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COMPLICATIONS OF TRAUMATIC BRAIN INJURY

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Abstract.
Introduction. Traumatic brain injury (TBI) is a prevalent neurological condition associated with significant anatomical damage and long-term functional consequences. These injuries often lead to progressive complications, especially in the intermediate and late stages of recovery. Chronic neurological, psychiatric, and somatic impairments are frequently linked to prior TBI, reflecting the sustained impact of both destructive and reparative processes within the brain.
Objective. This study aimed to analyze the clinical spectrum and complication patterns in patients with severe TBI. Particular attention was given to morphological classifications (tissue-related, cerebrospinal fluid-related, vascular), their syndromic manifestations, and the influence of predisposing factors such as alcohol intoxication. The study also evaluated the correlation between Glasgow Coma Scale (GCS) scores and hospital-acquired complications and mortality.
Materials and Methods. A total of 82 patients with severe TBI were assessed, including 16 females (19.5%) and 66 males (80.5%) aged between 14 and 89 years. Patients were classified by GCS scores and monitored for complications including pneumonia, pulmonary embolism, neuroinfections, and kidney injury. Alcohol intoxication at the time of trauma was also recorded.
Results. Hospital-acquired complications occurred in a substantial portion of patients, with pneumonia (18.3%), pulmonary-cardiac insufficiency (32.9%), and neuroinfections (7.3%) being most common. Complication rates increased with decreasing GCS scores, and all patients who developed complications ultimately died (100% fatality in complicated cases). Alcohol intoxication was present in 47.6% of patients. Overall mortality was 32.9%.
Conclusion. Severe TBI is associated with high rates of complications and mortality, particularly in patients with low GCS scores or those affected by alcohol. Identifying the dominant morphological and syndromic patterns aids in tailored treatment. Preventing hospital-acquired complications and addressing modifiable risk factors such as alcohol use are critical to improving outcomes.
Keywords. Traumatic brain injury, Glasgow Coma Scale, hospital complications, alcohol intoxication, neurotrauma classification, mortality, post-traumatic syndromes.

Аннотация.
Введение. Черепно-мозговая травма (ЧМТ) является широко распространённой патологией, сопровождающейся значительными анатомическими повреждениями и отдалёнными функциональными последствиями. Нарушения чаще проявляются в промежуточный и поздний периоды травматической болезни и накапливаются с течением времени, влияя на соматическое, психическое и неврологическое здоровье пациентов.
Цель. Целью данного исследования являлся анализ клинического спектра и частоты осложнений у пациентов с тяжёлой ЧМТ. Особое внимание уделялось морфологической и синдромальной классификации последствий (тканевые, ликвородинамические и сосудистые

формы), взаимосвязи между уровнем сознания по шкале комы Глазго (ШКГ), развитием госпитальных осложнений, наличием алкогольной интоксикации, а также показателям летальности.

Материалы и методы. В исследование были включены 82 пациента с тяжёлой ЧМТ: 16 женщин (19,5%) и 66 мужчин (80,5%) в возрасте от 14 до 89 лет. Проводилась оценка по шкале ШКГ, а также регистрация осложнений: пневмония, тромбоэмболия лёгочной артерии (ТЭЛА), нейроинфекции, острая почечная недостаточность и другие. Отдельно анализировалось влияние алкогольной интоксикации.

Результаты. Госпитальные осложнения были выявлены у значительного числа пациентов: пневмония — 18,3%, лёгочно-сердечная недостаточность — 32,9%, нейроинфекции — 7,3%. Частота осложнений увеличивалась при снижении баллов ШКГ. У всех пациентов с осложнениями был зафиксирован летальный исход (100%). Алкогольное опьянение присутствовало у 47,6% пациентов. Общая летальность составила 32,9%.

Вывод. Тяжёлая ЧМТ сопровождается высоким уровнем осложнений и смертности, особенно у пациентов с низкими баллами ШКГ и алкогольной интоксикацией. Выявление ведущего морфологического компонента и предотвращение госпитальных осложнений являются ключевыми для улучшения исходов. Профилактика травм, связанных с алкоголем, также требует внимания на уровне общественного здравоохранения.

Ключевые слова. Черепно-мозговая травма, шкала комы Глазго, госпитальные осложнения, алкогольная интоксикация, нейротравма, летальность, посттравматические синдромы.

Kirish. Kranioserebral travma (KST) keng tarqalgan nevrologik kasallik bo‘lib, u anatomik shikastlanishlar va uzoq muddatli oqibatlar bilan kechadi. Ushbu holat odatda o‘rta va kech bosqichlarda asoratlar bilan namoyon bo‘ladi. BMSga chalingan bemorlarda yillar davomida nevrologik, psixiatrik va somatik buzilishlar rivojlanib, ularning sog‘lig‘iga, ijtimoiy faolligiga va hayot sifatiga salbiy ta’sir ko‘rsatadi.

Maqsad. Ushbu tadqiqotning asosiy maqsadi og‘ir KST ga chalingan bemorlarda klinik ko‘rinishlar va asoratlar namoyon bo‘lishini o‘rganishdan iborat bo‘ldi. Ayniqsa, KST oqibatlarining morfologik va sindromal tasnifi (to‘qimalar, bosh miya suyuqligi va qon tomir tizimi bilan bog‘liq shakllar), alkogol ichimliklar iste’molining rolini baholash, GKSH (Glazgoning koma shkalasi) ko‘rsatkichlari bilan asoratlar va o‘lim ko‘rsatkichlari o‘rtasidagi bog‘liqlik o‘rganildi.

Materiallar va usullar. Tadqiqotda 82 nafar bemor ishtirok etdi: 16 nafar ayol (19,5%) va 66 nafar erkak (80,5%), yoshi 14 dan 89 yoshgacha. Har bir bemorning holati GKSH bo‘yicha baholandi. Bemorlar orasida pnevmoniya, o‘pka yurak yetishmovchiligi, tromboemboliya, neyroinfeksiyalar, buyrak shikastlanishi kabi kasalxonada yuzaga keladigan asoratlar kuzatildi. 47,6% bemorlar bosh miya shikastini alkogol ichimligi iste’moli fonida olgani aniqlandi.

Natijalar. Eng ko‘p uchraydigan kasalxona asoratlari quyidagilar bo‘ldi: pnevmoniya – 18,3%, yurak-o‘pka yetishmovchiligi – 32,9%, neyroinfeksiyalar – 7,3%. Asoratlar darajasi GKSH balining pasayishi bilan oshdi. Asoratlar rivojlangan barcha bemorlarda o‘lim qayd etildi (100%). Umumiy o‘lim ko‘rsatkichi 32,9%ni tashkil etdi.

Xulosa. Og‘ir KST yuqori darajadagi asoratlar va o‘lim xavfi bilan tavsiflanadi. GKSH past bo‘lgan va alkogol fonida shikastlangan bemorlarda bu xavf yanada ortadi. Asoratlarning ustuvor morfologik shaklini aniqlash va ularning oldini olish muhim ahamiyatga ega. Alkogol bilan bog‘liq travmalarni kamaytirish uchun profilaktik chora-tadbirlar zarur.

Kalit so‘zlar. Bosh miya shikastlanishi, Glazgo koma shkalasi, kasalxona asoratlari, alkogol ichimligi, nevrotravma, o‘lim darajasi, posttravmatik sindromlar.

Introduction. Traumatic brain injury is a mass pathology characterized by a high frequency of various consequences. Along with anatomical damage (skull defects, crush lesions, intracranial hematomas), neurotrauma triggers, among others, two oppositely directed processes: dystrophic-destructive and regenerative-reparative, which for months and years go in parallel with a constant or variable predominance of one of them, ultimately determining the presence or absence of certain consequences of brain damage.

Post-traumatic brain and skull disorders may present during the acute phase but are more commonly observed in the intermediate and late stages of traumatic disease. Complications arising from traumatic brain injury (TBI) are widespread and tend to accumulate progressively over time. These complications act like a persistent residue that builds up over the years, significantly impacting public health and underscoring the humanitarian, social, and economic relevance of the issue. A substantial proportion of patients with chronic neurological, psychiatric, or somatic conditions have a history of trauma and continue to experience the long-term consequences of TBI.

In addition to anatomical injuries such as skull defects, crush lesions, and intracranial hematomas, neurotrauma initiates two opposing processes: degenerative-destructive and regenerative-reparative. These processes unfold over months and years, often running in parallel, with one prevailing over the other at different times. The balance between them ultimately determines the development or absence of specific long-term effects of brain injury.

Clinical classification of TBI consequences. It is well established that the intracranial space consists of brain tissue (approximately 85%), cerebrospinal fluid (about 10%), and blood (around 5%). All of these, along with the hard and soft tissues of the skull, are susceptible to mechanical trauma. Based on this, the morphological consequences of traumatic brain injury (TBI) can be grouped into three main categories:

1. **Tissue-related changes:**
    - *Brain-related*: such as atrophy, scarring, adhesions, etc.
    - *Skull-related*: including bone defects, osteolysis, osteosclerosis, and others.
2. **Cerebrospinal fluid (CSF) dynamics disturbances**: including impaired circulation or resorption, CSF leakage (rhinorrhea), CSF accumulation (oma), and related conditions.
3. **Vascular abnormalities**: such as blood flow disturbances, ischemic changes, and thrombosis.

These morphological categories correspond to three clinical groups of TBI consequences:

1. Predominantly tissue-related;
2. Predominantly CSF-related;
3. Predominantly vascular-related [2].

We have classified the clinical manifestations of tissue-related TBI consequences as follows:

- **Post-traumatic brain atrophy**: either localized or diffuse;
- **Post-traumatic arachnoiditis**;
- **Post-traumatic pachymeningitis**;
- **Meningeal and cerebral scarring**: with or without foreign bodies;
- **Cranial nerve injuries**;
- **Bone defects in the skull**;
- **Post-traumatic skull deformities**;
- **Combined forms**.

Clinical manifestations related to CSF dynamic disturbances include:

- **Hydrocephalus**: active or passive;
- **Porencephaly**;
- **Meningoencephalocele**;
- **Chronic hygromas**;
- **CSF cysts**;
- **CSF rhinorrhea**: with or without pneumocephalus;
- **Combined forms**.

Clinical vascular consequences of TBI include:

- **Ischemic brain injuries**;
- **Chronic hematomas**;
- **Aneurysms**: true and false types;
- **Arteriosinus anastomoses**: such as carotid-cavernous and others;
- **Sinus thrombosis**;
- **Combined forms**.

Although these consequences often overlap in clinical practice, identifying the dominant component is essential for guiding therapeutic decisions and ensuring appropriate social support for patients.
Moreover, all TBI-related conditions should be further classified into **traumatic** and **iatrogenic** origins. This distinction is important in practice—for example, the majority of skull bone defects result from medical intervention.
Each clinical form of TBI consequences has its own symptomatology and progression pattern. However, several common post-traumatic syndromes can be observed across all forms:

1. Neurological deficits;
2. Cognitive and mental dysfunction;
3. Autonomic nervous system disturbances;
4. Epileptic syndromes.

Through detailed analysis of pathogenesis and recovery mechanisms, combined with neuroimaging techniques, we have developed and implemented conceptual strategies for treating the most surgically relevant consequences of TBI [3].
Objective. The primary aim of this study was to analyze the clinical manifestations and complication patterns of severe traumatic brain injury (TBI), with particular focus on the morphological and syndromic classification of TBI consequences. Additionally, the study aimed to assess the incidence and types of hospital-acquired complications, their correlation with the level of consciousness on the Glasgow Coma Scale (GCS), the influence of predisposing factors such as alcohol intoxication, and their impact on clinical outcomes, including mortality. A secondary objective was to reinforce the importance of identifying the predominant morphological component (tissue, CSF-related, or vascular) for optimizing treatment strategies and long-term patient management.
Materials and methods. The study included 82 patients, including 16 women (19.5%) and 66 men (80.5%). The age of the patients ranged from 14 to 89 years. 32 patients were of non-working age (39.0%), and 50 patients were of working age (61.0%).
Depending on the level of consciousness according to the Glasgow Coma Scale (GCS), the risk of complications was distributed as follows:
with a level of consciousness of 8-10 points - in 18 patients (21.9%), the risk of complications was observed in 22.2% of cases, i.e. in 4 people;
- with 6-7 points - in 15 patients (18.3%), complications developed in 3 patients (20.0%);

- with 3–5 points — in 12 patients (14.6%), complications were recorded in 6 patients, which is 50.0% in this subgroup.

In patients with severe traumatic brain injury (TBI), the frequency of hospital complications was as follows:

- hospital pneumonia — in 15 patients (18.3%);
- pulmonary heart failure — in 27 patients (32.9%);
- pulmonary embolism (PE) — in 6 patients (7.3%);
- post-traumatic neuroinfections — in 6 patients (7.3%);
- acute kidney injury — in 6 patients (7.3%);
- DIC syndrome (hemostasis system disorders) — in 3 patients (3.7%);
- acute pulmonary edema and shock of various etiologies were not diagnosed (0%).

It is worth noting that 39 patients (47.6%) developed severe TBI against the background of alcohol intoxication, which allows us to classify it as a significant predisposing factor. Mortality among patients with severe TBI was 32.9% (27 people). However, when complications developed, the fatal outcome was recorded in 100% of cases.

Result. The study included 82 patients with severe TBI, of whom 16 were female (19.5%) and 66 were male (80.5%). The age of the cohort ranged from 14 to 89 years. A total of 32 patients (39.0%) were of non-working age, while 50 patients (61.0%) were within the working-age group.

Assessment of consciousness levels using the Glasgow Coma Scale (GCS) demonstrated that:

- Among patients with GCS scores of 8–10 (n=18), complications were observed in 4 cases (22.2%);
- In patients with GCS scores of 6–7 (n=15), complications developed in 3 cases (20.0%);
- In the subgroup with the most severe impairment (GCS 3–5, n=12), complications were identified in 6 patients (50.0%).

Hospital-acquired complications in patients with severe TBI were widespread and included:

- **Hospital-acquired pneumonia** — in 15 patients (18.3%);
- **Pulmonary-cardiac insufficiency** — in 27 patients (32.9%);
- **Pulmonary embolism (PE)** — in 6 patients (7.3%);
- **Post-traumatic neuroinfections** — in 6 patients (7.3%);
- **Acute kidney injury (AKI)** — in 6 patients (7.3%);
- **Disseminated intravascular coagulation (DIC)** — in 3 patients (3.7%);
- **Acute pulmonary edema and shock of various etiologies** were not observed.

A noteworthy finding was that **39 patients (47.6%)** had sustained severe TBI while under the influence of alcohol, indicating **alcohol intoxication as a major predisposing factor** for both injury and complicated clinical course.

The **overall mortality rate** among patients with severe TBI was **32.9% (27 patients)**. Importantly, when hospital complications occurred, the **fatality rate reached 100%**, underlining the prognostic significance of secondary complications in the post-traumatic period.

Conclusion. This study highlights the multifaceted and progressive nature of traumatic brain injury and its consequences. The clinical outcomes of patients with severe TBI are significantly influenced not only by the initial anatomical damage but also by the interplay of degenerative and reparative processes, the level of consciousness at presentation, and the development of hospital-acquired complications.

The classification of post-traumatic changes into tissue-related, cerebrospinal fluid-dynamic, and vascular categories provides a valuable framework for diagnosis and individualized treatment planning. Common post-traumatic syndromes—neurological deficits, cognitive dysfunction,

autonomic disturbances, and epileptic episodes—were seen across morphological types, reinforcing the need for a comprehensive and multidisciplinary approach to care.

Alcohol intoxication was found to be a critical predisposing factor, present in nearly half of the cases, suggesting the need for preventive strategies and public health interventions targeting alcohol-related trauma.

The mortality data underscore the urgency of preventing and aggressively managing hospital complications, as their presence dramatically increases the risk of death. These findings emphasize the importance of early risk stratification, timely neuroimaging, and targeted interventions, especially in patients with the lowest GCS scores and those presenting with systemic vulnerability. Continued research into the mechanisms of neurotrauma and its long-term effects will be essential for improving outcomes, reducing disability, and guiding effective rehabilitation and social reintegration strategies for survivors of TBI.

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