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TRAUMATIC INJURIES OF THE POSTERIOR CRANIAL FOSSA: EPIDEMIOLOGY, PATHOGENESIS, CLINICAL PATTERNS, AND DIAGNOSTIC CHALLENGES

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Abstract.
Background: Traumatic injuries of the posterior cranial fossa (PCF) are uncommon but clinically critical due to the limited volume of the infratentorial compartment, early obstruction of cerebrospinal fluid pathways, and rapid brainstem compromise.
Objective: To summarize current concepts on the spectrum of traumatic PCF lesions, mechanisms of formation, typical clinical patterns, and factors complicating timely diagnosis.
Methods: Narrative synthesis of published data describing traumatic epidural and subdural hematomas of the PCF, intracerebellar and intrastem hematomas, cerebellar/brainstem contusions, and fourth-ventricle hemorrhage, with emphasis on epidemiology, injury biomechanics (impact/counter-impact), and clinical course variants (acute/subacute).
Results: PCF injuries often present with a polymorphic combination of general cerebral, meningeal, cerebellar, and brainstem symptoms. Even small-volume hematomas may rapidly cause occlusive hydrocephalus and tonsillar herniation. Classic “lucid interval” patterns are more typical for posterior fossa epidural hematomas, whereas PCF subdural hematomas and intracerebellar hematomas more frequently coexist with supratentorial lesions and primary brainstem injury, masking focal infratentorial signs. The high rate of combined supra- and subtentorial trauma further increases diagnostic uncertainty and influences outcomes.
Conclusion: Traumatic PCF lesions remain a diagnostically challenging and high-risk category of TBI. Early recognition of course variants, red-flag brainstem/occlusive features, and awareness of typical injury mechanisms are essential to improve triage and management decisions.
Keywords: posterior cranial fossa; traumatic brain injury; posterior fossa epidural hematoma; posterior fossa subdural hematoma; intracerebellar hematoma; brainstem compression; occlusive hydrocephalus; tonsillar herniation.

ТРАВМАТИЧЕСКИЕ ПОВРЕЖДЕНИЯ СТРУКТУР ЗАДНЕЙ ЧЕРЕПНОЙ ЯМКИ: ЭПИДЕМИОЛОГИЯ, ПАТОГЕНЕЗ, КЛИНИЧЕСКИЕ ВАРИАНТЫ И ДИАГНОСТИЧЕСКИЕ ТРУДНОСТИ

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Аннотация.
Актуальность: Травматические повреждения структур задней черепной ямки (ЗЧЯ) встречаются редко, но относятся к наиболее угрожающим вариантам ЧМТ вследствие малого объёма субтенториального пространства, раннего нарушения ликвороциркуляции и быстрого формирования компрессии ствола мозга.
Цель: Обобщить современные представления о спектре травматических поражений ЗЧЯ, механизмах их формирования, типичных клинических вариантах течения и причинах

диагностических трудностей.
Материалы и методы: Нарративный обзор данных литературы по травматическим эпидуральным и субдуральным гематомам ЗЧЯ, внутримозжечковым и внутристоволовым гематомам, ушибам мозжечка/ствола, кровоизлияниям в IV желудочек с акцентом на эпидемиологию, биомеханику травмы (удар/противоудар) и варианты клинического течения (острое/подострое).
Результаты: Клиническая картина повреждений ЗЧЯ полиморфна и включает общемозговые, менингеальные, мозжечковые и стволовые симптомы. Даже небольшие гематомы могут приводить к ранней окклюзионной гидроцефалии и тонзиллярной дислокации. «Классический» вариант с ясным промежутком чаще характерен для эпидуральных гематом ЗЧЯ, тогда как субдуральные и внутримозжечковые гематомы нередко сочетаются с супратенториальными повреждениями и первичным ушибом ствола, что маскирует локальную симптоматику. Высокая частота комбинированной супра- и субтенториальной травмы усиливает диагностическую неопределённость и влияет на исходы.
Заключение: Травматические поражения ЗЧЯ сохраняют статус сложной для клинической диагностики и прогностически неблагоприятной категории ЧМТ. Для улучшения результатов критически важны раннее распознавание вариантов течения, выявление стволовых/окклюзионных признаков и учёт типичных механизмов травмы.
Ключевые слова (Русский): задняя черепная ямка; черепно-мозговая травма; эпидуральная гематома ЗЧЯ; субдуральная гематома ЗЧЯ; внутримозжечковая гематома; компрессия ствола; окклюзионная гидроцефалия; тонзиллярная дислокация.

ORQA KALLA CHUQURCHASI TUZILMALARINING TRAVMATIK SHIKASTLANISHLARI: EPIDEMIOLOGIYA, PATOGENEZ, KLINIK KECHISH VARIANTLARI VA DIAGNOSTIK QIYINCHILIKLAR

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Dolzarblik: Orqa kalla chuqurchasi (OKCh) tuzilmalarining travmatik shikastlanishlari kam uchraydi, biroq infratentorial bo‘shliq hajmining kichikligi, likvor aylanishi yo‘llarining erta bloklanishi va miya sopining tez siqilishi sababli eng og‘ir TBI variantlariga kiradi.
Maqsad: OKCh travmalarining asosiy nosologik ko‘rinishlari, kelib chiqish mexanizmlari, klinik kechish variantlari hamda o‘z vaqtida tashxis qo‘yishni qiyinlashtiruvchi omillarni umumlashtirish.
Material va usullar: Travmatik OKCh epidural/subdural gematomalari, intratserebellar va intrastem gematomalar, miyacha/miya sopi kontuziyalari hamda IV qorincha qon quyilishi bo‘yicha nashr etilgan ma’lumotlarning narrатив tahlili; urilish/qarshi urilish biomekhanikasi va o‘tkir/ostkiri (subakut) kechish variantlariga urg‘u berildi.
Natijalar: OKCh shikastlanishlarida umumiy miya, meningeal, miyacha va sop belgilarining polimorf kombinatsiyasi kuzatiladi. Hatto kichik hajmli gematomalar ham erta okklyuzion gidrotsefaliya va bodomsimoncha churrasini (tonsillyar herniatsiya) chaqirishi mumkin. “Yorug‘ interval”li klassik kechish ko‘proq OKCh epidural gematomalariga xos, subdural va intratserebellar gematomalar esa ko‘pincha supratentorial shikastlar hamda birlamchi sop kontuziyasi bilan birga uchrab, infratentorial o‘choqli belgilarni niqoblaydi. Supra- va subtentorial kombinatsiyalangan travmalar diagnostik noaniqlikni oshiradi va natijalarga ta’sir ko‘rsatadi.
Xulosa: OKCh travmalari klinik tashxis uchun murakkab va yuqori xavfli TBI turidir. Kechish

variantlarini erta tanish, sop/okklyuzion “qizil bayroq” belgilarini aniqlash va travma mexanizmini hisobga olish klinik qarorlarni optimallashtirishda muhim.
Kalit so‘zlar (O‘zbek): orqa kalla chuqurchasi; bosh miya jarohati; OKCh epidural gematoma; OKCh subdural gematoma; intratserebellar gematoma; miya sopi siqilishi; okklyuzion gidrotsefaliya; tonsillyar herniatsiya.

Inroduction. Damage to the posterior cranial fossa (PCF) structures is a poorly studied form of traumatic brain injury (TBI), considered a severe and rare form of brain damage, presenting significant difficulties for clinical diagnosis and accompanied by a high mortality rate [27]. The term "damage to the posterior cranial fossa structures" refers to traumatic injuries to the structures located under the tentorium cerebelli: epidural and subdural hematomas, intracerebellar and intrastem hematomas, cerebellar and brainstem contusions, hemorrhages into the fourth ventricle [5, 9]. Damage to the PCF structures accounts for 0.1-0.6% of all cases of TBI [10, 18, 34, 48]. Damage to the PCF structures is significantly more common in patients with severe TBI, multiple hematomas and brain contusions. Among traumatic intracranial hematomas, the proportion of posterior cranial fossa hematomas (PCFH) is 2-3% [4, 11, 19]. In the pathogenesis of the occurrence and development of hematomas, the mechanism and nature of the injury, as well as the type of bleeding source, are of primary importance. In relation to the site of application of the force, hemorrhages, similar to contusion foci, are divided into impact and counter-impact [20]. A typical site of application of the traumatic agent is the cervicoccipital or parieto-occipital regions [4]. The resulting fracture with damage to the membranes and brain matter by the local impression type causes the development, first of all, of epidural PCFH. Much less frequently, PCFH (mainly subdural and intracerebellar) are formed by the counter-impact mechanism - when the traumatic agent is applied to the frontal region [18, 36]. Researchers distinguish three possible variants of damage to the PCF: 1 - the damage focus is limited only to the PCF formations; 2 - damage to the PCF structures in the form of epidural hematomas extending to the supratentorial level, sometimes in combination with foci of supratentorial brain contusions; 3 - damage to the PCF structures combined with supratentorial brain damage and not anatomically related [10, 27]. Among the traumatic pathology of the PCF formations, epidural hematomas are the most common, accounting for 4-14.3% of all epidural hematomas of the brain and 20-64% of all PCF injuries. In the structure of all traumatic brain injuries, they account for 0.3% [4, 12, 19, 23, 25, 41, 44, 48]. In most cases, epidural hematomas of the posterior cranial fossa are located above one of the cerebellar hemispheres; significantly less frequently (with damage to the sinus confluences), they are located bilaterally [2, 34]. In 35-66% of cases, epidural hematomas of the posterior cranial fossa extend supratentorially to the posterior surface of the occipital and temporal lobes, thereby displacing the transverse sinus and the sinus drainage site from the occipital bone [10, 18, 46]. The source of epidural hematomas of the posterior cranial fossa are the vessels of the damaged dura mater (DMA), the veins draining into the sinuses, and the veins crossing the epidural fissure.The relatively small volume of epidural hematomas of the PCF compared to supratentorial epidural hematomas is explained primarily by the small volume of the PCF and the fusion of the dura mater with the bone, which limits the spread of supratentorial hemorrhages [28, 31]. Larger supra- and subtentorial hematomas are formed in cases where the source of bleeding is the transverse sinus or supratentorial meningeal arteries, damaged during the spread of the fracture line from the PCF through the pyramids of the temporal bone into the middle cranial fossa [13, 24]. According to observations of specialists, epidural hematomas of the posterior cranial fossa more often than other injuries of the subtentorial localization occur without combination with other traumatic intracranial pathology [47, 54]. Anatomical features of the skull structure determine the high incidence of supratentorial injuries in trauma to the occipital region [46]. The severity of supratentorial injuries may vary from brain

contusions or minor hemorrhages to significant crush lesions and hematomas of the cerebral hemispheres. Most frequently, with a combination of sub- and supratentorial injuries, supratentorial intracranial hematomas, contusions, and hygromas are encountered [9]. Hematomas of the PCF differ from supratentorial hematomas in their relatively small volume (in most cases, up to 30 ml). This is explained by the much smaller size of the subtentorial space compared to the supratentorial space. Moreover, with PCF, due to their proximity to the main pathways of cerebrospinal fluid circulation, the effect of occlusive hydrocephalus, accelerating the increase in intracranial pressure, may be added early to the direct compressive effect. Sometimes PCFs reach a significant volume - 50-70 ml [26]. It should be taken into account that due to the small volume of the posterior cranial fossa and the ease of compression of the cerebrospinal fluid pathways, relatively small hematomas (15-30 ml) can often contribute to the herniation of the cerebellar tonsils into the occipitocervical dural infundibulum and compression of the medulla oblongata, which leads to a fatal outcome [4]. Subdural hematomas of the PCF are much less common; they account for approximately 5% of all PCF injuries and 0.5% of all traumatic subdural hematomas [37, 39, 52]. Their volume is significantly smaller and, as a rule, does not exceed 15-20 ml. The formation of subdural CPH is mainly associated with damage to the transverse or sigmoid sinuses, the veins draining into these sinuses, as well as the cortical vessels of the cerebellum. According to observations of clinicians, subdural hematomas of the posterior cranial fossa, unlike epidural hematomas, are extremely rarely the only result of trauma and are often accompanied by severe brain contusions and/or hematomas of other localizations, which determines the variability of their clinical manifestations [10, 14, 18, 58]. Intracerebellar hematomas of traumatic etiology are as rare as subdural hematomas of the posterior cranial fossa. [4, 24]. According to the available literature, Focal cerebellar injuries account for 15.3-26% of traumatic pathologies of the posterior cranial fossa [15, 28, 42]. The source of intracerebellar hematomas is also the cerebellar vessels. They are typically localized on the inferior surface of the hemisphere, corresponding to the longitudinal fissure of the occipital bone [20]. Cerebellar injuries are extremely rarely isolated [6]. Contusion foci are often accompanied by lamellar subdural hemorrhage [20]. Intracerebellar hematomas may be located in close proximity to hemorrhagic contusion foci or be associated with them. In this case, they are usually localized in the cerebellar hemispheres or represent completely isolated foci, located closer to the midline or in the cerebellar vermis. In cerebellar contusions, early fusion of hemorrhage foci occurs, forming a zone of continuous hemorrhagic necrosis [9, 38]. There is also evidence of the possibility of delayed development of intracerebellar hematomas [7, 16, 27]. Multiple hematomas of the posterior cranial fossa (subdural and intracerebellar) may be a consequence of the rupture of primarily occurring intracerebellar hematomas into the subdural space [38]. Multiple subtentorial hematomas are often caused by vascular anomalies, and trauma in these cases is a provoking factor in the development of hematomas both deep in the cerebellum and in the subdural space [13, 53, 58]. Thus, the presence of combined supra- and subtentorial injuries is characteristic of posterior cranial fossa injury. The combination of traumatic injuries to the structures of the PCF with supratentorial injuries occurs in 40-64.1% of cases [10]. Injuries to the PCF are characterized by a severe condition of patients upon admission, caused by both the severity of the brain injury and combined extracerebral injuries [10]. The clinical picture of PCF injuries is characterized by a combination of general cerebral, hemispheric, cerebellar and brainstem symptoms. The most common symptom in the presence of verbal contact with patients is headache. Headache occurs immediately after the injury, is characterized by significant severity and is practically not relieved by analgesics; pain in the occipital region is often noted. In some cases, headache is the only clinical manifestation of PCF injury, especially in the subacute type of the course. With the development of occlusion syndrome, forced position of the head, increased headache and vomiting when changing body position in space, bradycardia are observed [8]. Impaired consciousness is one of the most reliable signs of TBI. A certain level of consciousness is maintained by continuous efferent impulses from neurons in the

reticular formation of the brainstem. A reduction or cessation of this impulse flow during TBI leads to impaired consciousness in patients. Damage to the structures of the posterior cranial fossa, located in close proximity to the structures of the reticular formation, significantly affects the level and dynamics of consciousness [1, 8].27-65% of victims are admitted in a comatose state, some kind of disturbance of consciousness is detected in the overwhelming majority of patients, and only in isolated cases are victims admitted in a clear consciousness. In many patients, disturbances of consciousness rapidly worsen during the diagnosis and preparation for surgery, which is explained by the rapid development of brainstem dislocation and occlusive hydrocephalus [16]. Meningeal symptoms are observed in the majority of victims. And although the meningeal symptom is not specific for injuries to the posterior cranial fossa, a characteristic diagnostic sign in victims with injuries to the posterior cranial fossa can be the phenomenon of a significant predominance of neck muscle rigidity over Kernig's sign due to local irritation of the V, IX and X cranial nerves innervating the infratentorial part of the sheath [10, 29]. Brainstem symptoms in injuries to the posterior cranial fossa in most cases are caused by secondary brainstem dislocation, impaired hemodynamics, and hypoxia. Dislocation bulbar syndrome includes pain in the occipital and cervical regions, forced head position, paresthesia in the caudal Zelder zones and in the innervation zones of the first to third cervical segments, dysarthria, dysphagia, dysphonia, vomiting, hiccups, and bilateral pathological symptoms. Bradycardia, increased blood pressure, cyanosis, and acute muscle hypotension later develop. Death may occur as a result of sudden respiratory arrest. Secondary bulbar syndrome is often characterized by a rapid course. Following the manifestation of initial reversible symptoms, life-threatening and then irreversible dysfunction of the caudal brainstem occurs [28]. Neurological manifestations in posterior fossa hematomas are polymorphic. Cerebellar symptoms are most pronounced in intracerebellar hematomas with a subacute course. In subacute epidural and subdural hematomas, the clinical picture of the injury mainly consists of general cerebral symptoms with mildly expressed discoordination disorders [4]. Cerebellar symptoms in epidural hematoma of the PCF are caused by the direct impact of the hematoma on the cerebellum, circulatory disorders in the compression zone, and concomitant cerebellar contusion with tissue damage [12, 18]. Muscle hypotonia and coordination disorders are determined homolateral to the affected cerebellar hemisphere. Also, in patients with damage to the structures of the PCF, asynergy, ataxia, balance disorders, and large-scale spontaneous nystagmus can be detected [24]. Clinical signs of damage to the cerebral hemispheres in the form of mild hemiparesis, motor, sensory or amnestic aphasia, central paresis of the VII and XII cranial nerves, hemianopsia are observed in 17-25% of victims with trauma to the structures of the posterior cranial fossa [12, 23, 41, 47]. Examination of the fundus reveals signs of intracranial hypertension in many victims: swelling of the optic disc, retinal hemorrhages, tortuosity and plethora of the retinal veins [29].Damage to the structures of the PCF can be acute and subacute (from 3 days to 3 weeks), a chronic form of the course (more than 3 weeks) is extremely rare. Acute clinical course occurs in 50-84% of cases, subacute - in 13-27%, chronic - in 2-4.5%. Depending on the type of clinical course, there are variants: classic - with an expanded clear interval, a variant with an erased clear interval and without a clear interval. Diagnosis of damage to the formations of the PCF by nosological forms is extremely difficult due to the similarity of symptoms, but a number of clinical symptoms characteristic of various forms can be identified [8]. The following clinical signs are characteristic of epidural PCF: localization of the site of application of the traumatic agent in the cervicoccipital region; the presence of an occipital bone fracture confirmed by craniography (posterior semi-axial image) or, indirectly, by swelling and compaction of the soft tissues in the cervical-occipital region; local pain in the cervical-occipital region, a close connection between its intensification and a change in the position of the head and body in space; a pronounced tendency toward a fixed position of the head, with the preferred position of the patient on the side corresponding to the side of the hematoma; distinct rigidity of the occipital muscles; development of focal cerebellar-brainstem symptoms against the background of

clinical manifestations of cerebral compression syndrome with early occlusive features (hypotonia in the limbs, coordination disorders, bulbar disorders, spontaneous nystagmus, damage to the caudal cranial nerves, pyramidal signs; in this case, prevalence of pronounced cerebellar pathology on the side of the hematoma location may be observed, and comparatively mild pyramidal symptoms on the contralateral side) [40]. The classic variant of acute epidural hematomas of the PCF is characterized by a clearly expressed three-phase disturbance of consciousness: primary loss of consciousness at the time of injury - complete or almost complete recovery - secondary loss of consciousness. The classic variant with an extended lucid interval is characteristic of epidural hematomas of the PCF more than other injuries to the PCF structures. The lucid interval is usually short-lived, lasting tens of minutes. Upon regaining consciousness, patients complain of a severe headache localized in the occipital region. Against a background of pronounced general cerebral and meningeal symptoms, cerebellar symptoms are detected, highly likely indicating a pathological process in the posterior cranial fossa. Impaired consciousness worsens to the point of coma, and brainstem dislocation symptoms increase [30]. A variant with an erased lucid interval: the soporic state that develops immediately after the injury is followed by obtundation within a few hours. Against this background, signs of focal lesions of the posterior cranial fossa structures are detected. Parallel to or preceding their progression, a secondary deepening of impaired consciousness, leading to coma, develops in the next few hours or days.In the third variant, coma from the moment of injury without any lucid interval and with particularly pronounced bulbar disturbances in the next few hours or days ends in death if it cannot be prevented by immediate surgery. Variants with an erased lucid interval or without it are more common with epidural hematomas of the PCF, combined with supratentorial injuries or primary brainstem contusion. In this case, cerebellar symptoms are masked by brainstem and hemispheric symptoms, which greatly complicates diagnosis [8, 50]. The classic variant of the subacute course of epidural PCF is characteristic of small-scale injuries. The initial loss of consciousness after injury is short-lived. In the subsequent lucid interval, the patient's consciousness is restored or only moderate confusion remains. Against this background, an increase in headaches is noted over a period of several hours to several days, predominantly localized in the cervicoccipital region. A tendency toward a fixed head position is evident; attempts to change body and head position in space cause dizziness, vomiting, and a sharp worsening of headache. Focal symptoms of cerebellar and brainstem damage, as well as meningeal signs, are detected. Craniograms typically reveal a fissure of the occipital bone extending through the projection of the transverse sinus. Initial congestive optic nerve papillae are detected early, and the patient's general condition worsens with fluctuating changes in consciousness within the range of obtundation. If surgical intervention is not performed, severe swallowing and cardiovascular disorders develop, and secondary loss of consciousness, including coma, develops. A variant with an erased lucid interval: a soporotic-comatose state occurring immediately after the injury is followed within a few hours or days by profound obtundation. Signs of damage to the posterior cranial fossa structures predominate among the focal symptoms. Congestive optic nerve papillae are detected. After several days or 1-2 weeks, a secondary loss of consciousness occurs, leading to stupor or coma, with increasing impairment of vital functions [19, 33, 51]. Subdural hematomas, unlike epidural hematomas of the same location, can develop in the absence of cranial bone damage. In a significant number of cases, they are accompanied by cerebellar damage and primary brainstem contusion, which determines the variability of their clinical manifestations [57]. In most cases, the clinical picture of subdural hematomas is characterized by pronounced general cerebral symptoms with secondary impairment of consciousness after a lucid interval, increasing headaches, vomiting, and increased cerebrospinal fluid pressure. Among the focal symptoms, certain cerebellar-stem disorders are noted - muscle hypotonia, ataxia, nystagmus, suppression of corneal reflexes, etc. In some cases, it is possible to distinguish the phasic course and different rates of development of subdural hematomas of the PCF [8, 52].Subdural cerebral palsy develops acutely or subacutely. Isolated cases of chronic subdural

cerebral palsy have been described. Acute subdural cerebral palsy, depending on the dynamics of consciousness disturbances, is characterized by three clinical courses: with an expanded lucid interval; with an effaced lucid interval; or without a lucid interval. Subacute subdural cerebral palsy has two clinical courses: with an expanded lucid interval; and with an effaced lucid interval. In subacute subdural cerebral palsy, the course of TBI is often clearly phasic. Initial loss of consciousness after injury is followed by full or partial recovery, which lasts from several days to several weeks. Usually, it is possible to detect an increase in focal (cerebellar-brainstem) symptoms, which is combined with a deepening of general cerebral symptoms (increased headache, repeated vomiting, psychomotor agitation, bradycardia, the appearance of congestion of the optic nerve papillae, secondary loss of consciousness) [17, 37]. The clinical manifestations of intracerebellar hematomas are varied, which depends both on the features of their localization and on the nature and severity of concomitant injuries to the skull and brain. With intracerebellar hematomas and cerebellar contusions, cerebellar symptoms are most clearly expressed, which become dominant [29]. If intracerebellar hematomas are isolated, then an expanded or erased lucid interval is often noted. Severe clinical manifestations develop against the background of stupor with complaints of severe headaches and dizziness. Sometimes a tendency toward a forced head position is clearly evident, even during psychomotor agitation. Pulse, blood pressure, and external respiration are subject to various dynamic changes, due to the close proximity of the intracerebellar hematoma to vital centers of the brainstem. Cerebellar injuries are more typically acute. Acute intracerebellar hematomas exhibit three clinical patterns (with or without a clear lucid interval), while subacute forms exhibit two patterns (with or without a clear lucid interval). Cerebellar hemispheric injuries are more typically characterized by decreased muscle tone in the limbs, severe impairment of coordination tests, adiadochokinesia, and large-scale nystagmus. Symptoms are manifested homolateral to the damaged hemisphere. Injuries localized in the cerebellar vermis are characterized by bilateral cerebellar symptoms, which are less pronounced. Hypotension, static disturbances, and mild disturbances in coordination tests are predominantly observed [8, 21]. Rarely, hematomas of the fourth ventricle are encountered, which are characterized by an extremely severe condition of the victim from the moment of injury and threatening impairment of breathing and other vital functions [17, 24, 38].Depending on the dynamics of consciousness disorders, three variants of the clinical course are characteristic: with an expanded luminous interval; with an erased luminous interval; without a luminous interval. For subacute subdural cerebral hemorrhage, two variants of the clinical course are distinguished: with an expanded luminous interval; with an erased luminous interval. In subacute subdural cerebral hemorrhage, the phasic nature of the course of TBI is often clearly visible. The primary loss of consciousness after injury is replaced by its complete or partial recovery, which lasts from several days to several weeks. Usually, it is possible to detect an increase in focal (cerebellar-brainstem) symptoms, which is combined with a deepening of general cerebral symptoms (increased headache, repeated vomiting, psychomotor agitation, bradycardia, the appearance of congestive papillae of the optic nerves, secondary loss of consciousness) [17, 37]. The clinical manifestations of intracerebellar hematomas vary depending on their location and the nature and severity of any accompanying cranial and brain injuries. In intracerebellar hematomas and cerebellar contusions, cerebellar symptoms are most pronounced and become dominant [29]. If intracerebellar hematomas are isolated, a widened or blurred lucid interval is often observed. Pronounced clinical manifestations develop against a background of confusion with complaints of severe headaches and dizziness. Sometimes a tendency to a forced head position is clearly expressed, even during psychomotor agitation. Pulse, blood pressure, and external respiration are subject to various dynamic changes, which is due to the immediate proximity of the intracerebellar hematoma to vital centers of the brainstem. Cerebellar injuries are more typically acute. In acute intracerebellar hematomas, three clinical variants are observed (with a developed, erased, and without a clear space), while in subacute forms, two variants are encountered (with a developed and

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Conclusion. The above data demonstrate the extreme complexity of the problem of traumatic injuries to the posterior cranial fossa structures. Although the development of the main clinical symptoms and their severity in patients with traumatic injuries to the posterior cranial fossa directly depend on the nature, severity, and extent of the injury, their manifestation is highly individual and variable, significantly complicating clinical diagnosis and determining treatment criteria. The authors deliberately did not analyze the specifics of instrumental diagnostics and treatment in patients with traumatic injuries to the posterior cranial fossa structures; these issues will be addressed in subsequent studies.

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