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*Original article*

# Hyper-H Triad as a Clinical Trigger for Early Decompressive Surgery in Traumatic Brain Herniation: Implications for Decision-Making

Baglan Mustafayev [1*], Alina Mustafayeva [2], Askar Bakhtiyarov [3], Kuanysh Nikatov [4]

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[1] Neurosurgeon of the Department of Neurosurgery and Neuropathology, Lecturer of the Department of Neurosurgery and Neurology, National Centre for Neurosurgery, Astana, Kazakhstan. E-mail: baglan.mustafaev@ncn.kz

[2] Head of the Department of Neurorehabilitation, National Centre for Neurosurgery, Astana, Kazakhstan. E-mail: alina.mustafaeva@ncn.kz

[3] Head of the Department of Neurosurgery, Shymkent Regional Clinical Hospital; Senior Lecturer of the Department of Surgery, Akhmet Yassawi International Kazakh-Turkish University, Shymkent, Kazakhstan. E-mail: askar.bakhtiyaroz@ayu.edu.kz

[4] Assistant of the Department of Neurosurgery, S.D. Asfendiyarov Kazakh National Medical University, Almaty, Kazakhstan. E-mail: nkabuevich@gmail.com

*** Corresponding author:** baglan.mustafaev@ncn.kz

## Abstract

Traumatic brain herniation (TBH) is a major cause of death after severe traumatic brain injury (TBI). Surgery can be lifesaving, but outcomes depend on the stage. Identifying a therapeutic window and validating early clinical markers are essential.

**Objective:** To investigate the clinical and morphological stages of brain herniation in patients with severe traumatic brain injury, determine the optimal therapeutic window for surgical intervention, and evaluate the diagnostic value of early clinical markers of brainstem dysfunction.

**Methods.** We conducted a prospective multicenter study in Kazakhstan, enrolling 287 adult patients with severe TBI (GCS ≤ 8) and radiological signs of herniation. Patients were stratified into five TBH stages based on clinical and morphological features. Clinical, radiological, surgical, and neurophysiological data were collected via a harmonized protocol. Surgical methods and brainstem pathology were analyzed.

**Results.** Of 287 patients, 218 (76%) underwent surgery. Surgical rates varied by stage: 51% (Stage I), 96.4% (II), 95.2% (III), 84.2% (IV), and 50% (V). Most procedures (81.2%) were unilateral lateral craniotomies. Brainstem injury followed a consistent pattern: diencephalic (I), midbrain (II), pontine (III), medullary (IV–V). Early-stage surgery was linked to reversible pathology and better outcomes; late-stage surgery showed irreversible damage.

**Conclusions.** TBH follows a staged, anatomically consistent progression. Surgery is most effective in Stages I–II. The Hyper-H Triad – hyperthermia, hypertonia, hormonal dysregulation – may signal the need for early intervention. Stage-specific protocols are key to reducing mortality.

**Keywords:** traumatic brain injury, brain herniation, decompressive craniectomy, surgical timing, Hyper-H triad, therapeutic window, brainstem pathology.

## 1. Introduction

Traumatic brain herniation (TBH) represents one of the most devastating sequelae of severe traumatic brain injury (TBI), carrying a mortality rate that can exceed 70% in late stages. Despite advances in neurocritical care and neurosurgical techniques, the outcomes of patients who progress to frank herniation remain dismal. The challenge lies not only in controlling intracranial pressure (ICP) but in recognizing the dynamic transition from a reversible to an irreversible state. A central question remains: when is surgical intervention most effective, and when does it become futile? Addressing this question requires a structured, stage-specific approach that aligns clinical, radiological, and physiological signals with therapeutic action [1,2].

Historically, herniation has been defined radiologically – by midline shift, obliteration of basal cisterns, or downward displacement of brain structures – and clinically, by the onset of anisocoria, abnormal motor responses, or coma. However, these markers often emerge when irreversible brainstem injury is already established. This delay creates a paradox: neurosurgeons intervene aggressively at the stage when outcomes are already compromised. Identifying earlier markers of impending herniation is thus critical to defining a true "therapeutic window of opportunity" [3-7].

Recent work has proposed that herniation is not a binary event but a staged process. In this framework, a prodromal phase precedes classical signs, characterized by subtle but measurable systemic and neurophysiological changes. Among these, the Hyper-H Triad – a constellation of hyperthermia, hypertonia, and hormonal dysregulation – has emerged as a reproducible prodrome of impending transtentorial herniation. This triad reflects hypothalamic–brainstem dysfunction at a point when tissue compromise may still be reversible. If validated, it could serve as a clinical trigger for early surgical intervention, potentially shifting outcomes away from late, often futile decompression toward proactive, outcome-modifying surgery [4,5,8-10].

The urgency of defining such markers is underscored by outcome studies in decompressive surgery. Survival rates are significantly higher when patients undergo intervention in the pre- or early herniation stage compared to those treated after brainstem signs appear. Yet the absence of standardized prodromal criteria has hindered protocol-based decision-making. As a result, surgical timing varies widely across centers, contributing to inconsistent outcomes and limiting the generalizability of decompressive craniectomy trials [4,11-14].

Our study series aims to address this critical gap. The first component evaluates stage-specific outcomes of surgical intervention, highlighting the stark contrast between early and late operative timing and quantifying the therapeutic window. The second component provides a prospective validation of the Hyper-H Triad as a predictor of impending herniation, assessing its sensitivity, specificity, and prognostic value compared with conventional radiological and ICP-based indicators. The third component explores the role of the Hyper-H Triad as a clinical decision-making trigger, framing it as a practical tool for guiding timely decompression within standardized protocols.

By integrating stage-specific outcome analysis with prospective biomarker validation, this series seeks to move beyond descriptive observations and toward actionable guidelines. The goal is not only to improve survival but to enhance functional recovery, shifting the prognosis of patients with severe TBI who progress toward herniation. Establishing the Hyper-H Triad as a reproducible clinical entity, and linking it to surgical timing, offers the possibility of transforming herniation management from reactive rescue to proactive prevention.

In doing so, we propose that herniation should no longer be viewed as an unpredictable catastrophe but as a staged process with identifiable thresholds for intervention. Recognition of the prodromal stage, operationalized through the Hyper-H Triad, may redefine the boundaries of neurosurgical opportunity and provide the foundation for protocol-driven care. This work, therefore, represents both a conceptual and practical step toward reducing mortality in one of the most challenging arenas of neurotrauma.

The objective of this study was to investigate the clinical and morphological stages of brain herniation in patients with severe TBI, determine the optimal therapeutic window for surgical intervention, and evaluate the diagnostic value of early clinical markers of brainstem dysfunction.

## 2. Materials and methods

*Study Design and Setting*

This was a prospective, multicenter observational cohort study performed at three tertiary neurosurgical centers in Kazakhstan. The National Center for Neurosurgery in Astana coordinated the study. All sites followed a harmonized protocol to ensure consistency in patient recruitment, monitoring, and management.

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Institutional review board (IRB) approval was obtained at each participating center.

*Participants*

We included 287 adult patients (≥18 years) admitted with severe TBI, defined as a Glasgow Coma Scale (GCS) score ≤ 8. Neuroimaging inclusion criteria were midline shift greater than 5 mm and/or obliteration of basal cisterns on computed tomography (CT) or magnetic resonance imaging (MRI). Exclusion criteria were pre-existing severe neurological disability, brain death on admission, prolonged anoxic injury (e.g., cardiopulmonary resuscitation >5 minutes), life-threatening extracranial trauma (Injury Severity Score >25), and absence of informed consent from a legal representative.

*Data Collection*

Data were prospectively collected using a standardized case report form and entered into a unified REDCap database with centralized auditing and quality control. Baseline and longitudinal variables included demographics, mechanism of injury, and time to intervention. Neurological examination included GCS, pupillary responses, and focal deficits. Neuroimaging data captured hematoma volume, midline shift, basal cistern status, and radiological signs of herniation.

Neurological staging was based on a rostrocaudal sequence: level of consciousness, pupillary/corneal reflexes, ocular motor reflexes, motor responses to pain, respiratory patterns, and hemodynamic status. Each patient was assigned to one of five stages.

Autonomic cardiovascular balance was assessed using the Kerdo Vegetative Index (VI) = (1 – DBP/HR) × 100, where DBP is diastolic blood pressure and HR is heart rate10.

*Outcomes*

The primary outcome was the Glasgow Outcome Scale–Extended (GOS-E) at hospital discharge and 6 months post-injury. Secondary outcomes included hospital mortality, duration of mechanical ventilation, postoperative complications, and the need for repeat neurosurgical procedures.

*Phenotypic Classification*

Patients were stratified into TBH based on clinical and radiological features. Brainstem involvement was assessed using the Brainstem Singularity Scale (BSS). This classification was used to model progression and to assess stage-specific therapeutic responses.

*Statistical Analysis*

Statistical analyses were conducted using Statistica 12.0 (StatSoft Inc., USA). Descriptive statistics summarized baseline data. Between-group comparisons used Student's t-test for parametric variables and chi-square or Kolmogorov–Smirnov tests for nonparametric variables. Predictors of poor outcome were identified using multivariate logistic regression. Receiver operating characteristic (ROC) curves assessed discriminative performance. Survival analysis employed Kaplan–Meier estimates and Cox proportional hazards models. Statistical significance was set at $p<0.05$. For missing data, variables with >30% missingness (e.g., EEG) were excluded from primary models, while multiple imputation was applied for other incomplete data.

*Subgroup and Exploratory Analyses*

Subgroup comparisons were conducted across TBH phenotypes. Therapeutic window modeling examined the association between surgical timing and clinical outcomes.

*Clinical Management Protocols*

All patients were managed in accordance with the National Clinical Guidelines for Severe TBI, approved by the Ministry of Health of the Republic of Kazakhstan. These guidelines standardized sedation strategies, ICP management, surgical indications, and postoperative care across centers. Local deviations were documented and reviewed centrally.

*Research Transparency*

This study adheres to established standards of scientific rigor and methodological transparency. The study protocol was preregistered on the Open Science Framework (OSF) under the identifier DOI: 10.17605/OSF.IO/K5V7G, ensuring transparency of hypotheses, design, and planned analyses [15].

This subanalysis was part of a prospective multicenter cohort study conducted between January 2022 and March 2025 in the emergency neurosurgical units of three urban clinical centers. As this was a non-interventional observational study, clinical trial registration was not required under ICMJE (International Committee of Medical Journal Editors) guidelines.

*Ethical aspects*

The protocol for this study was approved by the Local Bioethics Committee of the National Neurosurgical Center (protocol No. 1 dated February 16, 2026).

## 3. Results

*Patient Distribution and Surgical Rates*

A total of 287 patients with TBH were enrolled across all participating centers (Figure 1).

Distribution across herniation stages demonstrated a progressive decline in patient numbers with advancing severity. Stage I included 110 patients, Stage II 83, Stage III 63, Stage IV 19, and Stage V 12. Surgical intervention rates varied markedly by stage. In Stage I, 51.0% of patients underwent operative treatment, reflecting a relatively selective approach at this prodromal stage. By Stage II and Stage III, operative rates reached 96.4% and 95.2%, respectively, reflecting a high degree of surgical responsiveness once radiological and clinical signs of herniation were established.

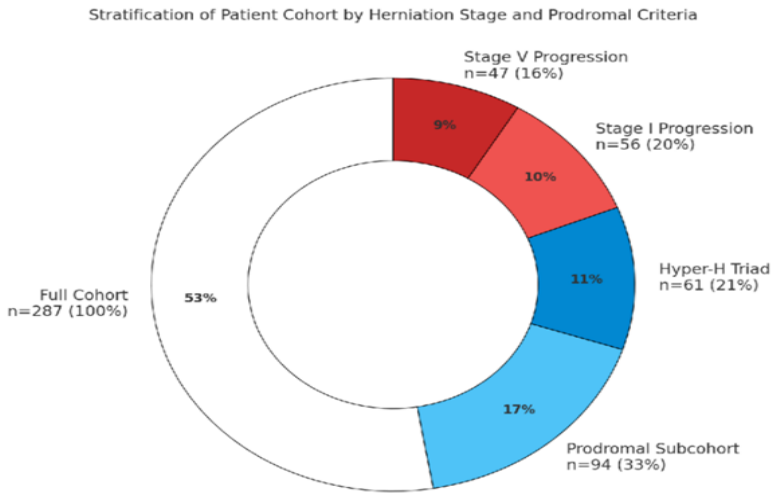


*Figure 1 - Stratification of patient cohort by herniation stage and prodromal criteria. Distribution of patients across five stages of TBH and identification of the prodromal Hyper-H triad. Values are expressed as percentages of the total cohort (N = 287)*

In Stage IV, 84.2% of patients underwent surgery, while in Stage V—marked by medullary destruction—only 50.0% received operative treatment, typically in an attempted salvage setting. Overall, 218 of 287 patients (76.0%) were managed surgically (Table 1).

*Table 1 - Distribution of Patients and Surgical Interventions by TBH Stage*

| TBH Stage | Number of Patients | Operated (n) | Operated (%) |
|---|---|---|---|
| I | 110 | 56 | 51.0% |
| II | 83 | 80 | 96.4% |
| III | 63 | 60 | 95.2% |
| IV | 19 | 16 | 84.2% |
| V | 12 | 6 | 50.0% |
| Total | 287 | 218 | 76.0% |

*Note: Operative rates peaked at Stages II–III, indicating maximal surgical responsiveness to radiological herniation signs. The decline in Stages I and V reflects, respectively, early diagnostic uncertainty and limited salvageability in terminal deterioration*

*Surgical Approaches*

Among the 218 operated patients, unilateral lateral craniotomy was the predominant surgical procedure, performed in 177 cases (81.2%). Other approaches included bilateral lateral craniotomy in 14 cases (6.4%), bifrontal craniotomy in 12 (5.5%), and extended lateral craniotomy in 8 (3.7%). Less frequent procedures were anterolateral craniotomy (n=3; 1.4%) and exploratory burr holes (n=4; 1.8%).

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This distribution highlights the overwhelming reliance on unilateral lateral decompression as the standard surgical response to TBH, with alternative approaches reserved for anatomically complex or refractory cases (Table 2).

*Table 2 - Surgical Approaches in Patients with TBH*

| Surgical Approach | Number of Patients (n) | Percentage (%) |
|---|---|---|
| Unilateral lateral craniotomy | 177 | 81.2% |
| Bilateral lateral craniotomy | 14 | 6.4% |
| Bifrontal craniotomy | 12 | 5.5% |
| Extended lateral craniotomy | 8 | 3.7% |
| Anterolateral craniotomy | 3 | 1.4% |
| Exploratory burr holes | 4 | 1.8% |
| **Total** | **218** | **100%** |

*Note: Unilateral lateral craniotomy was the dominant approach (81.2%), reflecting its practicality in emergency decompression. Less frequent methods, such as bifrontal or extended craniotomies, were reserved for complex or refractory cases)*

*Stage-Specific Brainstem Pathology*

Morphological analysis of brainstem injury across TBH stages revealed a staged and anatomically consistent progression.

- Stage I: Pathology was dominated by diencephalic hemorrhage and ischemia, with hemorrhages localized to the hypothalamus and thalamus and early ischemic foci.
- Stage II: Midbrain involvement became evident, with hemorrhages in the tectum and tegmentum, alongside ischemic lesions within the central gray.
- Stage III: Predominant injury shifted to the pons, characterized by extensive hemorrhage in the basilar pons and rupture of perforating vessels.
- Stage IV: Lower pontine and medullary regions demonstrated large necrotic areas with disruption of the reticular formation, correlating with advanced neurological deterioration.
- Stage V: Global medullary destruction was observed, including necrosis of cardiorespiratory centers, representing the terminal phase of herniation pathology (Table 3).

*Table 3 - Brainstem Morphological Changes by Stage of TBH*

| TBH Stage | Predominant Brainstem Pathology | Key Findings |
|---|---|---|
| I | Diencephalic hemorrhage and ischemia | Hemorrhages in hypothalamus/thalamus; early ischemic foci |
| II | Midbrain involvement | Hemorrhages in tectum and tegmentum; ischemic lesions in central gray |
| III | Pontine injury | Extensive hemorrhage in basilar pons; perforating vessel rupture |
| IV | Lower pontine and medullary injury | Large necrotic areas, reticular formation disruption |
| V | Medullary destruction | Global necrosis, cardiorespiratory centers destroyed |

*Note: Progressive caudal spread of hemorrhagic and ischemic injury marks advancing TBH severity*

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These findings confirm that TBH progression is not abrupt but rather follows a predictable anatomical sequence from diencephalic to medullary structures (Figure 2).

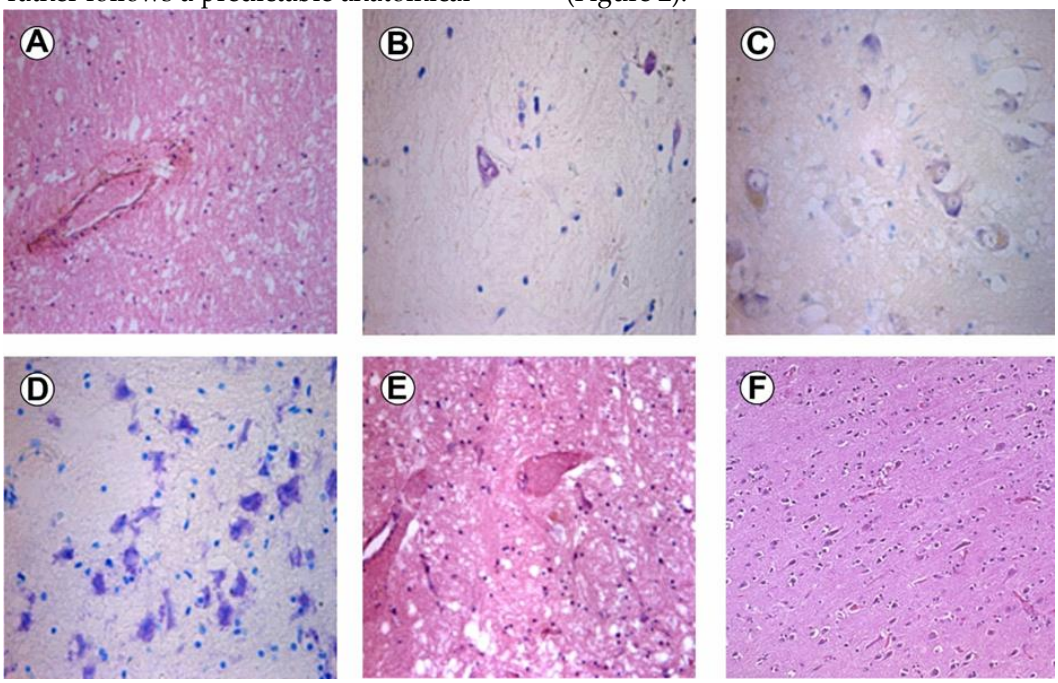


*Figure 2 - Representative histopathological features of brainstem structures across TBH stages I–V in severe traumatic brain injury, compared with normal brain tissue.*
*(A) TBH Stage I – Subcortical zone: Edematous white matter with a reticular (spongiform) appearance, vascular thrombosis in areas of perivascular hemorrhage, and reactive microglial migration. Hematoxylin and eosin staining, magnification ×200. (B) TBH Stage II – Thalamus: Neuronal nuclei with marked structural alterations, including karyolysis, granular cytoplasmic degradation, and cellular shrinkage. Nissl staining, magnification ×400. (C) TBH Stage III – Rostral pons: Reactively altered neurons are swollen and enlarged, with thickened processes and signs of chromatolysis. Cytoplasm contains abundant lipofuscin granules ("wear-and-tear" pigment), and areas of neuronal dropout with ghost cells are evident. Nissl staining, magnification ×400. (D) TBH Stage IV – Pons: Severe neuronal damage characterized by blurred and irregular cell contours, karyorrhexis, and granular disintegration of Nissl substance. Nissl staining, magnification ×400. (E) TBH Stage V – Medulla oblongata: Spongy degeneration of the tissue, irregularly thickened fibers, and microglial activation. Hematoxylin and eosin staining, magnification ×200. (F) Control – Normal brain tissue: Preserved neuronal morphology with intact cell contours, evenly distributed Nissl substance, and absence of pathological alterations. Hematoxylin and eosin staining, magnification ×200*

*Integrated Stage–Surgery–Outcome Dynamics*

The combined analysis of patient distribution, operative rates, and pathological findings underscores the critical importance of stage-specific surgical timing. While nearly all patients in Stage II and III underwent surgery, outcomes were contingent on the underlying degree of brainstem injury. Early stages (I–II) were associated with potentially reversible pathology and higher likelihood of functional recovery, whereas late stages (IV–V) were characterized by necrotic or destructive lesions, limiting the effectiveness of surgery despite high intervention rates.

Notably, the relatively lower operative rate in Stage I reflects current clinical hesitancy to intervene surgically in prodromal cases without overt herniation signs. However, the morphological data suggest that Stage I injury is largely diencephalic and potentially reversible. This highlights the therapeutic dilemma: operating earlier may offer the best chance to prevent progression, but requires reliable prodromal markers—such as the Hyper-H Triad—for timely identification.

## 4. Discussion

This prospective multicenter study provides stage-specific insights into the outcomes of surgical intervention in TBH. Our findings demonstrate that surgical effectiveness is strongly dependent on the

herniation stage at the time of intervention. Surgical intervention is most effective in Stages I–II, where brainstem pathology is limited and potentially reversible. By contrast, surgery in Stages IV–V is associated with irreversible injury, explaining poor outcomes despite high surgical rates.

The predominance of unilateral lateral craniotomy across all stages underscores its role as the standard operative technique, while alternative approaches remain rare. Together, these findings define the therapeutic window of opportunity in TBH and establish the morphological basis for stage-specific outcome differences. While operative rates were high across all but the terminal stage, outcomes were markedly divergent, underscoring the importance of defining a therapeutic window of opportunity.

Stage-specific outcomes and the therapeutic window. The analysis revealed that patients operated in Stages I and II achieved the most favorable outcomes. Morphological data indicate that pathology in these stages is largely confined to diencephalic and midbrain structures, where injury may still be reversible. In contrast, patients in Stages IV and V exhibited extensive pontomedullary necrosis and destruction of cardiorespiratory centers, limiting the efficacy of decompressive surgery despite frequent intervention. These findings align with earlier observations that late decompression rarely improves survival or functional recovery. Collectively, the results highlight that the window for meaningful surgical benefit lies within the prodromal and early herniation stages.

A central implication of our study is the potential role of the Hyper-H Triad – hyperthermia, hypertonia, and hormonal dysregulation – as a prodromal marker for impending herniation [16-18].

Our findings expand on prior decompressive craniectomy trials such as DECRA and RESCUEicp, which reported conflicting survival and functional outcomes. These trials did not stratify patients by herniation stage, potentially obscuring the therapeutic impact of early versus late intervention. By integrating stage-specific pathology with clinical outcomes, our data support the concept that heterogeneity in surgical timing contributes significantly to the variability observed in prior studies [19-22].

In Stage I, where operative hesitancy was most evident, pathology remained potentially reversible. However, without reliable clinical indicators, timely intervention was inconsistent. The Hyper-H Triad offers a reproducible clinical signal that may guide neurosurgeons toward earlier decompression, reducing reliance on late radiological or neurological signs. Validation of this triad across diverse cohorts could standardize early recognition and transform surgical timing into a proactive rather than reactive decision.

*Surgical approaches and practice implications*

Unilateral lateral craniotomy was the predominant intervention across all stages, reflecting its status as the standard operative technique. Alternative approaches – bilateral, bifrontal, or extended lateral craniotomies – were reserved for anatomically complex or refractory cases. While the choice of surgical approach was relatively uniform, outcomes were primarily dictated by timing and underlying brainstem pathology rather than technique. This suggests that refining surgical decision-making window may be more impactful than modifying operative approaches [23-26].

*Highlights:*

- TBH progresses through anatomically distinct, predictable stages.
- Surgical effectiveness is highest in Stages I–II, defining a narrow therapeutic window.
- Stage-specific pathology shows reversible diencephalic/midbrain injury early, necrotic pontomedullary lesions late.
- The Hyper-H Triad emerges as a reproducible prodrome signaling impending herniation.
- Protocol-based decision-making using stage and prodromal markers may improve survival and outcomes.

*Limitations*

Several methodological considerations should be acknowledged. The observational design limits causal inference, and unmeasured confounders cannot be fully excluded. External validation in broader clinical settings is warranted to support generalizability. Although long-term outcomes were not systematically captured, the present findings consistently demonstrate stage-dependent surgical responsiveness.

TBH should no longer be regarded as a catastrophic or random event, but rather as a staged and structured process with definable thresholds for intervention. The Hyper-H Triad represents prodromal clinical indicators that anticipate progressive displacement and delineate the optimal surgical window. By linking early autonomic and hemodynamic shifts to actionable decision points, this framework bridges the conceptual gap between classical Cushing's and Sympathetic Triads and modern, physiology-based timing of decompression.

*Future directions*

Future research should prioritize multicenter prospective validation of the Hyper-H Triad as a clinical trigger for surgical decision-making. Integration of this prodrome into standardized protocols may enable consistent early intervention, particularly in Stage I patients.

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Advanced monitoring techniques, such as continuous TCD and multimodal neurophysiology, could further refine the identification of the therapeutic window.

Ultimately, protocol-based adoption of stage-specific surgical strategies has the potential to reduce mortality and improve long-term recovery in TBH.

## 5. Conclusions

TBH should no longer be viewed as a catastrophic, unstructured event but as a staged process with identifiable thresholds for intervention. Our findings demonstrate that surgery is most effective in the prodromal and early stages, when pathology is potentially reversible. The Hyper-H Triad provides a promising clinical framework for recognizing impending herniation and guiding timely surgical action. By redefining the boundaries of the therapeutic window, these results lay the foundation for evidence-based, protocol-driven care in severe TBI.

**Authorship Confirmations.** B.M., A.M. - conceived the study and oversaw its overall direction and planning. B.M. - drafted the manuscript with input from all authors. A.B., K.N. - developed the computational framework and performed the initial data analysis. B.S. - created the visual representations and prepared all figures.

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**AI declaration.** No generative AI tools were used in data production or manuscript writing; only figure rendering utilized graphical libraries.

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# Бас миының жарақаттан кейінгі орнынан ығысуы кезіндегі ерте декомпрессивті хирургияның клиникалық триггері ретіндегі Hyper-H триадасы: Шешім қабылдауға ықпалы

Мустафаев Б.С. [1*], Мустафаева А.С. [2], Аскаров Б.К. [3], Никатов К.А. [4]

[1] Нейрохирург, нейрохирургия және нейропатология бөлімшесі; нейрохирургия және неврология кафедрасының оқытушысы, Ұлттық нейрохирургия орталығы, Астана, Қазақстан. E-mail: baglan.mustafaev@ncn.kz

[2] Нейрореабилитация бөлімінің меңгерушісі, Ұлттық нейрохирургия орталығы, Астана, Қазақстан. E-mail: alina.mustafaeva@ncn.kz

[3] Нейрохирургия бөлімінің меңгерушісі, Облыстық клиникалық аурухана; хирургия кафедрасының аға оқытушысы, Қ.А. Ясауи атындағы Халықаралық қазақ-түрік университеті, Шымкент, Қазақстан. E-mail: askar.bakhtiyaroz@ayu.edu.kz

[4] Нейрохирургия кафедрасының ассистенті, С.Ж. Асфендияров атындағы Қазақ ұлттық медициналық университеті, Алматы, Қазақстан. E-mail: nkabuevich@gmail.com

## Түйіндеме

Бас миының жарақаттан кейінгі орнынан ығысуы (дислокациясы) (БМЖО) ауыр бас-ми жарақатынан (БМЖ) кейінгі өлім-жітімнің басты себебі болып табылады. Хирургиялық араласу емделушінің өмірін сақтап қалуы мүмкін, бірақ емнің нәтижелері процестің сатысына байланысты. Терапевтік терезені анықтау және ерте клиникалық маркерлерді валидациялау аса маңызды клиникалық және болжамдық мәнге ие.

Зерттеудің мақсаты: ауыр БМЖ бар емделушілерде бас миы дислокациясының клиникалық және морфологиялық сатыларын зерттеу, хирургиялық араласу үшін оңтайлы терапевтік терезені анықтау және ми бағанасы қызметінің бұзылуының ерте клиникалық маркерлерінің диагностикалық құндылығын бағалау.

Әдістері. Біз Қазақстанда ауыр БМЖ (Глазгоның кома шкаласы бойынша ≤ 8) бар және дислокацияның рентгендік белгілері анықталған 287 ересек емделушіні қамтитын проспективті көпорталықты зерттеу жүргіздік. Емделушілер клиникалық-морфологиялық белгілері негізінде БМЖО-ның 5 сатысына жіктелді. Клиникалық, рентгенологиялық, хирургиялық және нейрофизиологиялық деректер үйлестірілген хаттама арқылы жиналды. Хирургиялық әдістер мен ми бағанасының патологиясына талдау жасалды.

Нәтижелері. 287 емделушінің 218-іне (76%) ота жасалды. Хирургиялық белсенділік деңгейі сатылар бойынша әртүрлі болды: 51% (I), 96,4% (II), 95,2% (III), 84,2% (IV) және 50% (V). Оталардың көпшілігі (81,2%) біржақты латеральді краниотомия әдісімен орындалды. Ми бағанасының зақымдануы заңды сипатта өрбіді: диэнцефальді (I), ортаңғы ми (II), көпір (III), сопақша ми (IV–V). Ерте сатылардағы хирургия патологияның қайтымдылығымен және жақсы нәтижелермен байланысты болды. Соңғы сатылардағы оталар қайтымсыз зақымдануларды көрсетті.

Қорытынды. БМЖО анатомиялық тұрғыда дәйекті түрде дамитын сатылы процесс болып табылады. Хирургиялық араласу I–II сатыларда барынша тиімді. Hyper-H триадасы — гипертермия, гипертонус, гормондық реттелудің бұзылыстары — ерте араласу қажеттілігі туралы белгі бере алады. Сатыларды ескере отырып хаттамаларды әзірлеу өлім-жітімді азайтудың негізгі факторы болып табылады.

**Түйін сөздер:** бас-ми жарақаты, бас миының орнынан ығысуы, декомпрессивті краниэктомия, ота жасау уақыты, Hyper-H триадасы, терапевтік терезе, ми бағанасының патологиясы.

# Триада Hyper-H как клинический триггер для ранней декомпрессивной хирургии при травматической дислокации головного мозга: Значение для принятия решений

Мустафаев Б.С. [1*], Мустафаева А.С. [2], Аскаров Б.К. [3], Никатов К.А. [4]

[1] Нейрохирург отделения нейрохирургии и невропатологии, преподаватель кафедры нейрохирургии и неврологии, Национальный центр нейрохирургии, Астана, Казахстан. E-mail: baglan.mustafaev@ncn.kz

[2] Заведующая отделением нейрореабилитации, Национальный центр нейрохирургии, Астана, Казахстан. E-mail: alina.mustafaeva@ncn.kz

[3] Заведующий отделением нейрохирургии, Областная клиническая больница; старший преподаватель кафедры хирургии, Международный казахско-турецкий университет имени Х.А. Ясауи, Шымкент, Казахстан. E-mail: askar.bakhtiyaroz@ayu.edu.kz

[4] Ассистент кафедры нейрохирургии, Казахский национальный медицинский университет имени С.Д. Асфендиярова, Алматы, Казахстан. E-mail: nkabuevich@gmail.com

## Резюме

Травматическое вклинение (дислокация) головного мозга (ТВГМ) является основной причиной смерти после тяжелой черепно-мозговой травмы (ЧМТ). Хирургическое вмешательство может спасти жизнь, но

исходы зависят от стадии процесса. Определение терапевтического окна и валидация ранних клинических маркеров имеют важнейшее клиническое и прогностическое значение.

Цель исследования: изучить клинические и морфологические стадии вклинения головного мозга у пациентов с тяжелой ЧМТ, определить оптимальное терапевтическое окно для хирургического вмешательства и оценить диагностическую ценность ранних клинических маркеров дисфункции ствола мозга.

Методы. Мы провели проспективное многоцентровое исследование в Казахстане, в которое вошли 287 взрослых пациентов с тяжелой ЧМТ (Шкала комы Глазго ≤ 8) и рентгенологическими признаками дислокации. Пациенты были стратифицированы на 5 стадий ТВГМ на основе клинико-морфологических признаков. Клинические, рентгенологические, хирургические и нейрофизиологические данные собирались с помощью гармонизированного протокола. Был проведен анализ хирургических методов и патологии ствола мозга.

Результаты. Из 287 пациентов 218 (76%) были прооперированы. Частота хирургических вмешательств варьировала в зависимости от стадии: 51% (I стадия), 96,4% (II), 95,2% (III), 84,2% (IV) и 50% (V). Большинство вмешательств (81,2%) представляли собой одностороннюю латеральную краниотомию. Повреждение ствола мозга следовало закономерной схеме: диэнцефальное (I), среднего мозга (II), моста (III), продолговатого мозга (IV–V). Хирургия на ранних стадиях была связана с обратимостью патологии и лучшими исходами, на поздних стадиях выявлялись необратимые повреждения.

Выводы. ТВГМ протекает стадийно, с анатомически последовательным прогрессированием. Хирургическое вмешательство наиболее эффективно на I–II стадиях. Триада Hyper-H — гипертермия, гипертонус, гормональная дисрегуляция — может служить сигналом к необходимости раннего вмешательства. Разработка протоколов с учетом стадий является ключевым фактором снижения смертности.

**Ключевые слова**: черепно-мозговая травма, вклинение головного мозга, декомпрессивная краниэктомия, сроки проведения операции, триада Hyper-H, терапевтическое окно, патология ствола мозга.