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SEVERE TRAUMATIC BRAIN INJURY WITH INTRACRANIAL HEMATOMAS

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Abstract.

The article is devoted to the experience of treating severe traumatic brain injury with intracranial hematomas for the period from 2019 to 2021 in the Andijan branch of the Republican Scientific Center for Emergency Medical Care. 101 patients underwent a comprehensive examination and treatment.

Key words: severe traumatic brain injury, hematoma, treatment.

BOSH MIYA OG'IR JAROXATLARIDA KALLA SUYAGI ICHI GEMATOMALARI

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Annotatsiya.

Maqola Respublika shoshilinch tez tibbiy yordam ilmiy markazi Andijon filialida 2019 – 2021 yillarda og'ir bosh miya jaroxatlari intrakranial gematomalarni davolash tajribasiga bag'ishlangan. 101 nafar bemor kompleks tekshiruv va davolash muolajalarini olgan.

Kalit so'zlar: bosh miya og'ir jaroxati, gematoma, davolash.

ТЯЖЁЛЫЕ ЧЕРЕПНО-МОЗГОВЫЕ ТРАВМЫ С ВНУТРИЧЕРЕПНЫМИ ГЕМАТОМАМИ

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Аннотация.

Статья посвящена опыту лечения тяжёлой черепно-мозговой травмы с внутричерепными гематомами за период с 2019 по 2021 г. в Андижанском филиале республиканского научного центра экстренной медицинской помощи. У 101 пациента проведено комплексное обследование и лечение.

Ключевые слова: тяжёлая черепно-мозговая травма, гематома, лечение.

Relevance: Intracranial hematomas are one of the main causes of death in severe traumatic brain injuries. These formidable complications require urgent measures to eliminate them.

Material and methods: the study is based on the analysis of 101 patients, for the period from 2019 to 2021, who underwent a comprehensive examination and treatment at the Andijan branch of the Republican Scientific Center for Emergency Medical Care (AFRSCEMC). This analysis included patients who underwent a full dynamic MSCT study, starting from the first day after injury. Statistical analysis was carried out using the main clinical, MSCT and MRI data (the volume of intracranial hematoma, the severity and extent of cerebral edema, the degree of displacement of the transparent septum). The patients were divided into three groups depending on the cause of brain compression: the first group included 46 victims with cerebral compression by intracerebral hematomas; the second group - 34 victims with compression of the brain by meningeal hematomas, which were accompanied by non-rough parenchymal lesions; the third group - 21 victims with compression of the brain by meningeal hematomas, which were accompanied by severe parenchymal lesions. With a volume of less than 30 ml, all patients were treated conservatively, and with a volume of more than 40 ml, only surgical treatment was carried out. In the case of the volume of the bruised focus from 30 to 40 ml, both surgical and conservative treatment was used.

Results: In intracerebral hematomas, autotrauma was the most common cause of injury (in one third of the victims). Their average age was the highest. The severity of their condition did not differ significantly from the severity of the condition of patients in the second group (t=l.6; p>0.05). The average volume of hematomas and the displacement of median structures were the smallest in this group of patients.

The second group was characterized by the youngest age and less severe condition of patients upon admission. The period of unconsciousness was shorter. A detailed examination of the cause of traumatic brain injury in this group failed to reveal the predominance of any mechanism of injury. The mean volume of meningeal hematomas was twice that of intracerebral hematomas. The displacement of the median structures is more pronounced - 3.6 mm.

The third group was characterized by a more severe condition, the duration of the unconscious state was the greatest with this type of brain compression. The severity of the injury in this group of patients was also

indicated by the maximum total volume of intracranial hematomas, and, accordingly, the maximum displacement of the median structures.

An analysis of the dynamics of depression of the level of consciousness revealed that in one third of patients, an unconscious state occurred after a "lucid interval". Most often, a light gap was observed among patients of the second and third groups (42%). At the same time, it was the most typical for shell hematomas. Along with the lucid interval, the main neurological symptoms were analyzed separately in all patients who had a lucid interval, regardless of the etiology of brain compression.

Clinical and computed tomography comparisons revealed a close relationship between coma duration and outcomes for all patients with brain compression ($r=0.6$; $p<0.01$). Outcomes in these patients significantly correlated with the severity of the condition at admission ($r=0.5$; $p<0.01$), the volume of intracranial hematoma ($r=0.3$; $p<0.05$), the severity of brain swelling ($r=0, 4$; $p<0.01$), the degree of compression of the base cisterns ($r=0.3$; $p<0.05$), the degree of displacement of the median structures ($r=0.2$; $p<0.05$). When determining the same patterns in patients with a light interval, strong correlations of outcomes with the volume of hematoma, the severity of cerebral edema, the degree of displacement of the median structures and the degree of compression of the base cisterns were revealed ($p<0.05$). In patients who lost consciousness immediately after injury, these correlations were weakly expressed and not significant ($p>0.05$). This indicates that in patients with a light interval, in whom the primary injury was less severe, the further course of the disease depends on the development of secondary damaging factors of the brain - an increase in the volume of the hematoma, an increase in cerebral edema and, accordingly, an increase in brain displacement.

Patterns of brain compression in patients of different ages differed. Based on the correlation analysis, it was found that the outcomes in patients under 40 years of age ($n=146$) significantly depended on the volume of the compressive substrate ($r=0.3$; $p<0.01$), the prevalence of cerebral edema ($r=0.5$; $p <0.01$), degree of compression of the ventricular system ($r=0.3$; $p<0.01$) and base cisterns ($r=0.3$; $P<0.01$). Whereas at the age of over 40 years ($n=56$), the outcomes of injury did not correlate with these indicators. This indicates that in patients under 40 years of age, there are more rigid volumetric ratios in the cranial cavity, the violation of which, due to additional volume and compression of the brain, has a more pronounced effect on the outcome of injury.

The severity of the injury, which was assessed by the Glasgow Coma Scale at admission, had a significant effect on outcomes. Outcomes in patients admitted in a soporous or comatose state (n=124) correlated only with the severity of concomitant cerebral edema (r=0.3; p<0.01). Outcomes in patients admitted in a state of stunning (n=72) correlated with the volume of intracranial hematoma (r=0.3; p<0.01) and the severity of cerebral edema (r=0.3; p<0.01). Therefore, in stunned patients, hematoma volume and associated cerebral edema have the most important prognostic value.

A high degree of correlation was found between the duration of the coma and outcomes for all patients. In patients with a coma duration of up to 3 days inclusive (n=120), the outcomes of injury correlated with the volume of intracranial hematomas (r=0.2; p<0.05), the prevalence of cerebral edema (r=0.3; p<0.01). In patients with a coma duration of more than 3 days (n=76), outcomes correlated only with the prevalence of concomitant cerebral edema (r=0.3; p<0.01). The data obtained indicate that in the case of a rapid exit from the unconscious state, the outcomes are closely related to the volume of the hematoma. However, if decompensation has occurred and the coma continues for more than 3 days, then the outcomes already depend on the severity of secondary damaging ones.

The volume of intracranial hematoma has a significant effect on outcomes in the entire group of patients. With an intracranial hematoma volume over 30 mm3 (n=109), the outcomes depended on the severity of concomitant cerebral edema (r=0.3; p<0.01), on the degree of compression of the ventricular system (r=0.2; p<0.05) , base cisterns (r=0.2; p<0.05), massive blood loss. With a volume of up to 30 mm3 (n=87), the outcomes did not depend on these indicators.

The prevalence of cerebral edema was significantly correlated with outcomes in the whole group. When edema spread perifocally or within one lobe (n=122), the outcomes were closely related to the volume of intracranial mass (r=0.2; p<0.05). In the case of edema spreading to one hemisphere or its generalization (n=74), there was no correlation between computed tomography parameters and outcomes.

Conclusions: with an intracranial hematoma up to 30 ml, the existing compensatory possibilities of the CSF spaces have not yet been exhausted, and such indicators as displacement, compression of the ventricles and cisterns of the base do not correlate with outcomes. With a hematoma volume of more than 30 ml, the compensatory possibilities of the cerebrospinal fluid

spaces are depleted, and the degree of compression of the ventricular system and base cisterns affects the outcomes. When cerebral edema spreads perifocally or within a lobe of the brain, the volume of intracranial hematoma affects the outcomes, however, in the case of generalized cerebral edema, the volume of the hematoma, the degree of compression of the ventricular system and base cisterns do not affect the outcome of injury.

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