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КЛАССИФИКАЦИЯ И КЛИНИЧЕСКАЯ СИМПТОМАТИКА ЧЕРЕПНО-МОЗГОВЫХ ТРАВМ ДЛЯ ОБУЧЕНИЯ СТУДЕНТОВ МЕДИКОВ СТАРШИХ КУРСОВ

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Аннотация

Актуальность проблем связанных с черепной мозговой травмой (ЧМТ) и ее последствиям, продолжает привлекать внимание общества и специалистов в сфере медицины в том числе в нейрохирургии. Черепно-мозговая травма занимает 3 место среди ведущих причин смертности и инвалидности населения, как и детского так и взрослого. Цель этой статьи является облегчением понимания студентов медиков в изучении черепно-мозговой травмы, ее классификации и клинической картины.

Ключевые слова: черепно-мозговая травма (ЧМТ), сотрясение головного мозга, ушиб головного мозга, диффузное аксональное повреждение головного мозга, сдавление головного мозга, сдавление головы, гематомы внутричерепные.

THE CONCEPT OF CLASSIFICATION AND CLINICAL SYMPTOMS OF TRAUMATIC BRAIN INJURY AIMED AT FACILITATING THE TRAINING OF SENIOR MEDICAL STUDENTS

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ABSTRACT

The relevance of problems related to traumatic brain injury (TBI) and its consequences continues to attract the attention of society and specialists in the field of medicine, including neurosurgery. Traumatic brain injury ranks 3rd among the leading causes of death and disability in the population, both children and adults. The purpose of this article is to facilitate the understanding of medical students in the study of traumatic brain injury, its classification and clinical picture.

Keywords: traumatic brain injury (TBI), concussion, brain contusion, diffuse axonal brain injury, brain compression, head compression, intracranial hematomas.

Introduction:

Traumatic brain injury (TBI) is a traumatic injury to the skull, meninges, brain substance, cranial nerves and vessels of the meninges and brain, accompanied by general cerebral, focal neurological, meningeal symptoms with possible morphological changes.

The causes of traumatic brain injury are mainly criminal injuries, road accidents, falling from a height, an earthquake. Every year in Russia, more than 30,000 people die from traumatic brain injuries as a result of road accidents.

Classification of traumatic brain injury:

Traumatic brain injury is classified variously according to the following parameters:

1 - On the biomechanism of TBI development:

* Shock-proof when the head is exposed to traumatic force, this is possible with a direct blow to the head and a fall from a height.

* Acceleration-deceleration the impact of traumatic force on the head in the absence of direct contact with the head during injury, as usually happens in a car accident.

*Combined

2 - According to the genesis of brain damage TBI:

* Primary brain damage due to direct exposure to traumatic factors.

* Secondary brain damage due to other intracranial causes such as cerebral edema, increased intracranial pressure (ICP), mixed median structures(dislocation), and brain deformity.

3-By type of traumatic brain injury:

* Isolated TBI – in the absence of any extracranial injuries, that is, only a head injury with damage to the bones of the skull, meninges, or brain substance.

* Combined TBI - in addition to craniocerebral injuries, extra-cranial injuries are added as injuries of other localizations (injuries of the neck, chest, spine, abdomen, pelvis, limbs).

* Combined TBI - with simultaneous exposure to different types of traumatic energies, including: mechanical, chemical, thermal or radiation.

4 - By the nature of the TBI injury:

* Closed TBI is a variety of injuries in the absence of a violation of the integrity of the integument of the head or there are wounds to the soft tissues of the scalp without damage to the aponeurosis. Various types of fractures of the bones of the arch that are not accompanied by an injury to the adjacent soft tissues of the head and aponeurosis are considered as closed TBI.

* Open TBI is injuries in which there are soft tissue wounds of the head with aponeurosis damage, or fractures of the base of the skull with aponeurosis damage accompanied by liquorrhea, bleeding from the nose or ear. In turn, open TBI extends to non-penetrating without damage to the dura mater and penetrating with damage to the dura mater (TMO)[1].. .

With open craniocerebral injuries, there is a high risk of primary and secondary infection of the meninges and brain substance with the possible development of meningitis; encephalitis; meningoencephalitis; meningococcemia; abscess and empyema of the brain; eventually sepsis.

5-Downstream of TBI:

* Acute period of TBI - from the moment of receiving a traumatic brain injury to stabilization at various levels of functional disorders up to 2 months for mild TBI, for moderate TBI up to 4 months, for severe TBI up to 6 months.

* The intermediate period of TBI is an early recovery period from stabilization of functional disorders to their stable compensation, from a few days to 2 weeks for mild TBI, for moderate TBI up to 5 weeks, for severe TBI up to 10 weeks.

* The long-term period of TBI is a late recovery period, characterized by clinical recovery, possible rehabilitation of impaired functions, or the appearance and progression of functional disorders caused by new injuries and pathological conditions. With clinical recovery, the period lasts up to 2 years, and with a progressive course, it is not limited.

6 - By type of brain injury TBI:

*Focal with local brain damage;

* Diffuse with diffuse brain damage;

* Combined with focal-diffuse brain damage [2].

7 - By clinical forms and severity of TBI:

I- Minor traumatic brain injuries:

1-Concussion of the brain (SGM)– one of the mild forms of traumatic brain injury, characterized by the absence of any signs of damage to the bone structure of the skull, brain substance with reversible functional disorders of the brain.

Clinical symptoms of concussion:

* General brain symptoms: loss of consciousness from a few seconds to 10-15 minutes, brief memory loss often retrograde amnesia is noted (the patient does not remember events that occurred before the traumatic brain injury); single vomiting; nausea; dizziness of a systemic nature; headache of varying intensity.

* Vegetative symptoms: tinnitus; moderate tachycardia or bradycardia; fluctuating blood pressure; sweating.

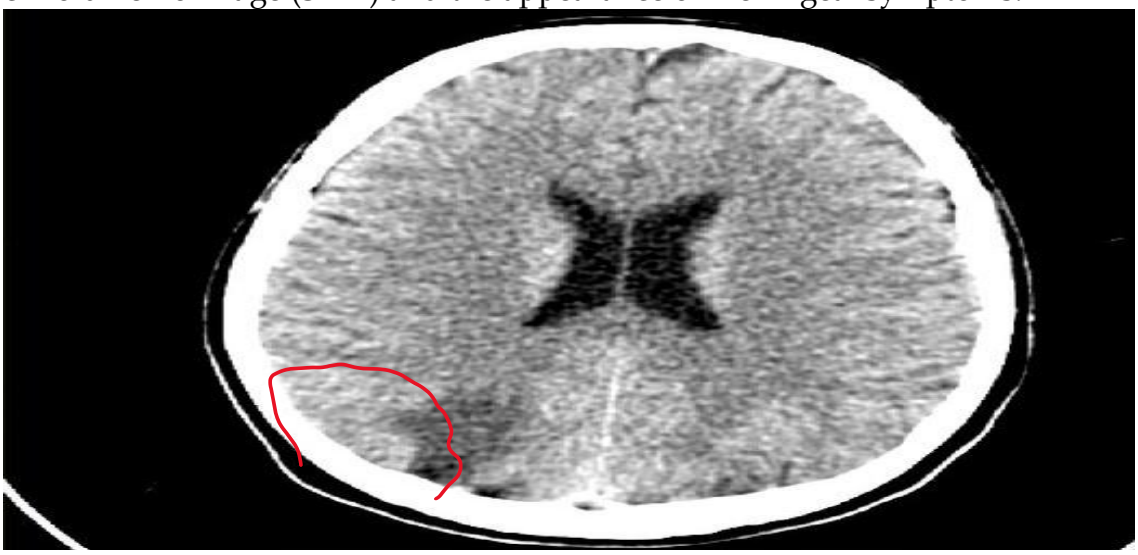
* Neurological symptoms: fine-pitched nystagmus, lability and anisoreflexia.

With SGM, the following fact should be noted: the possibility of a brief appearance of meningeal symptoms as rigidity of the neck muscles, which disappears within 3 to 7 days.

NB: If we are talking about SGM, there are no fractures of the bones of the base or vault of the skull and blood or CSF leaks from the nasal passages or ear.

Within 7-10 days, the patient's condition improves and the symptom is completely regressed.

2-Mild brain contusion – UGM)-it consists of a mild form of traumatic brain injury, characterized by the presence of damage to the bone structure (cranial vault) of the skull with subarachnoid hemorrhage (SAH) and the appearance of meningeal symptoms.



CT signs: mild brain contusion in the right occipital lobe.

Clinical symptoms of mild brain contusion (UGM):

* General brain symptoms: loss of consciousness from a few seconds from 15 minutes to 1 hour; prolonged memory loss for at least 2-3 hours; often retrograde amnesia; double or triple vomiting; nausea; dizziness of a systemic nature; severe headache.

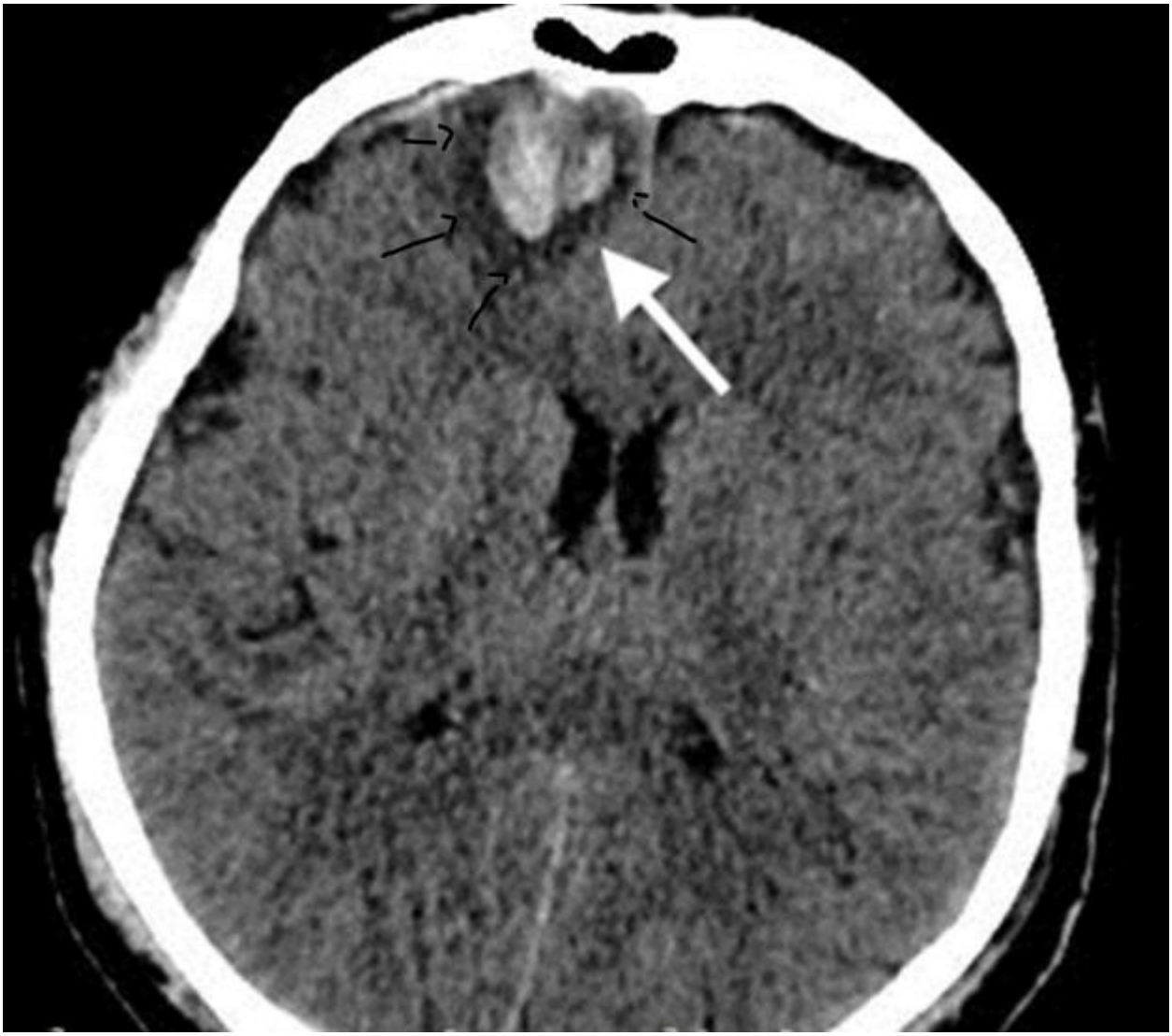
* Vegetative symptoms: tachycardia; bradycardia; fluctuating blood pressure; sweating.

* Neurological symptoms: clonic nystagmus; mild anisoreflexia; meningeal symptoms (neck muscle rigidity, Kerning's symptom, Brudzinski's symptom); pyramidal insufficiency.

NB: In mildUMM, subarachnoid hemorrhage (SAH) is mandatory. Suppression of symptoms is noted on the 14th - 18th day (2-3 weeks) [3].

II-Severe craniocerebral injuries:

1-Brain contusion of moderate severity-characterized by the presence of a skull base fracture; SAH; the appearance of intracerebral hemorrhage; meningeal; focal and general cerebral symptoms.



CT signs: moderate brain contusion (contusion hemorrhagic component) in the frontal lobe on the right.

Clinical symptoms of moderate brain contusion (UGM):

* General brain symptoms: loss of consciousness from a few seconds from 1 hour to 6 hours; prolonged memory loss of at least 3 hours; retrograde, con - or anterograde amnesia; repeated vomiting; nausea; dizziness of a systemic nature; severe headache.

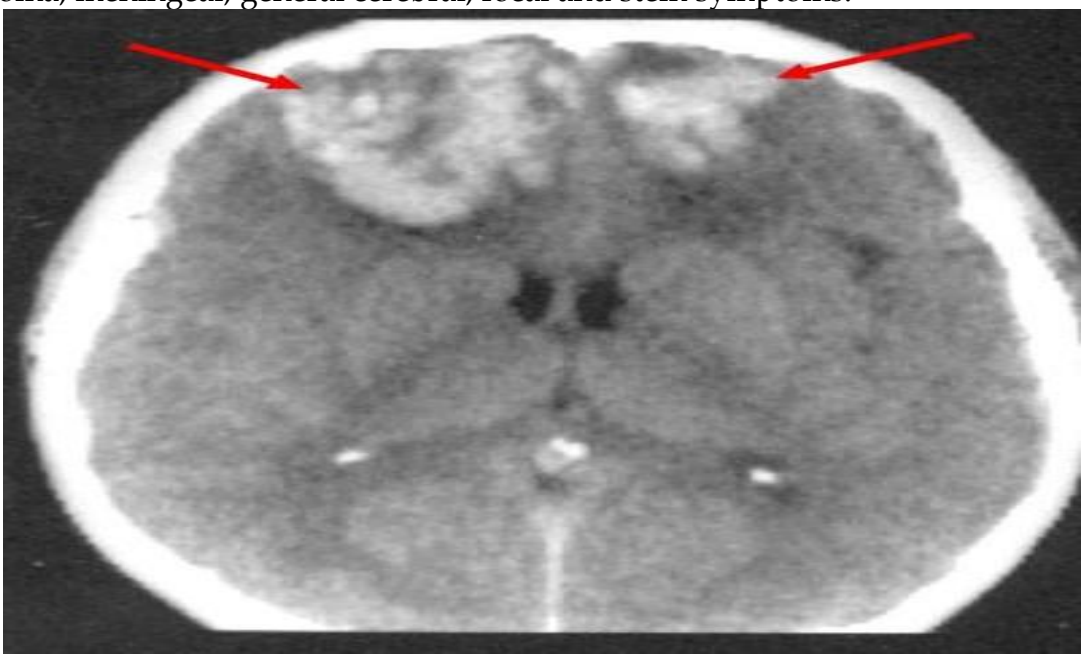
* Vegetative symptoms: pronounced tachycardia of heart rate up to 120 beats / min; bradycardia of 40-50 beats / min; symptomatic hypertension up to 140-180/90-100mmHg;

sweating, tachypnea without violation of the respiratory rhythm; violation of the sleep – wake cycle during the day in the form of drowsiness and insomnia at night.

*Neurological symptoms: psychomotor agitation; nystagmus; mild meningeal symptoms; meningeal symptoms; dissociation of the tendon reflex and muscle tone; focal symptoms associated with the localization of brain contusion (paresis, dysarthria, aphasia, dysphagia, hypesthesia, anesthesia, miosis, mydriasis, anisocoria, etc.).

NB: Otorrhea, nazorrhea, and subfebrile fever occur in moderate SGM. Improvement in the patient's condition is observed in 21-35 days or more.

2 – Severe brain contusion (UGM) - characterized by the presence of a fracture of the base and vault of the skull; SAH; the appearance of; intracerebral hemorrhage or intracerebral hematoma; meningeal; general cerebral; focal and stem symptoms.



CT signs: severe brain contusion with intracerebral hematoma in both frontal lobes.

Clinical symptoms of severe brain contusion (UGM):

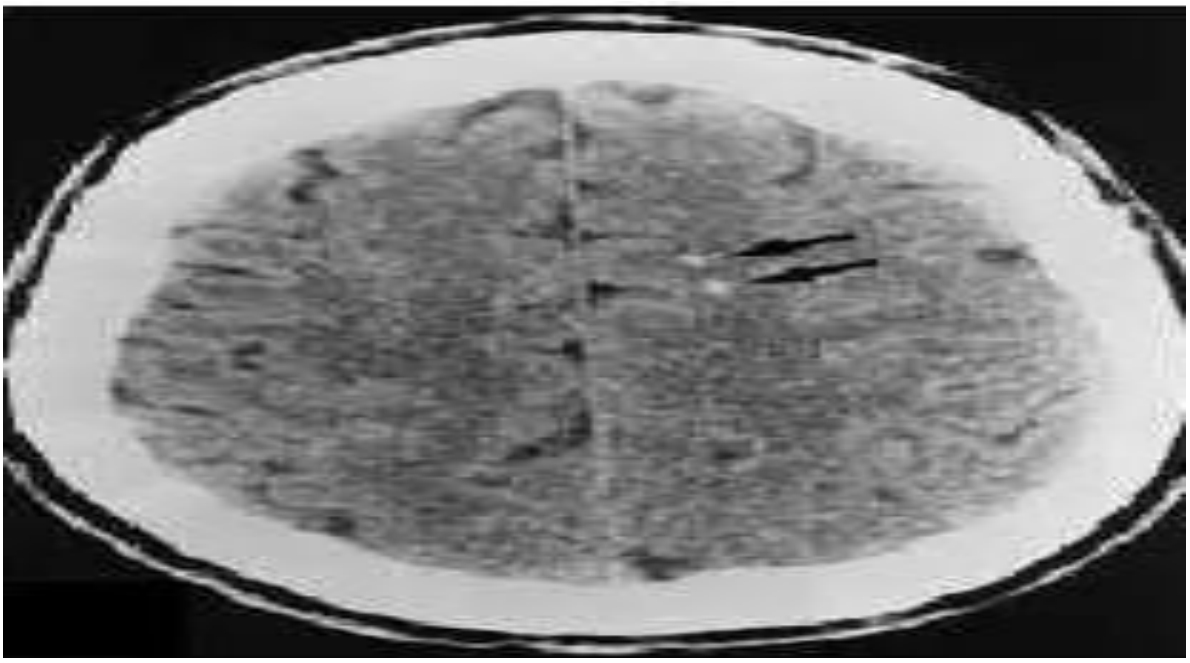
* General brain symptoms: loss of consciousness from 6 hours to several weeks to months usually after an injury, patients immediately go into a coma and come out of a coma with prolonged retrograde, con - or anterograde amnesia.

* Vegetative symptoms (disorders of vital functions): paroxysmal tachycardia of heart rate greater than 120 beats / min; or bradycardia of heart rate less than 40 beats / min; malignant hypertension of blood pressure above 180/100 mmHg or hypotension of blood pressure below 90-80 / 60-50 mmHg; profuse sweating, tachypnea greater than 30-40 or bradypnea 8-12 d / min with respiratory rhythm disturbances; threatening hyperthermia.

* Neurological symptoms: psychomotor agitation; meningeal signs; abnormal foot symptoms Babinsky's symptom often occurs when a motor neuron of the corticospinal pathway(pyramidal pathway) or cara of the brain is affected; contralateral hemiparesis or hemiplegia; alternating syndrome; multiple nystagmus; mydriasis on the side of the lesion or bilateral mydriasis; miosis; ptosis, swallowing disorders; paresis vision loss; floating eyeballs; convulsive, abnormal tendon reflexes, aphasia, anesthesia, dysphagia, dysarthria.

NB: With severe SGM, threatening hyperthermia is noted. Regression of symptoms is very slow over 2-6 months.

3-Diffuse axonal brain injury (DAP) is one of the most severe forms of PTHM that usually occurs in young people and children. Its mechanism of development is acceleration-deceleration with rotation, in which extensive damage to the axonal cells of the brain and the brain stem(micro-hemorrhages) occurs.



CT signs: diffuse axonal brain damage with petechial hemorrhages.

Clinical symptoms of diffuse axonal brain injury (DAP):

* Common brain symptoms: prolonged comatose state.

* Vegetative symptoms (disorders of vital functions): hypersalivation, hyperhidrosis, hyperthermia; respiratory disorders; bradycardia.

* Neurological symptoms: signs of decerebration or decortication; hypotension, hyporeflexia or areflexia, meningeal signs, paresis of the extremities; loss of para cephalic reflexes; facial synkinesis, bradykinesia; increased ICP (intracranial pressure); mental disorders.

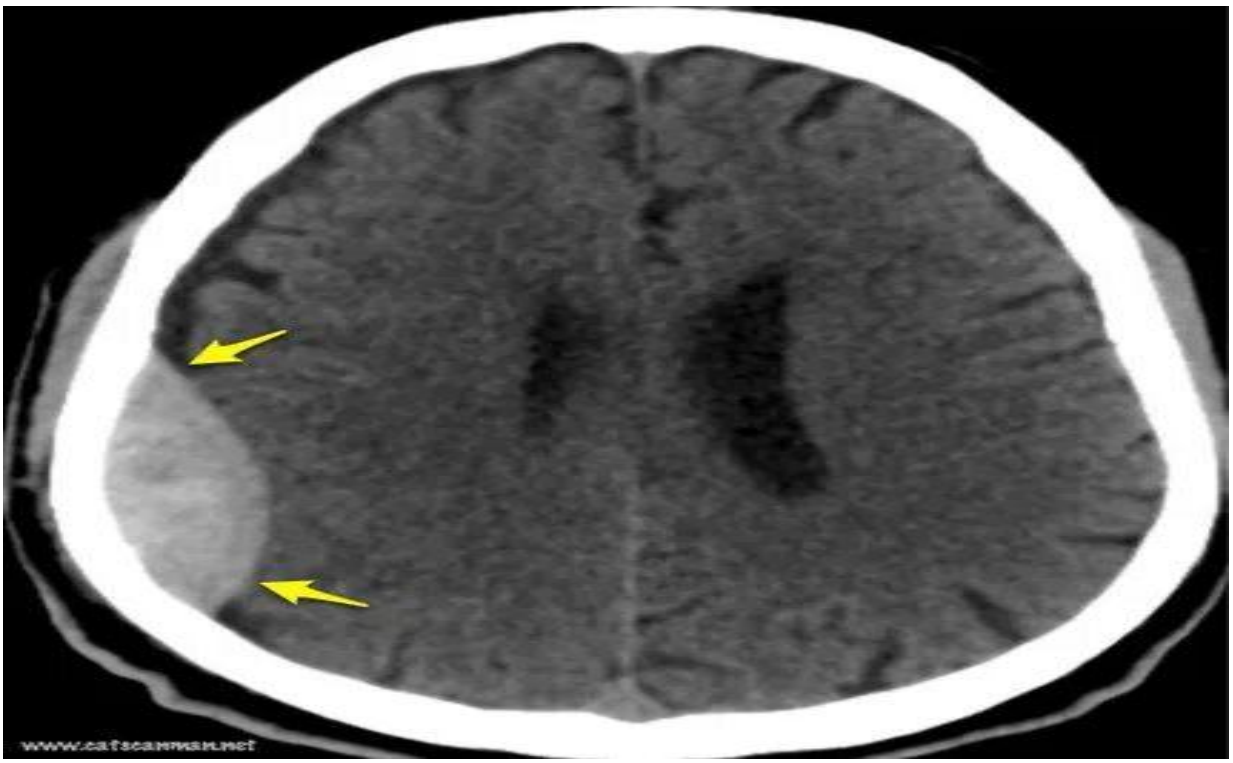
NB: In DAP of the brain, the transition from a comatose state to a transient and vegetative state occurs in stages. The severity of DAP is calculated from the moment the patient enters a coma until he comes out of it, and it can last for days, months, or years.

3-Brain compression-there is brain compression without brain contusion and brain compression with brain contusion.

The main causes of brain compression:

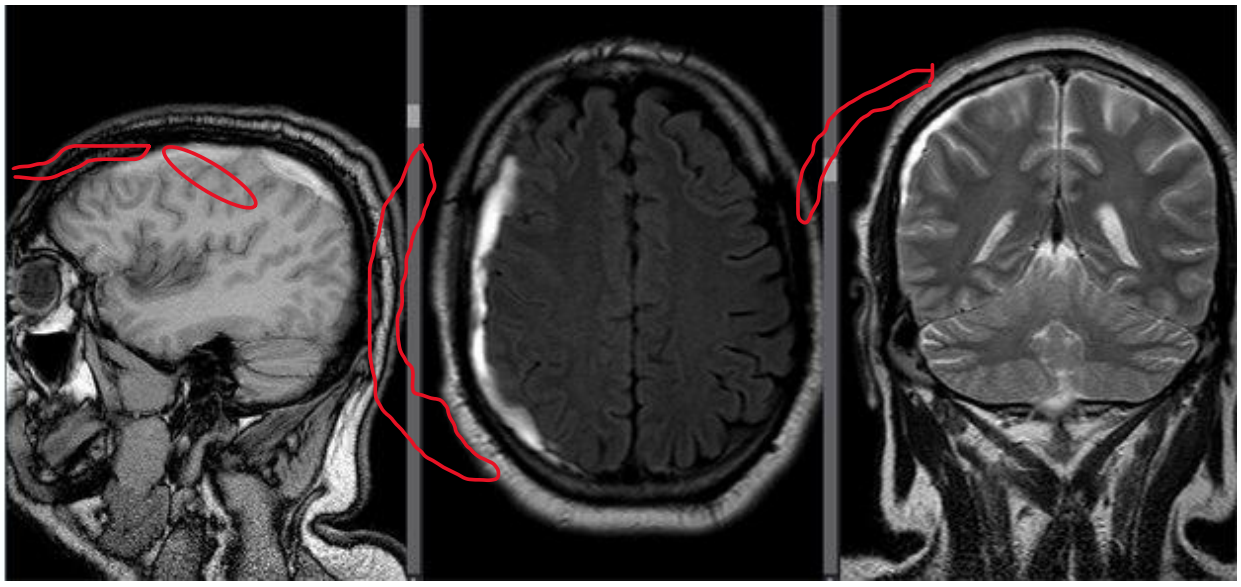
Intracranial hematomas, including epidural; subdural; intracerebral; intraventricular hematomas.

An epidural hematoma is an accumulation of blood between the inner surface of the bones of the skull (arch or base) and the dura mater of the brain.



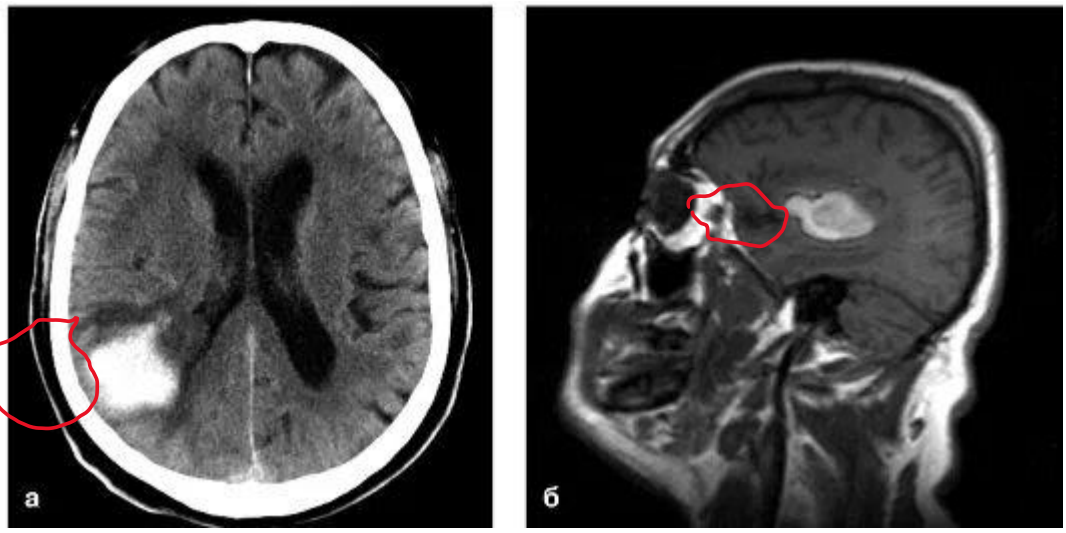
CT signs: Acute epidural hematoma in the right parietal region.

A subdural hematoma is an accumulation of blood between the inner surface of the bones of the dura mater of the brain and the web of the brain membrane.



CT signs: subdural hematoma in the right temporal-parietal region.

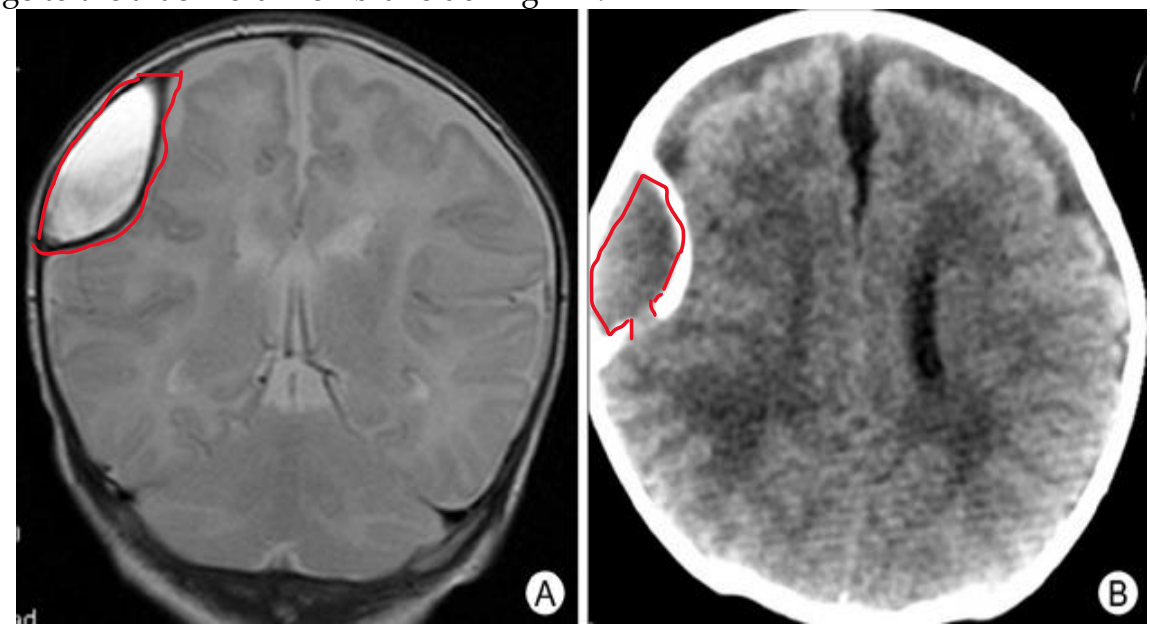
An intracerebral hematoma is an accumulation of blood in the substance of the brain.



CT signs: intracerebral hematoma in the parietal lobe on the right.

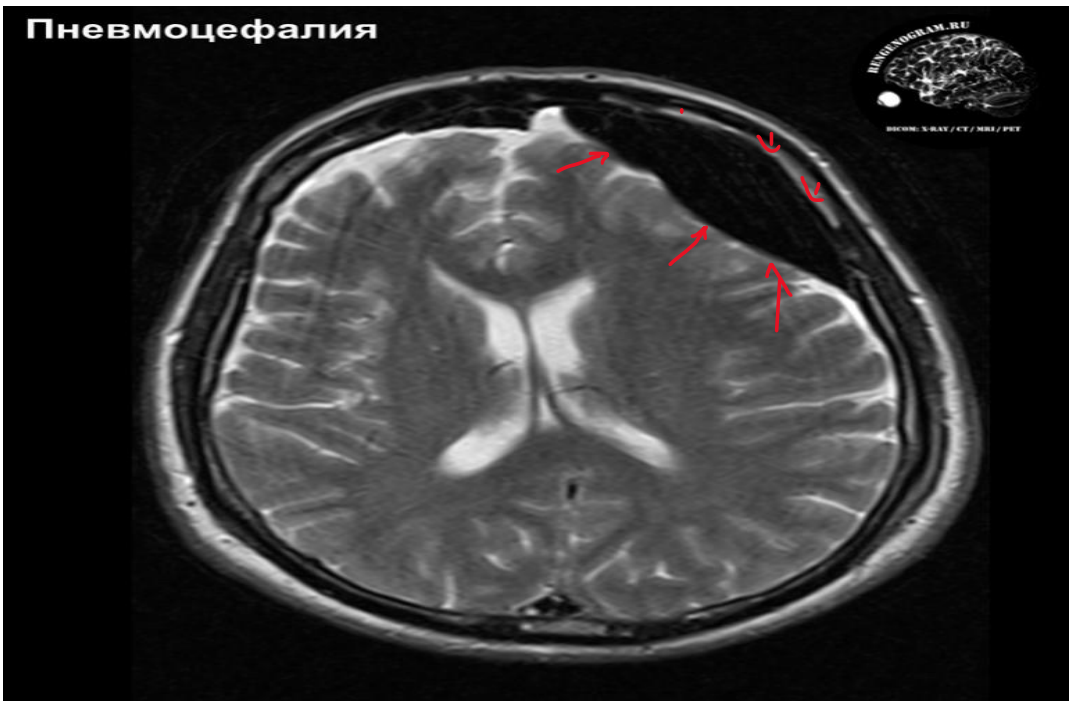
Intracranial hematomas in the course last for acute up to 3 days, subacute up to 3 weeks, chronic (with capsule formation) for more than 3 weeks.

Subdural hygroma – puncture of the cerebrospinal fluid in the subdural space due to damage to the arachnoid membrane during TBI.



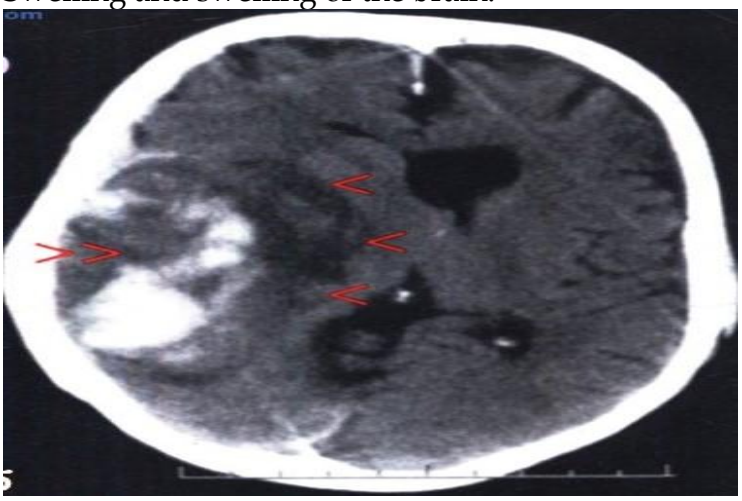
CT signs: subdural hygroma in the right temporal-parietal region.

Pneumocephalus – accumulation of air inside the skull as a consequence of TBI.



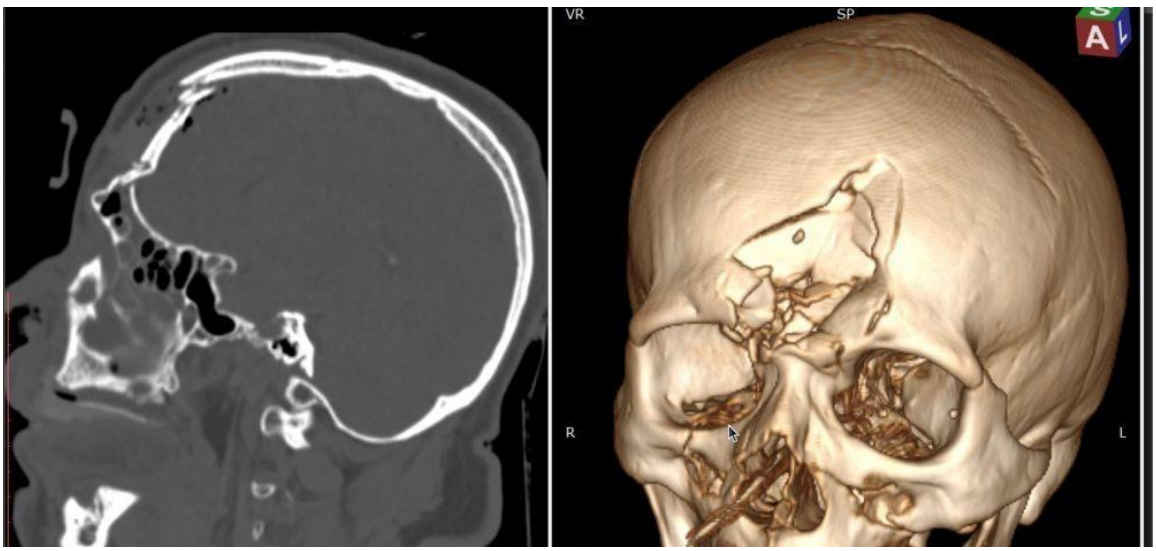
CT signs: pneumocephalus in the left frontotemporal region.

Swelling and swelling of the brain.



CT signs: intracerebral hematoma with perifocal cerebral edema in the right parietal region.

Compression of the brain by bone fragments in immersive, decompression fractures of the skull bones.



CT signs: compression fracture of the frontal region, upper orbital wall and zygomatic bone on the right. Compression by bone fragments of the frontal lobe of the brain.

Clinical symptoms of acute brain compression:

Holy gap; mydriasis from the side of the hematoma; contralateral hemiparesis; or plegia; anisocorrhea; headaches; dizziness of a systemic nature; vomiting; hyperreflexia; hypertension, convulsions; dysarthria, dysphonia, hyper or hypotension, bradycardia, tachypnea, dislocation syndrome; meningeal symptoms.

NB: Compression of the brain with an increasing dislocation syndrome of 5 mm or more requires immediate neurosurgical intervention (decompression trepanation of the skull and removal of a hematoma or hygroma).

5-Head compression is a very rare form of severe TBI because it is not common. Usually occurs in patients who have received TBI in emergency situations (catastrophes) The essence of trauma is that the head in such situations is squeezed simultaneously from both sides by objects.

Clinical symptoms of acute compression of the head:

Initially, the head is deformed.

Damage; hematomas; edema; soft tissues of the head; bone structure of the skull (compressed fractures); brain matter (edema and bruises of the brain, ICH, VHG, VHG, VHG). Dislocation syndrome.

Tissue necrosis is accompanied by intoxication; the risk of infection.

Common brain symptoms: severe excruciating headache; repeated vomiting; loss of consciousness.

Vegetative symptoms: tachycardia or bradycardia; symptomatic hypertension, sometimes followed by hypotension; profuse perspiration; tachypnea or bradypnea.

Neurological symptoms: clonic nystagmus; divergent strabismus; bilateral mydriasis; bulbar, meningeal symptoms; pyramidal insufficiency; increased intracranial pressure, stem symptoms with a possible fatal outcome (respiratory or cardiac arrest) [4].

NB: when the brain is compressed, there are 3 degrees of severity:

- Mild severity of brain compression from 30 minutes to 5 hours.
- The average severity of brain compression is from 6 to 48 hours.
- Severe degree of brain compression above 2 days.

Conclusion: understanding the mechanisms of development of craniocerebral injuries and their classification allows us to more deeply visualize and evaluate the condition of patients in the acute phase of CTM. Further tactics of management and treatment of neurosurgical patients with TBI depend directly on the basic level of knowledge and training of a neurosurgeon, so you should take the study of traumatic brain injury very seriously.

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