preventing secondary brain injury, and correcting systemic physiological disturbances. Surgical interventions, including decompressive craniectomy and evacuation of intracranial hematomas, were performed when indicated based on clinical and radiological criteria.

Treatment outcomes were assessed using the Glasgow Outcome Scale (GOS), which classifies outcomes into good recovery, moderate disability, severe disability, vegetative state, and death. For analytical purposes, good recovery and moderate disability were considered favorable outcomes, whereas severe disability, vegetative state, and death were classified as unfavorable outcomes. Statistical analysis was descriptive in nature, focusing on the distribution of outcomes across different categories of brain injury type and TDS severity. Percentages were calculated to illustrate outcome trends and prognostic associations.

Results. Analysis of treatment outcomes demonstrated a strong dependence on both the type of brain damage and the severity of traumatic dislocation syndrome.

In patients with diffuse brain injury accompanied by depression of consciousness corresponding to coma grade I, favorable outcomes in the form of good recovery or moderate disability were observed in only 8.3% of cases. Severe disability was documented in 33.3% of patients, while 16.7% evolved into a persistent vegetative state. Mortality in this subgroup reached 41.7%, underscoring the grave prognosis associated with diffuse traumatic brain damage even at relatively less profound levels of coma. In contrast, patients with diffuse brain injury and deeper impairment of consciousness (coma grade II) exhibited an even more unfavorable outcome profile. In this subgroup, no cases of good recovery or moderate disability were recorded. Severe disability occurred in only 3.4% of patients, whereas vegetative state was observed in 27.6%. Mortality was exceedingly high, reaching 69%, reflecting extensive involvement of subcortical and brainstem structures and limited potential for neurological recovery. When outcomes were analyzed according to the stage of traumatic dislocation syndrome, a clear gradient of worsening prognosis with increasing TDS severity was identified.

In early stages of TDS (stages I and II), outcomes were comparatively favorable. In TDS stage I, good recovery or moderate disability was achieved in 96.3% of patients, with no cases of severe disability or vegetative state, and a mortality rate of only 3.7%. In TDS stage II, favorable outcomes were observed in 48.1% of patients, while severe disability occurred in 19.8%, vegetative state in 5.7%, and mortality increased to 26.4%. These findings highlight the critical importance of early detection and intervention before the progression of dislocation processes.

At TDS stage III, the prognosis deteriorated dramatically. No cases of good recovery or moderate disability were observed. Severe disability was recorded in 4.9% of patients, vegetative state in 8%, and mortality reached 87.1%. This stage reflects advanced brainstem involvement with profound impairment of vital regulatory functions. In TDS stage IV, outcomes were almost uniformly fatal. Favorable outcomes and severe disability were not observed. Vegetative state was documented in only 3.6% of patients, while mortality reached 96.4%, indicating near-complete failure of compensatory mechanisms and irreversible brainstem damage.

At TDS stage V, representing terminal dislocation syndrome, all patients (100%) died despite intensive therapeutic measures. Comparative analysis revealed that patients with diffuse brain injury and coma grade I had outcomes that were generally less favorable than those observed in early stages of traumatic dislocation syndrome. However, in cases of diffuse brain injury with coma grade II, the prognosis was relatively more favorable compared to late stages of TDS (stages III–V), in which mortality approached absolute values.

Discussion. The present study confirms that both the morphological type of brain injury and the severity of traumatic dislocation syndrome are decisive factors influencing the outcomes of severe traumatic brain injury. The data demonstrate a clear stratification of prognosis based on these parameters, reflecting underlying pathophysiological mechanisms and the potential reversibility of brain damage.

Diffuse brain injury, particularly when associated with early and sustained depression of consciousness, represents one of the most severe forms of traumatic brain damage. The predominance of unfavorable outcomes in this group can be attributed to widespread axonal

disruption, involvement of deep midline and brainstem structures, and limited surgical treatment options. Unlike focal mass lesions, diffuse injury lacks a clearly defined target for decompression, and therapeutic efforts are largely supportive and aimed at mitigating secondary injury processes. The exceptionally high mortality and vegetative state rates observed in patients with diffuse brain injury and deeper coma underscore the devastating impact of brainstem dysfunction. These findings are consistent with previous neuropathological and neuroimaging studies demonstrating that diffuse axonal injury affecting the reticular activating system and brainstem nuclei is a major determinant of persistent unconsciousness and poor neurological recovery.

Traumatic dislocation syndrome, on the other hand, represents a dynamic and potentially modifiable process, particularly in its early stages. The favorable outcomes observed in TDS stages I and II emphasize the importance of timely diagnosis and aggressive management of intracranial hypertension and mass effect. Early surgical decompression, optimization of cerebral perfusion, and meticulous neurointensive care can prevent progression to irreversible brainstem damage. The sharp transition to overwhelmingly fatal outcomes in TDS stages III–V reflects the narrow therapeutic window inherent to severe brain displacement and herniation. Once critical brainstem centers are compromised, compensatory mechanisms fail, and the likelihood of meaningful neurological recovery becomes negligible. These observations highlight the prognostic value of TDS staging as a practical clinical tool for outcome prediction and treatment planning. The comparative analysis between diffuse brain injury and traumatic dislocation syndrome reveals important nuances. While diffuse brain injury with coma grade I already carries a poor prognosis compared to early TDS stages, its outcomes may still be relatively better than those associated with advanced dislocation syndromes. This suggests that the extent and localization of brainstem involvement, rather than the mere depth of consciousness impairment, play a pivotal role in determining survival and functional outcome. From a clinical perspective, these findings underscore the necessity of early and accurate differentiation between diffuse and dislocation-type brain injuries, as well as precise staging of traumatic dislocation syndrome. Such differentiation informs not only prognosis but also therapeutic priorities, including the urgency of surgical intervention and the intensity of neurocritical care.

Conclusion. The results of this study demonstrate that the type of brain damage and the severity of traumatic dislocation syndrome exert a profound and independent influence on the outcomes of severe traumatic brain injury. Diffuse brain injury, particularly when accompanied by early and deep depression of consciousness, is associated with predominantly unfavorable outcomes and high mortality. Traumatic dislocation syndrome shows a clear stage-dependent prognosis, with favorable outcomes possible in early stages and almost universal mortality in advanced stages. Outcomes in patients with diffuse brain injury and coma grade I are less favorable than those observed in early stages of traumatic dislocation syndrome. At the same time, diffuse brain injury with coma grade II may carry a relatively better prognosis compared to late stages of traumatic dislocation syndrome, in which outcomes are overwhelmingly fatal. These findings highlight the critical importance of early diagnosis, accurate neuroimaging assessment, and timely therapeutic intervention in patients with severe traumatic brain injury. Incorporation of brain injury type and traumatic dislocation syndrome staging into clinical decision-making algorithms may improve prognostic accuracy and contribute to the optimization of treatment strategies in this challenging patient population.

References.

1. Bullock, R., Chesnut, R. M., Ghajar, J., Gordon, D., Hartl, R., Newell, D. W., ... Wilberger, J. E. (2006). Surgical management of traumatic brain injury. *Neurosurgery*, 58(3), S2-1–S2-62.
2. Maas, A. I. R., Menon, D. K., Adelson, P. D., Andelic, N., Bell, M. J., Belli, A., ... Yaffe, K. (2017). Traumatic brain injury: Integrated approaches to improve prevention, clinical care, and research. *The Lancet Neurology*, 16(12), 987–1048.
3. Gennarelli, T. A., & Graham, D. I. (1998). Neuropathology of the head injuries. *Seminars in Clinical Neuropsychiatry*, 3(3), 160–175.
4. Likhterman, L. B. (2009). *Traumatic brain injury: Pathogenesis, diagnosis, treatment*. Moscow: GEOTAR-Media.

5. Adams, J. H., Graham, D. I., Murray, L. S., & Scott, G. (1982). Diffuse axonal injury due to nonmissile head injury in humans. *Journal of Neurology, Neurosurgery & Psychiatry*, 45(11), 1034–1044.
6. Jennett, B., & Bond, M. (1975). Assessment of outcome after severe brain damage. *The Lancet*, 305(7905), 480–484.
7. Marshall, L. F., Marshall, S. B., Klauber, M. R., Van Berkum Clark, M., Eisenberg, H., Jane, J. A., ... Foulkes, M. A. (1991). A new classification of head injury based on computerized tomography. *Journal of Neurosurgery*, 75(1S), S14–S20.
8. Ghajar, J. (2000). Traumatic brain injury. *The Lancet*, 356(9233), 923–929.
9. Becker, D. P., Miller, J. D., Ward, J. D., Greenberg, R. P., Young, H. F., & Sakalas, R. (1977). The outcome from severe head injury with early diagnosis and intensive management. *Journal of Neurosurgery*, 47(4), 491–502.
10. Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. *The Lancet*, 304(7872), 81–84.
11. Saatman, K. E., Duhaime, A. C., Bullock, R., Maas, A. I. R., Valadka, A., & Manley, G. T. (2008). Classification of traumatic brain injury for targeted therapies. *Journal of Neurotrauma*, 25(7), 719–738.
12. Unterberg, A. W., Stover, J., Kress, B., & Kiening, K. L. (2004). Edema and brain trauma. *Neuroscience*, 129(4), 1021–1029.
13. Roozenbeek, B., Maas, A. I. R., & Menon, D. K. (2013). Changing patterns in the epidemiology of traumatic brain injury. *Nature Reviews Neurology*, 9(4), 231–236.
14. Carney, N., Totten, A. M., O'Reilly, C., Ullman, J. S., Hawryluk, G. W. J., Bell, M. J., ... Ghajar, J. (2017). Guidelines for the management of severe traumatic brain injury. *Neurosurgery*, 80(1), 6–15.
15. Yablonskiy, P. K., & Khitrin, L. K. (2005). Intracranial hematomas in traumatic brain injury. *Neurosurgical Journal*, 4, 15–23.