



CLINICAL, DIAGNOSTIC, AND SURGICAL ASPECTS OF BILATERAL MULTIPLE TRAUMATIC INTRACRANIAL HEMATOMAS

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Abstract.

Background. Bilateral multiple traumatic intracranial hematomas represent one of the most severe forms of traumatic brain injury, associated with high mortality, significant neurological deficits, and considerable challenges in surgical decision-making.

Objective. To identify clinical features, neuroimaging characteristics, and surgical management strategies for bilateral multiple traumatic intracranial hematomas.

Materials and methods. A total of 117 patients with multiple traumatic intracranial hematomas were analyzed, including 49 cases with bilateral hemispheric involvement. All patients underwent detailed neurological examination, skull radiography, and brain computed tomography.

Results. Bilateral hematomas were most commonly associated with a coup–contrecoup mechanism of injury. Focal neurological deficits predominated contralateral to the larger hematoma. Anisocoria was usually mild, and midline shift was uncommon. Surgical tactics were primarily guided by hematoma volume and CT signs of brain compression.

Conclusion. Computed tomography plays a decisive role in diagnosis, prognosis, and selection of surgical strategy in patients with bilateral multiple traumatic intracranial hematomas.

Keywords: traumatic brain injury, bilateral hematomas, intracranial hemorrhage, computed tomography, neurosurgery.

КЛИНИКО-ДИАГНОСТИЧЕСКИЕ И ХИРУРГИЧЕСКИЕ АСПЕКТЫ ДВУПОЛУШАРНЫХ МНОЖЕСТВЕННЫХ ТРАВМАТИЧЕСКИХ ВНУТРИЧЕРЕПНЫХ ГЕМАТОМ

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Аннотация.

Актуальность. Двуполушарные множественные травматические внутричерепные гематомы относятся к наиболее тяжелым формам черепно-мозговой травмы и характеризуются высокой летальностью, выраженным полиморфизмом клинических проявлений и сложностью выбора хирургической тактики.

Цель исследования. Выявить особенности клинического течения, нейровизуализационных признаков и хирургического лечения двуполушарных множественных травматических внутричерепных гематом.

Материалы и методы. Проанализированы результаты обследования и лечения 117 пациентов с множественными травматическими внутричерепными гематомами, из которых у 49 диагностированы двуполушарные поражения. Всем больным выполнены клинико-неврологическое обследование, рентгенография черепа и компьютерная томография головного мозга.

Результаты. Установлено, что двуполушарные гематомы чаще формируются по механизму «удар–противоудар» и сопровождаются преобладанием очаговой симптоматики, контралатеральной по отношению к гематоме большего объема. Анизокория выражена

слабо, смещение срединных структур наблюдается редко. Тактика хирургического лечения определялась объемом гематом и данными компьютерной томографии.

Заклучение. Компьютерная томография является ключевым методом диагностики и выбора оптимальной хирургической тактики при двуполушарных множественных травматических гематомах.

Ключевые слова: черепно-мозговая травма, двуполушарные гематомы, внутричерепные гематомы, компьютерная томография, нейрохирургическое лечение.

IKKI YARIMSHARLI KO'P SONLI TRAVMATIK INTRAKRANIAL GEMATOMALARNING KLINIK-DIAGNOSTIK VA JARROHLIK JIHATLARI

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SamDTU huzuridagi neyroxirurgiya va neyroeabilitatsiya ixtisoslashtirilgan ilmiy-amaliy markazi

Annotatsiya.

Dolzarbli. Ikki yarimsharli ko'p sonli travmatik intrakranial gematomalar bosh miya jarohatlarining eng og'ir shakllaridan biri bo'lib, yuqori o'lim ko'rsatkichi, klinik belgilar polimorfizmi va jarrohlik taktikasini tanlashdagi murakkablik bilan tavsiflanadi.

Tadqiqot maqsadi. Ikki yarimsharli ko'p sonli travmatik intrakranial gematomalarning klinik kechishi, neyroximoya (KT) belgilari va jarrohlik davolash xususiyatlarini aniqlash.

Material va usullar. Ko'p sonli intrakranial gematomalari bo'lgan 117 nafar bemorning klinik va instrumental tekshiruv natijalari tahlil qilindi, shundan 49 nafarida ikki yarimsharli shikastlanish aniqlangan.

Natijalar. Ikki yarimsharli gematomalar ko'pincha «zarba–qarshi zarba» mexanizmi asosida shakllanadi. Klinik jihatdan katta hajmli gematomaga qarama-qarshi tomonda o'choqli simptomatika ustunlik qiladi. O'rta tuzilmalar siljishi kam kuzatiladi.

Xulosa. Kompyuter tomografiya ikki yarimsharli ko'p sonli travmatik intrakranial gematomalarda diagnostika va jarrohlik taktikasini tanlashda asosiy usul hisoblanadi.

Kalit so'zlar: bosh miya jarohati, ikki yarimsharli gematomalar, intrakranial qon quyilishi, kompyuter tomografiya, neyroxirurgiya.

Introduction. In recent years, traumatic brain injury (TBI) has increasingly become not only a medical but also a major socio-economic and national public health problem. Over the past decades, the rapid development of high-speed transport, expansion of road networks, and progressive urbanization have been associated with a steady increase in both the incidence and severity of brain injuries [1,4]. According to data from international researchers, the financial burden related to acute treatment and long-term rehabilitation of patients with both mild and severe TBI is extremely high and continues to grow annually, placing substantial strain on healthcare systems worldwide [5]. Injuries to the central nervous system currently account for approximately 30–40% of the overall structure of traumatic injuries, underscoring their epidemiological and clinical significance [4]. Multiple traumatic intracranial hematomas represent a relatively uncommon but particularly severe form of TBI. They account for up to 0.74% of all traumatic brain injuries and occupy the second position among all intracranial hematomas in terms of clinical severity and prognostic relevance [2]. According to other authors, multiple hematomas constitute approximately 8–10% of all space-occupying intracranial hemorrhages [1]. Despite their relatively low incidence, multiple hematomas are associated with high rates of neurological deterioration, surgical complexity, and mortality, which necessitates detailed analysis of their clinical course and treatment strategies. The aim of the present study was to identify specific features of the clinical course and surgical management of bilateral hemispheric multiple traumatic intracranial hematomas.

Material and methods. The study included 117 patients with multiple traumatic intracranial hematomas aged from 14 to 82 years. Among them, 17 were women and 100 were men, reflecting the known predominance of males in severe head trauma epidemiology. Bilateral multiple

hematomas were diagnosed in 49 patients, whereas 68 patients had unilateral multiple hematomas. All patients underwent comprehensive clinical and neurological examination, neurophysiological assessments, skull radiography, and computed tomography (CT) of the brain as the primary neuroimaging modality for diagnosis and treatment planning.

Results and discussion. Patients with bilateral hemispheric hematomas were subdivided into three groups according to the morphological type and localization of hemorrhages: 27 patients with bilateral extra-axial (meningeal) hematomas, 9 patients with bilateral intracerebral hematomas, and 13 patients with combined extra-axial and intracerebral hematomas located in opposite hemispheres. Bilateral hematomas most frequently developed according to a coup–contrecoup mechanism of injury, which is characteristic of high-energy blunt head trauma. In this context, extra-axial hematomas were more commonly observed on the side of the direct impact, whereas intracerebral hematomas predominantly formed in the contralateral hemisphere, consistent with inertial displacement and rotational acceleration mechanisms described in experimental and clinical studies [6,7]. The distribution of patients according to hematoma combinations demonstrated that bilateral subdural hematomas were the most frequent variant, followed by combinations of subdural and intracerebral hematomas. Less commonly observed were bilateral epidural hematomas and mixed epidural–intracerebral patterns. This heterogeneity highlights the complex biomechanical nature of bilateral brain injury and the variability of pathological substrates involved.

The clinical course of bilateral hemispheric hematomas was characterized by progressive general cerebral and focal neurological symptoms. Headache was reported in 21 patients (43%) who were admitted in a state of obtundation. In the remaining patients, who were in stupor or coma, headache could not be reliably assessed. Recurrent vomiting was observed in 24 patients (49%), reflecting increased intracranial pressure and brainstem involvement. Meningeal signs were present in 28 patients (57%), indicating irritation of the meninges due to blood dissemination in the subarachnoid space. Focal neurological deficits were typically more pronounced contralateral to the hemisphere containing the hematoma of greater volume, often combined with extrapyramidal and subcortical–cerebellar motor disturbances. Oculomotor disorders were identified in 18 patients (37%) with bilateral hematomas. Narrowing of the palpebral fissure and ptosis were noted in 2 patients (4%) on the side of the larger hematoma. Divergent strabismus was detected in 9 patients (18.4%), while partial oculomotor nerve paresis on the side of the larger hematoma was observed in 7 patients (14.3%). In cases of equal hematoma volume, oculomotor deficits more frequently corresponded to the side of the epidural hematoma. Mild anisocoria was diagnosed in 7 patients (14.3%), whereas pronounced mydriasis was consistently observed on the side of the larger hematoma, which corresponds with published neurosurgical observations [3,8]. In patients with bilateral hematomas, exophthalmos more often appeared on the side of the larger lesion and regressed following surgical decompression, suggesting a venous congestion mechanism secondary to increased intracranial pressure. Bilateral subdural hematomas rarely produced marked oculomotor disturbances. As reported by A.N. Konovalov et al., anisocoria is uncommon in bilateral hematomas and may occur only after surgical removal of one of the lesions, reflecting altered intracranial dynamics [3].

Motor deficits were identified in 18 patients (36%). Pyramidal signs were predominantly unilateral, while bilateral pyramidal symptoms—more pronounced contralateral to the larger hematoma—were observed in 5 patients (10.2%). The severity of motor impairment was largely determined by the localization and volume of hematomas, as well as the degree of underlying brain contusion and secondary ischemic damage. The morphological type of hematoma also played a critical role. Even relatively small intracerebral hematomas in the setting of severe brain contusion resulted in more pronounced motor deficits compared with larger extra-axial hematomas on the opposite side. In cases of equal hematoma volume, motor dysfunction was primarily determined by the presence of an epidural hematoma, reflecting its rapid mass-effect and arterial origin [9,10]. In several patients with bilateral subdural hematomas, the clinical picture resembled a subcortical–cerebellar syndrome characterized by generalized rigidity, bradykinesia, plastic hypertonia, and tremor. Primary brainstem symptoms were observed in 4 patients (8%), while secondary brainstem signs dominated the clinical presentation in 7 patients (14%) at later stages of disease progression,

often masking focal hemispheric symptoms and indicating advanced intracranial hypertension and transtentorial herniation. Skull radiography revealed linear fractures in 15 patients (30.6%) and depressed skull fractures in 4 patients (8.2%). Echoencephalography showed no midline shift in most cases; however, in 5 patients (10.2%), displacement of the M-echo by 3–4 mm was detected. Computed tomography was performed in 42 patients (85.7%) and allowed reliable preoperative identification of bilateral hematomas. In 7 patients (14.3%), the second hematoma was detected intraoperatively, emphasizing the diagnostic value of modern neuroimaging and the limitations of older techniques.

Management of bilateral hematomas was based on the level of consciousness, volume of each hematoma, total volume of pathological intracranial mass, and the presence of CT and neurological signs of brain displacement. The introduction of CT fundamentally changed the surgical approach to bilateral hematomas by enabling visualization of brainstem lesions and secondary ischemic changes. CT signs of brainstem injury were identified in 11 patients (22.4%), all of whom had a fatal outcome, highlighting the prognostic importance of stem involvement [11,12]. In bilateral hematomas, surgical removal was indicated for those lesions responsible for significant brain compression as determined by CT. In patients with bilateral extra-axial hematomas, 6 patients (12.3%) had laminar hematomas with a volume less than 30 ml on one side; in these cases, resection craniotomy was performed only on the side of the larger hematoma. Bilateral craniotomy with evacuation of both hematomas was carried out in 21 patients (42.8%). Among 9 patients (18.4%) with bilateral intracerebral hematomas, 3 patients (6.1%) were managed conservatively due to small hematoma volume, 4 patients (8.1%) underwent bilateral hematoma evacuation, and 2 patients (4.1%) underwent unilateral surgery. In combined extra-axial and intracerebral hematomas, the same principle was applied: surgical removal targeted the lesion causing the dominant mass effect and brain compression as identified by CT imaging.

Conclusion. Bilateral hemispheric traumatic hematomas are characterized by marked clinical polymorphism and complex pathophysiological mechanisms. In most cases, unilateral neurological deficits predominate despite bilateral structural lesions. Focal symptoms are typically more pronounced contralateral to the hemisphere containing the larger hematoma. Unlike other types of intracranial hematomas, anisocoria in bilateral hematomas is usually mild and poorly expressed. Midline shift is generally absent due to the compensatory bilateral mass effect. Therapeutic strategy and surgical tactics should be determined primarily by computed tomography findings, which remain the cornerstone of diagnosis, prognostication, and treatment planning in patients with bilateral traumatic intracranial hematomas.

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