ORIGINAL ARTICLE

CONTENTS

Clinical-and-neurological symptoms and dynamics of certain biomarkers in the acute and remote periods of combat traumatic brain injury

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ABSTRACT

Aim: To study biochemical markers of brain tissue damage, the levels of which could be used to assess the degree of damage to the nervous tissue and predict the course and outcome of the disease.

Materials and Methods: There were 38 patients in the acute period and 33 in the remote period of MBT were examined. The patients' complaints, levels of brain-specific proteins S-100β, BDNF and inflammatory biomarkers such as interleukins IL-8 and IL-10 were studied in detail.

Results: The study of complaints showed their similarity in both the acute and remote periods, apart from an increase in the number of patients with meteorological sensitivity. An increase in S-100β levels and a decrease in BDNF levels indicate a slowdown in the rate of recovery processes in this category of patients and a deterioration in brain plasticity processes. An increase in IL-8 and IL-10 levels indicates further progression of the disease.

Conclusions: The study of these biomarkers contributes to the prognosis and course of the consequences of mine-blast trauma and the choice of therapeutic agents in treatment.

KEYWORDS: mine-blast trauma, brain-specific interleukin proteins

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INTRODUCTION

Mine-blast trauma (MBT) is the main cause of disorders in the central nervous system (CNS) and contributes to further structural, functional and neurochemical disorders in the brain. Traumatic damage to the CNS ranks second in frequency after bullet and shrapnel wounds to the torso and limbs in the structure of combat injuries. In MBT, in addition to the direct impact of the high-pressure zone on the brain, the corresponding receptors of the torso and auditory apparatus are affected by high temperatures and extremely loud sounds. As a result, an extremely powerful flow of afferent impulses is formed in the CNS, leading to the destruction of the entire structural and functional organisation of the brain as the highest integrative control centre of the entire body. Extreme inhibition of brain structure activity causes desynchronisation and imbalance in their activity, i.e. dysregulatory pathology, which impairs the functioning of the brain's functional systems [1].

At the same time, autoimmune processes, a significant increase in antibody production, and an increase in humoral immunity indicators play a significant role in the pathogenesis of MBT.

Post-traumatic changes are a combination of compensatory-adaptive and established pathological processes, which can subsequently develop independently and determine the patient's condition and the course of the disease.

It should be noted that damage to brain tissue in MBT leads to metabolic activation of glial neurons, as well as to energy deficiency and membrane depolarization. Ultimately, the chain of pathological reactions leads to cytotoxic edema and osmotic lysis of cells and, on the other hand, to the initiation of secondary biochemical reactions, among which apoptosis and inflammation play a major role [5].

The diagnosis of mild MBT poses certain difficulties due to the barely noticeable subjective nature of the symptoms. Traditional neuroimaging methods often do not reveal abnormalities in this pathology, which has led to growing interest in various biomarkers that can be objective indicators of brain injury.

These include the S-100β protein, which is found mainly in astrocytes, and the BDNF protein, which is found in the hippocampus, basal parts of the forebrain, as well as fibroblasts, astrocytes, and various types of neurons, and belongs to the neurotrophins, which stimulate and support the development of neurons. Its deficiency reduces the plasticity of neurons and disrupts cognitive functions [2, 4, 6, 7].

The cytokine imbalance that occurs in the acute period of traumatic brain injury persists and maintains a chronic inflammatory process and contributes to the progression of brain damage.

One of such cytokines is interleukins 8 and 10 (IL-8, IL-10). It has been found that IL-8 is released into the cerebrospinal fluid after a brain injury, and its role in the pathophysiology of traumatic brain injury (TBI) may be linked to blood brain barrier dysfunction.

The role of IL-10 in the pathophysiology of TBI is controversial in the literature, although its increase in 6 months period after TBI may indicate long-term chronic inflammation [3].

Damage to the nervous system, as a result of MBT further leads to trophic dysregulation, which is one of the universal components of pathogenesis. Therefore, the study of biochemical markers of brain tissue damage, the levels of which could be used to assess the degree of damage to the nervous tissue and predict the course and outcome of the disease, is protein S100β, a specific protein, the level of which could be used to assess the degree of damage to the nervous substance and predict the course and outcome of the disease, is protein S100β, a specific protein synthesized mainly in astroglial cells (more than 85-90% of their total content in the nervous system). The content of S100β protein in blood serum ranges from 0.005 to 0.105 μg/l [7].

AIM

To study biochemical markers of brain tissue damage, the levels of which could be used to assess the degree of damage to the nervous tissue and predict the course and outcome of the disease.

MATERIALS AND METHODS

There were 38 patients in the acute period and 33 patients in the remote period of mild mine-blast traumatic brain injury who were hospitalised at the neurosurgical clinic of the State Institution 'P.V. Voloshin Institute of Neurology, Psychiatry and Narcology of the National Academy of Medical Sciences of Ukraine' (Kharkiv, Ukraine) during the period of 2022–2025. The control group consisted of men between the ages of 27 and 36 with no history of traumatic brain injury, neuroinfections, somatic pathology, or hereditary diseases.

The study included patients who met the following criteria: a) age between 25 and 37; b) no history of somatic or neurological disorders; c) only a mild traumatic brain injury (with no alcohol poisoning, chest injuries, tuberculosis at the time of injury, and other somatic and mental disorders). These criteria allowed us to exclude factors that could significantly affect the course of the disease. The complaints and neurological status of these patients were studied in detail.

To study the degree of brain damage based on the level of biochemical markers that could be used to assess the degree of brain damage and predict the further course of the disease, we studied the indicators of such biomarkers as S-100β protein, BDNF, and interleukins IL-8 and IL-10.

The level of S-100β protein was studied in blood serum using electrochemiluminescence analysis on an Elecsys 1010 automatic analyzer (Germany), and BDNF was studied using the ELISA immunoassay method with the Human BDNF ELISA Kit in accordance with the manufacturer's instructions. The content of pro-inflammatory cytokines – interleukins (IL-8, IL-10) in blood plasma was determined by immune-enzymatic method using standard kits from "Calbiotech" (Germany) and "Diaclone" (France). Statistical analysis of the results was performed using standard statistical methods with the "Statistika-6.0" and "Microsoft Excel-2010" statistical software packages. The reliability of the results was assessed using Student's t-test. A condition of $p < 0.05$ was considered statistically significant.

ETHICS

This work complies with the principles of the Declaration of Helsinki.

RESULTS

The duration of mine-blast cerebral trauma at the time of admission to the hospital ranged from 3 to 7 days. These patients were referred for hospitalization with a diagnosis of acute mine-blast trauma, which was diagnosed by neurologists at a military hospital. At the time of injury, 33 of the 38 patients examined lost consciousness (for 5 to 20 minutes). These patients were re-examined and treated in 10–12 months after the injury.

The main complaints in the acute period were constant headache, dizziness, a hearing impairment, memory loss of current events, 'sleep-wake' cycle

Table 1. Main complaints in the acute and long-term periods of mine-blast trauma upon admission to the hospital

Complaints	the acute period MBT (n=38)	the remote period MBT (n=33)	p
headache	36	28	< 0.05
dizziness	32	27	< 0.05
nausea	19	11	
hearing impairment	30	26	< 0.05
memory loss	31	32	< 0.05
sleep disorders	35	26	< 0.05
fluctuations in BP	13	18	
Meteorological sensitivity	3	17	
irritability	34	32	< 0.05

Source: compiled by the authors of this study

Table 2. Indicators of brain-specific proteins in the acute and remote periods in patients with mine-blast trauma

Brain-specific proteins	control indicators	the acute period (n=38)	the remote period (n=33)
S-100β	54.1 ± 1.88ng/l	128.3 ± 6 ng/l	97.2 ± 6.4 ng/l
BDNF	459 ± 21.4 ng/l	388 ± 10.2 ng/l	231± 24.6ng/l

Source: compiled by the authors of this study

Table 3. Data on biomarkers of neuro-inflammation interleukin 8 and interleukin 10 in patients in the acute and remote periods of mine-blast trauma

control values IL-8, IL-10 (0-10 pg/mg)		the acute period of MBT (n=38)		the remote period of MBT (n=33)	
IL - 8	IL - 10	IL - 8	IL - 10	IL - 8	IL - 10
0 - 10		2	-	-	-
11 - 20		7	9	5	4
21- 30		24	16	12	27
31 - 36		5	13	16	2
Total		38	38	33	33

Source: compiled by the authors of this study

disorders, and rapid fatigue. In the remote period, the main complaints were headache, nausea, hearing impairment, noise in the head, meteorological sensitivity, fluctuations in blood pressure (BP), memory loss, daytime sleepiness, irritability, sleep disorders and daytime sleepiness (Table 1).

As can be seen from Table 1, headaches were observed in the acute period of MBT in almost all examined patients (in 36 out of 38 men, $p < 0.05$). A similar picture was observed in patients with the long-term consequences of MBT (in 28 out of 33 men, $p < 0.05$). Slightly fewer patients complained of nausea (19 in the acute period and 11 in the long-term period), while the number of patients with meteorological sensitivity increased (5 in the acute period and 17 in the long-term period). In the rest of the complaints, everything was practically the same in both the acute and remote periods of mine-blast trauma.

A study of the neuroglial protein S-100β showed (Table 2) that it increased almost threefold in the acute period compared to the control group and decreased slightly in the remote period, which may indicate the onset of secondary auto-destructive biochemical processes and the onset of traumatic brain disease. The levels of the brain-derived neurotrophic factor BDNF in the acute period were reduced compared to the control figures, and an even greater decrease was observed in the remote periods of MBT, which indicates a slowdown in the rate of recovery processes in this category of patients and deterioration in brain plasticity processes.

The study of biomarkers of inflammation of such as interleukins 8 and 10 showed that there is an increase in these indicators in the acute and long-term effects of mine-blast trauma (Table 3).

As we can see from Table 3, the highest neuro-inflammation indicators in the acute period of MBT were within the range of 21-30 pg/mg for both IL-8 and IL-10 (in 24 and 16 patients, respectively). In the long-term period of MBT, high levels were within the range of 31-36 (IL-8) in 16 patients and within the range of 21-30pg/mg (IL-10) in 27 patients.

DISCUSSION

At the moment of receiving a mine-blast injury, a series of mechanisms are triggered at both the cellular and molecular levels, leading to a disorder of physiological processes, which subsequently negatively affects the neural system and brain function. Pro-inflammatory factors, such as interleukins and tumour necrosis factor, are activated in response to damage and can exacerbate the negative effects of inflammation, contributing to further tissue damage.

Brain-specific proteins are key components in the pathogenesis and further recovery after cerebral trauma. Studying them helps to understand the mechanisms of trauma, develop therapeutic approaches, and assess the prognosis after trauma.

At the same time, MBT leads to the release of biomolecules into the extracellular space and further into the blood. It is the release of proteins and metabolites into the blood that is a molecular marker of the severity of injury progression (S-100β, BDNF, and others) [6].

The main complaints we received from patients with MBT, both in the acute and remote periods, indicate the presence and persistence in most of them of complaints such as headache, dizziness, hearing and memory impairment, sleep disorders, increased irritability and meteorological sensitivity (which were not observed in patients before the injury). All this indicates that pathological processes continue and cause persistent disorders in the central nervous system.

The increase in S-100β neuroglial protein levels compared to control figures after 9–12 months can be explained by the onset of secondary auto-destructive biochemical processes, indicating the onset of traumatic brain disease and requiring specialized treatment at this stage.

A decrease in the levels of the brain-derived neurotrophic factor BDNF, both in the acute and even more in the long-term period of MBT, is one of the reasons for the slowdown in the rate of reparative processes and brain plasticity that occur in these patients.

Data on the levels of neuro-inflammation biomarkers interleukin 8 and interleukin 10 in patients in the acute and long-term periods of mine-blast trauma showed an increase in levels from 21 to 36pg/mg. The study of these biomarkers is crucial for improving our understanding of the pathophysiology of combat traumatic brain injury and, accordingly, for the diagnosis or prediction of recovery and early outcomes of MBT [7].

CONCLUSIONS

1. In patients in both the acute and remote periods of MBT, the same set of complaints persists, indicating a disorder of the body's compensatory and adaptive mechanisms.
2. An increase in the levels of S-100β protein indicates the onset of secondary auto-destructive biochemical processes, which indicates the onset of traumatic brain injury.
3. A decrease in the levels of the brain-derived neurotrophic factor BDNF is one of the reasons for the slowdown in the rate of reparative processes in individuals who have suffered MBT, since BDNF plays an adaptive role in the formation of brain plasticity.
4. A significant increase in IL-8 and IL-10 levels indicates that chronic inflammation, which is activated after cerebral trauma, leads to neuro-inflammation. These biomarkers can be used not only for diagnosis or prognosis of recovery, but also for long-term outcomes of MBT.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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