SECONDARY TRAUMATIC FACTORS IN PATIENTS WITH DIFFUSE LESIONS OF THE BRAIN TISSUE IN JOINT HEAD INJURIES.

Mirzayuldashev N.Yu. Isakov B.M. Davlatov B.N. Mamadaliev A.B. Hakimov M.N.

Andijan State Medical Institute https://doi.org/10.5281/zenodo.7793626

Key words: spinal cord injuries, diffuse injuries of the brain tissue, secondary traumatic factors.

Relevance: now there is a constant growth rate of injury in all countries of the world. As a result of all the injuries that have arisen, the disability of the axiom, the central nervous system injuries among the causes leading to the death cases are in the first place (30-40%), and the mortality cases are lagging behind the indicators of cardiovascular and oncological diseases with much observation among the most active layer of axioms (18-45 years). [1,3,4,5].

In connection with the growing incidence of injuries, there is a rapid increase in the level and frequency of severity of brain injuries (V.V. Gebedev I soavt., 1981; 1995; 2001, A.Poppet hand., 1979; R. Spence et hand., 1990) and up to 50-70% of them are caused by external joint injuries of the skull. Bunda head brain tissue in patients with diffuse injuries secondary brain damage in addition to the factors that damage the head Brain Injury Clinic, had a serious impact on the course and outcome.

Objective of the study: to study secondary traumatic omillarni in patients treated with diffuse lesions of the brain tissue in the Departments of Neuroreanization and intensive therapy.

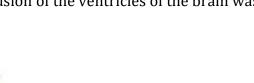
Research sources and methods: 193 out of 615 brain-affected patients who were hospitalized in 2015-2020 years in RSHTYOIM AF, diffuse lesions of the brain tissue were identified. The qualifying criteria for these patients are: a) those who lost consciousness after receiving shikasat; b) those who did not have a cranial hematoma and brain lat eating outbreaks requiring surgical intervention while on the basis of computer tomogram findings.

The age of the injured patients was from 16 to 76 years, the average age taken was 44.2 + -1.2 years. Diffuse damage to the brain tissue was observed in the younger period (21-45yosh atrophy) (46%).

Mainly in patients with diffuse lesions of the brain tissue, the cause of the lesion was YTX – 134 units (69%). The remaining patients (31 units) are injured as a result of the fall, while 214 cases constitute household traumas. According to observations, the mechanism of trauma in diffuse lesions of the brain tissue of the head was more: "acceleration-deceleration".

In 129 patients (67%) of the injuries, there were closed head injuries and 64 open head injuries (33%). Fracture of the Skull Bones was detected in 124 patients (including 32% cases of skull fracture and 39% cases of skull base fracture).

Subaroxnoidal blood transfusions of different levels were detected in 169~(87~%) injured patients. In 24 patients (13%), intracranial blood transfusion of the ventricles of the brain was detected.



In 193 (100%) patients, a brain injury came hamrox with various types of joint injuries. 51% of the patients, including those with damaged limbs and pelvic bones, 20% had chest injuries, 21% had spine and spinal cord injuries, and 4% had abdominal and abdominal overgrowth injuries.

The degree of occurrence of systemic traumatic factors in patients with diffuse brain tissue.

The degree of occurrence of systemic traditiatic factors in patients with diffuse brain tissu			
Бош миянинг	Текширув олиб	Шу омиллар	%
иккиламчи шикаст	борилган беморлар	аниқланган	
омиллари	сони	беморлар сони	
Артериал гипотензия (сАД<90 mmHg)	108	58	54
Гипоксемия (PaO2<60mmHg)	93	38	41
Гипернатриемия (Na>145 ммоль/л)	96	30	31
Гипонатриемия (Na<135 ммоль/л)	96	33	34
Яққол гипокапния (PaCO2>50mmHg)	61	22	36
Гиперкапния (PaCO2>50mmHg)	61	8	13
Гипертермия (T>38°C1 соат ва ундан кўп вақт давомида)	161	63	39

Results and muhokamalar: groups of patients with diffuse lesions of the brain tissue were structured and all investigated indicators of them were studied in dynamics. Indicators of Terminal case patients were not included in this study. Among patients with diffuse lesions of the brain tissue, more arterial hypotension and hypoxemia were observed. Cases of violations of sodium metabolism - hypernatremia and hyponatremia were the same. It should be noted that all secondary systemic injury factors were clearly observed in patients with diffuse lesions of the brain tissue of Type 3 and 4.

According to other researchers, these secondary systemic injury factors are also threeraydi more than the results of the group we are conducting our research. [9,10].

In all patients who have secondary cranial external trauma factors, the negative effects of musculoskeletal damage are increased to a different extent. Only if hyponatremia is observed,



INTERNATIONAL BULLETIN OF APPLIED SCIENCE AND TECHNOLOGY

 $UIF = 8.2 \mid SJIF = 5.955$

negative consequences increase, but these differences do not have a statistical significance. The presence of hypoxemia and hypotonia battery increased up to 93% of negative consequences 93% of negative consequences. The combined presence of hypotension and hypoxemia in only 7% of cases resulted in a good result.

In addition, the presence in the battery of hypotonia and hypoxemia led to the fact that all investigated patients were far away in the state of comatose. If these factors were not present, the comatose condition was observed to be short (1,7+-0,3). If any of the listed cases is added to this case, the periodicity of the comatose case increases to 7.2+1.1 Days (t=4.82; p<0.001). Violations of sodium metabolism and osmotic hemostasis were observed in patients with diffuse lesions of brain tissue of Type 3 and 4 (74% of cases), and in patients with diffuse lesions of brain tissue of Type 1 and 2, they were observed infrequently. In our observations, moderate normative violations of the sodium Exchange were observed, which made it possible to correct them. Hypernatremia and hyponatremia were observed at the same frequency, but the hyperosmolary condition had a much more negative effect than the hypoosmolary condition. If the priority of the hypertensive case is observed, the negative consequences will sharply increase to 93 %. In the case of hyponatremia, this negative outcome is 52% of cases. These data suggest that the most positive result of diffuse lesions of the brain tissue was observed in patients whose indicators of blood plasma were found in the stomach, and the negative consequences were no more than 40%.

The most positive okibates were observed when they were found in the normative indices of carbon dioxide. In obvious hypocapnia, however, negative consequences are manifested more often (77%). And in hypercapnia, these were 88%. With this, obvious hypocapnia and obvious hypercapnia are the most negative prognostic factors, which can be observed in patients with diffuse lesions of the brain tissue.

Hyperthermia in 3 and 4 groups of diffuse lesions of the brain tissue was observed in many cases. This is most often associated with complications of purulent inflammation, which develops on the one hand, and on the other hand, with "Central violations" of thermoregulation potentials in the cerebral cortex in patients with diffuse lesions of the brain tissue (3gurux), and mainly due to asymmetric enlargement of the cerebral cortex (4-group of diffuse lesions of the brain tissue). Hyperthermia without doubt significantly reduces the number of patients who can recover well and increases the number of patients with negative results. In addition, comatose, like the above damaging factors, increases the duration of the condition to 2 marotabs (up to 3,1+-0.5-7,9+-0.8 days; 1=5,09; p<0,01)

The conducted studies showed that among systemic lesions, the effect of hypotonia and hypoxemia on the degree and outcome of pronounced articulation of the brain in patients with diffuse lesions of the brain tissue was observed. The severity of the condition, the course of the disease and the duration of the comatose state in patients with diffuse lesions of the brain tissue, in which "minimal" changes are detected in the conclusions of the computer tomogram, may be associated with secondary lesions of the head brain (hypoxemia and hypotension). In this related case, primary brain tissue diffuse axonal lesions caused by traumatic Genesis may be observed in patients with this type of brain injury, as well as diffuse brain tissue diffuse lesions caused by systemic hypoxia/ischemia.[2,3,5].

In addition, it was found that the presence of hypotonia, hypoxia and hyperthermia in patients with diffuse damage to the brain tissue affects the periodicity of the comatose state. In these cases, the number of survivors with positive recovery is fundamentally reduced. Therefore,

IBAST | Volume 3, Issue 3, March

INTERNATIONAL BULLETIN OF APPLIED SCIENCE AND TECHNOLOGY

 $UIF = 8.2 \mid SJIF = 5.955$

IBAST ISSN: 2750-3402

the dynamics of the clinical condition of patients after a head injury, the expected results depend on the effectiveness of therapeutic measures aimed at eliminating secondary injuries and complications of the head brain. Conclusion.

- 1. Secondary systemic brain injury factors have a significant effect on the course and outcome of the disease in patients with diffuse lesions of the brain tissue, including in patients with mild trauma (according to the conclusions of the computer tomogram 1 and 2 group of patients with diffuse lesions of the brain tissue).
- 2. The legality found is that further research is aimed at investigating these systemic damaging ome and relative non-severe injuries, in which cases secondary traumatic factors are the cause of disability and death cases.

References:

- 1. Potapov A.A., Bregina N.N., Amcheslavsky V.G. I dr..// VnedreniesovremennixrecomendationiOtsenka IX vlyaniyanatactikuirezultatilecheniyatyajeloycherepno--Mozgovoy OtchetnydokladnasessiiuchenogoSoveta NII neurosurgiiim. N.N.Burdenko RAMN.- Moscow.-2002.
- 2.Cherepno-mozgovaya trauma. Klinicheskoerukovodstvo. Pad Red. eked.A.N. Konovalova, prof.Liechtenstein, prof. A.A. Potapova.- Moscow.-2001.
- 3. Liechtenstein L.B., **Potapov** A.A. Classification cherepno-Mozgovoy Klinicheskoerukovodstvopocherepno-Mozgovoy trauma. Pod redaktsieyKonovalova A.N., Liechtenstein L.B., Potapova A.A.- Moscow.- The antidote.-1998.- what?1.
- 4. Zimmerman R. Craniocerebral trauma. McCraw-Hill.-1999.
- 5. Orison W.W. Neuroimaging and neurotrauma. WB company. 2000.