INTERNATIONAL JOURNAL OF
INTEGRATIVE AND
MODERN MEDICINE

Forensic Medical Assessment of the Distance of Traumatic Intracranial Hemorrhages

Saibov Khakim Mavlonovich
Bukhara State Medical Institute

Abstract: The variety of morphological manifestations of traumatic brain injury (TBI), their different forensic significance determine subsequent situations associated with the forensic medical examination of TBI, therefore it is advisable to determine a specific plan for the forensic medical examination of TBI.

Keywords: traumatic brain injury, forensic medicine, stroke, fracture.

Relevance. Forensic medical practice shows that there are objective difficulties in establishing the timing of traumatic brain injury with the presence of intracranial hemorrhages based on the morphological (histological and histochemical) changes proposed to date, especially in cases where intracranial hemorrhages are its only manifestation. Many histochemical, immunohistochemical and molecular biological markers of the body's response to damage cannot be used on cadaveric material with which a forensic expert deals. A retrospective analysis of the circumstances of the incidents and clinical data revealed that in cases of traumatic brain injury with the presence of intracranial hemorrhages, the duration of the post-traumatic period has little connection with the gender, age and state of alcohol intoxication of the victims. The source of bleeding into the epidural and subdural spaces can be either damaged arterial or venous vessels. Some subdural hematomas (up to 30%) in the presence of brain contusions are of a mixed nature, which indicates their formation from the blood of both damaged arterial trunks of the cerebral cortex and transitional veins of the subdural space. In studies of pathological manifestations and changes in the dynamics of methemoglobin concentrations, the nature and location of the source of bleeding were of little use in judging the duration of the injury. By studying the distribution of observations by volume of hematomas removed at different times during the acute period of injury, it was found that the percentage of hematomas of different volumes detected in the first 3 hours after injury remained almost the same in subsequent periods, even on the 3rd day after injury. Moreover, this pattern is not affected by either the type of intracranial hematomas or the nature of intracranial bleeding. This assumption is confirmed by studies of the morphological and biochemical evolution of hematomas, which make it possible to prove that intracranial hematomas, regardless of their type and source of bleeding, are formed in the main volume up to 3 hours after injury, possibly within the first minutes or hour. This hypothesis does not contradict the opinion of other authors. The pathomorphological signs to be assessed were: the area of collagen fibers of the dura mater with altered tinctorial properties, manifestations of a traumatic reaction in the vessels and their walls. In the substrates of hematomas adjacent to the dura mater, the percentage of changed and unchanged erythrocytes, the severity of leukocytosis represented by changed and unchanged neutrophilic leukocytes, the number of macrophages, free hemosiderin, lymphocytes, fibroblasts, as well as the state of fibrin and productive reactions during capsule formation. Histological examination of smears and smears - impressions from the liquid part of hematomas, as well as sections from their elastic part, revealed changes that occur only after a certain period of time after the injury. Features of pathomorphological changes made it possible to divide all studied cases into six groups. If death does not occur immediately after the injury, but within three hours, then histological examination naturally reveals distinct reactive changes to injury from the dura mater and the hemorrhage substrate, of which 8 are practically alternative (changes in the tinctorial properties of collagen fibers with basophilia phenomena , eosinophilia and edema, the phenomena of spasm and vascular paresis, the ratio of changed and unchanged red blood cells, the number of leukocytes and lymphocytes). The onset of

death in the period of 4-12 hours is distinguished by the appearance of 4 more significant signs in the damaged area - plasmatic saturation of the vessel walls, an increase in leukocytosis with the appearance of destroyed leukocytes, the identification of single macrophages and the identification of fibrin in the form of individual threads. With an increase in survival time to 13-24 hours, additional criteria for the duration of the process appear in the damage zone: on the part of the dura mater - the phenomenon of necrosis of collagen fibers; in the vascular walls against the background of increasing plasma impregnation, the phenomenon of fibrinoid necrosis; in the substrates of intracranial hematomas, clumps of free hemosiderin are detected, fibrin predominates in the form of a network. Upon death, 2-3 days after the injury in the area of damage in the dura mater, the phenomena of basophilia and eosinophilia of collagen fibers disappear against the background of increasing edema and necrosis. Fibrinoid necrosis predominates in the walls of blood vessels. In hematoma substrates, the number of unchanged and changed erythrocytes is almost equal; against the background of pronounced leukocytosis, the majority of neutrophilic leukocytes are in the stage of decay. The number of macrophages and free hemosiderin increases, fibrin mainly in the form of clots; the first fibroblasts appear. With death in the interval of 4-14 days, on the part of the dura mater, the phenomena of necrosis of collagen fibers and fibrinoid necrosis of vessel walls decrease, the formation of a connective tissue capsule occurs with an increase in the number of fibroblasts. In the substrates of intracranial hematomas, the number of degeneratively changed erythrocytes prevails over unchanged ones, and the number of leukocytes decreases. The number of macrophages increases, most of them with the presence of hemosiderin in the cytoplasm, the amount of free hemosiderin decreases. The number of lymphocytes increases to the maximum number. If the injury persists for more than 15 days, the lesion is characterized by a pronounced productive reaction with the obligatory formation of a connective tissue capsule with newly formed vessels and bleeding from them into the hematoma cavity, as a result of which the number of unchanged red blood cells increases again. There is a progressive decrease in leukocytes in varying degrees of decay, macrophages and lymphocytes, as well as a maximum increase in the number of fibroblasts, both in the dura mater and in the substrates of intracranial hematomas. Considering that biochemical reactions occur to a sufficient extent autonomously and can be recorded both in the initial stages after the occurrence of damage and at different stages of their development, it was proposed to use methemoglobin as a quantitative biochemical indicator of the evolution of the blood clot. Methemoglobin is a physiological component of the blood; it performs protective functions, neutralizing endogenously formed hydrocyanic acid, hydrogen sulfide and other poisons. Data on the concentration of methemoglobin in the blood of healthy adults are very contradictory; its value in the blood does not exceed 3 - 5% (in women it is higher than in men), on average it is about 1%. During life, methemoglobin is reduced to free hemoglobin under the action of enzymes contained in red blood cells.

A morphological study of the membranes and substance of the brain in acute TBI using specific stains for fibrin suggests that the severity of TBI, in addition to the direct impact of mechanical energy, is influenced by complex and diverse mechanisms of hemodynamic and hemorheological disorders, both local and generalized. The trigger for circulatory disorders is mechanical trauma to the brain, when damage to the membranes and substance of the brain is accompanied by a natural physiological reaction of vascular spasm. At the same time, activation of the coagulation and platelet hemostasis systems develops, compensated by the activation of the anticoagulant and fibrinolytic systems. Therefore, in all cases of acute TBI, discirculatory and dysgemic disorders of the central nervous system are accompanied by the development of disseminated intravascular coagulation syndrome with the formation of platelet-fibrin microcoagulations in the microcirculation system. As a result, secondary ischemic, hemorrhagic and necrotic phenomena develop in vital organs, including the brain, which affects the course and outcome of TBI. The main morphological substrate in the diagnosis of DIC, including its stages, is • the presence in the lumen of the microvasculature of microthrombi - having a fibrin framework or consisting entirely of fibrin. The formation of fibrin in the lumen of blood vessels occurs both as a result of isolated damage to the vascular wall and as a result of a generalized imbalance in the hemostasis system and has a number of clearly defined successive stages. At different stages of its development and regression, fibrin is able to perceive very specific (selective)

dyes. In this case, the color of fibrin, when using special staining methods, directly indicates its age. This technique is used to develop differential diagnostic criteria when establishing the duration of occurrence of TBI, which allows us to draw the following conclusions when summarizing the material: - in the first minutes after causing TBI, in the area of damage to the membranes and substance of the brain, exclusively discirculatory changes are noted, characterized predominantly by anemia of all structural areas bloodstream (arterial, capillary, venous) with symptoms of vasospasm and vascular collapse; focal disorders of hemorheology are observed in the form of plasma separation, focal plasma saturation of vessel walls, focal plasmorrhagia; focal edema and loosening of the soft meninges; damaged brain tissue looks like small cavities with an optically empty lumen; intact brain tissue without any changes, only focal degenerative changes in neurons are noted in the superficial parts of the cortex, the most sensitive to hypoxia; - when death occurs within a period of up to 1 hour after infliction of TBI, predominantly dyscirculatory changes are also noted, which, however, have different features in different areas: with persistent vasospasm in the zone of traumatic destruction, in undamaged areas uneven reactive plethora, dystonia and vascular paresis are noted ; hemorheological disorders develop in the form of stasis and sweetness of erythrocytes with plasma separation, plasma saturation of vessel walls and plasmorrhagia; in areas of brain damage, freely lying unchanged red blood cells are visible; in the molecular layer of the cerebral cortex - fine-mesh rarefaction, in other layers of the cortex - focal swelling of neurocytes with mild chromatolysis, focal swelling of neuroglial cells with single “honeycomb-like structures”; - when death occurs several hours after the injury (2-6), uneven blood filling of the vessels, vascular spasm is replaced by pronounced congestive venous plethora with pronounced hemorheological disorders (stasis, aggregation, sludge of erythrocytes in the microvasculature, phenomena of focal thrombosis of large and medium-caliber veins) . In areas of traumatic hemorrhage, the onset of lysis of red blood cells is noted with the destruction of the cytoplasm and their transformation into a homogeneous, sometimes granular oxyphilic mass, pronounced plasma separation, and the appearance of leukocyte infiltration of varying intensity at the periphery of the hemorrhage sites (which is especially noticeable in the pia mater). In connection with venous stagnation, the phenomena of tissue hypoxia are expressed to varying degrees - pronounced perivascular edema with the formation of “muff-like” cavities, hemo- and plasmorrhagia, interstitial edema, swelling and dystrophic changes in cellular elements with a weakly expressed tissue reaction in the form of microglial satellitetosis. There is pronounced fibrinoid swelling of fibrous structures in the walls of blood vessels and in the meninges with focal fibrinoid necrosis, with uneven lympho-leukocyte infiltration of varying intensity and initial symptoms of leukodiapedesis. When stained for fibrin using the OCG method, diffuse protein precipitation of the vascular wall is noted with a bright yellow staining, which contrasts especially clearly with the swollen, crimped blue-blue collagen fibers. In the lumen of the vessels and perivascularly, single or numerous clumps of “young” yellow-orange fibrin are found; - when death occurs within a period of time from 6 to 24 hours in areas of traumatic hemorrhages - leaching and lysis of erythrocytes with uneven separation of plasma and abundant uneven leukocyte infiltration; with loss of fibrin on the periphery in the form of threads or delicate mesh masses. The phenomena of congestive venous congestion are even more pronounced; in the lumen of vessels of different sizes, numerous blood clots of erythrocytes, platelets and a few leukocytes are detected, as well as diffuse fibrinoid swelling of the walls with focal necrosis and abundant leukocyte infiltration. In the brain substance there are severe discirculatory-dystrophic changes caused by tissue hypoxia, necrobiosis and foci of necrosis of neurocytes with microglial satellitetosis and initial phenomena of neuronophagia. In the cortex and white matter there is uneven swelling of neuroglial cells with the formation of “honeycomb-like structures”. In the white matter - swelling, focal tortuosity and fragmentation of axons, periaxial microglial satelitis. The formation of areas of necrosis begins near larger foci of traumatic destruction with secondary hemorrhages from unchanged red blood cells. When stained for fibrin using the OCG method, there is uneven thickening and loosening of the walls of blood vessels with diffuse yellow-orange staining. In some places, the thickened walls of the vessels have a reddish or red tint - fibrinoid necrosis. In the lumen of the vessels there are masses of aggregated ocher-yellow erythrocytes with phenomena of focal sludge and homogenization (while the destroyed erythrocytes retain a bright ocher-yellow color). Among the red

blood cells are lumps and fibrin threads of orange and reddish color; isolated fibrin aggregates completely obstruct the lumen of the capillaries like blood clots. The perivascular spaces are significantly expanded, optically empty or filled with unchanged red blood cells and homogeneous masses with a yellow-orange color of varying intensity. In foci of hemorrhage, fibrin is found both at the periphery and in the center of the foci in the form of delicate mesh-like masses of orange-red, sometimes violet-red color.

Literature.

1. Олимова А. З., Шодиев У. М. Репродуктив Ёшдаги эркакларда бепуштлик сабаблари: Бухоро тумани эпидемиологияси //Scientific progress. – 2021. – Т. 2. – №. 7. – С. 499-502.
2. Zokirovna O. A., Abdurasulovich S. B. Ovarian Diseases in Age of Reproductive Women: Dermoid Cyst //IJTIMOIY FANLARDA INNOVASIYA ONLAYN ILMIY JURNALI. – 2021. – Т. 1. – №. 6. – С. 154-161.
3. Olimova A. Z. ECHINOCOCCOSIS OF LIVER OF THREE MONTHLY WHITE RAT //Scientific progress. – 2022. – Т. 3. – №. 3. – С. 462-466.
4. Олимова А. З. Морфологические и морфометрические особенности печени белых беспородных трех месячных крыс после тяжёлой черепно-мозговой травмы вызванной экспериментальным путём //BARQARORLIK VA YETAKCHI TADQIQOTLAR ONLAYN ILMIY JURNALI. – 2021. – Т. 1. – №. 6. – С. 557-563.
5. Oglu M. Z. M., Zokirovna O. A. МОРФОЛОГИЧЕСКИЕ И МОРФОМЕТРИЧЕСКИЕ ПАРАМЕТРЫ ПЕЧЕНИ БЕЛЫХ БЕСПОРОДНЫХ КРЫС, ПЕРЕНЕСШИХ ЭКСПЕРИМЕНТАЛЬНУЮ ЧЕРЕПНО-МОЗГОВУЮ ТРАВМУ ПОСЛЕ МЕДИКАМЕНТОЗНОЙ КОРРЕКЦИИ //JOURNAL OF BIOMEDICINE AND PRACTICE. – 2023. – Т. 8. – №. 1.
6. Олимова А. З., Турдиев М. Р. БУХОРО ШАҲРИДА МЕЪДА ВА ЎН ИККИ БАРМОҚЛИ ИЧАК ЯРАСИ УЧРАШ ЭПИДЕМИОЛОГИЯСИ //Oriental renaissance: Innovative, educational, natural and social sciences. – 2022. – Т. 2. – №. 4. – С. 642-647.
7. Zokirovna O. A. Modern Concepts of Idiopathic Pulmonary Fibrosis //American Journal of Pediatric Medicine and Health Sciences. – 2023. – Т. 1. – №. 3. – С. 97-101.
8. Zokirovna O. A. Pathology of Precancerous Conditions of the Ovaries //American Journal of Pediatric Medicine and Health Sciences. – 2023. – Т. 1. – №. 3. – С. 93-96.
9. Зокировна, Олимова Азиза и Тешаев Шухрат Джумаевич. «Морфологические аспекты печени белых беспородных крыс после тяжелой черепно-мозговой травмы, вызванной экспериментально в виде дорожно-транспортного происшествия». *Scholastic: Journal of Natural and Medical Education* 2.2 (2023): 59-62.
10. Zokirovna O. A. Comparative characteristics of the morphological parameters of the liver at different periods of traumatic brain injury //Euro-Asia Conferences. – 2021. – С. 139-142.
11. Zokirovna O. A. Macroand microscopic structure of the liver of threemonthly white rats //Academic research in educational sciences. – 2021. – Т. 2. – №. 9. – С. 309-312.
12. Олимова А. З. Частота Встречаемости Миомы Матки У Женщин В Репродуктивном Возрасте //BARQARORLIK VA YETAKCHI TADQIQOTLAR ONLAYN ILMIY JURNALI. – 2021. – Т. 1. – №. 6. – С. 551-556.
13. Zokirovna O. A., Abdurasulovich S. B. Ovarian Diseases in Age of Reproductive Women: Dermoid Cyst //IJTIMOIY FANLARDA INNOVASIYA ONLAYN ILMIY JURNALI. – 2021. – Т. 1. – №. 6. – С. 154-161.
14. Zokirovna O. A. Cytological screening of cervical diseases: pap test research in the bukhara regional diagnostic center for the period 2015-2019. – 2022.

15. Zokirovna O. A., PREVALENCE R. M. M. EPIDEMIOLOGY OF CANCER OF THE ORAL CAVITY AND THROAT IN THE BUKHARA REGION //Web of Scientist: International Scientific Research Journal. – 2022. – Т. 3. – №. 11. – С. 545-550.

16. Olimova A. Z. The frequency of occurrence of my uterus In women of reproductive age //JOURNAL OF ADVANCED RESEARCH AND STABILITY (JARS). – 2021. – Т. 1. – №. 06. – С. 551-556.

17. Olimova Aziza Zokirovna. (2023). MODERN PRINCIPLES OF THE EFFECT OF HEMODIALYSIS THERAPY ON HEART RATE. *International Journal of Integrative and Modern Medicine*, *1*(1), 80–85. Retrieved from http://medicaljournals.eu/index.php/IJIMM/article/view/28

18. Olimova Aziza Zokirovna. (2023). PATHOMORPHOLOGICAL CHARACTERISTICS OF THE EPIDIDYMIS UNDER IRRADIATION. *International Journal of Integrative and Modern Medicine*, *1*(1), 96–100. Retrieved from http://medicaljournals.eu/index.php/IJIMM/article/view/31

19. Olimova Aziza Zokirovna. (2023). THE INCIDENCE OF CANCER OF THE ORAL CAVITY AND PHARYNX IN THE BUKHARA REGION. *International Journal of Integrative and Modern Medicine*, *1*(1), 86–89. Retrieved from http://medicaljournals.eu/index.php/IJIMM/article/view/29

20. Olimova Aziza Zokirovna. (2023). INFLUENCE OF ALCOHOL INTOXICATION ON THE HEART TISSUE OF RATS IN THE EXPERIMENT. *International Journal of Integrative and Modern Medicine*, *1*(1), 90–95. Retrieved from http://medicaljournals.eu/index.php/IJIMM/article/view/30

21. Olimova Aziza Zokirovna. (2023). Modern Aspects of the Etiology of Gastric Ulcer and Its Complications. *American Journal of Pediatric Medicine and Health Sciences (2993-2149)*, *1*(3), 163–166. Retrieved from http://grnjournal.us/index.php/AJPMHS/article/view/208

22. Zokirovna O. A., Jumaevich T. S. Morphological Aspects of the Liver of White Outbred Rats After Severe Traumatic Brain Injury Caused Experimentally in the Form of a Road Accident //Scholastic: Journal of Natural and Medical Education. – 2023. – Т. 2. – №. 2. – С. 59-62.

23. Aziza Zokirovna Olimova GASTRIC ULCER AND ITS COMPLICATIONS // Scientific progress. 2022. №3. URL: https://cyberleninka.ru/article/n/gastric-ulcer-and-its-complications (дата обращения: 28.09.2023).

24. Olimova Aziza Zokirovna. (2022). TECHNIQUE FOR CUTTING BIOPSY AND SURGICAL MATERIAL IN THE PRACTICE OF PATHOLOGICAL ANATOMY AND FORENSIC MEDICINE. *Web of Scientist: International Scientific Research Journal*, *3*(7), 116–120. https://doi.org/10.17605/OSF.IO/PSQ59

25. Zhumayevich N. F., Zokirovna O. A. PATHOMORPHOLOGY OF GASTRIC CANCER //BARQARORLIK VA YETAKCHI TADQIQOTLAR ONLAYN ILMIY JURNALI. – 2022. – С. 330-333.

26. Zokirovna O. A. Epidemiological and Etiological Data of Morphogenesis and Pathomorphology of Congenital Heart Diseases in Children //American Journal of Pediatric Medicine and Health Sciences. – 2023. – Т. 1. – №. 4. – С. 88-91.