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# Neuropeptides as Markers of Brain Damage

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Research on biomarkers for diagnosing various brain lesions has been ongoing for more than 25 years, but at present, the ideal biomarker has not been found. Among biochemical markers, the determination of the level of neurospecific proteins is being actively studied. Given the large number of neuropeptides that are currently being studied, the purpose of our work is to consider only some of them, which, in our opinion, are of greatest interest. It is protein S100β, neuron-specific enolase , myelin basic protein, brain-derived neurotrophic factor. They are being actively studied to identify, determine the prognosis and severity of strokes [1; 2], traumatic brain injury (TBI) [3-5], chronic cerebral ischemia (CCI) [6], brain tumors [7], cognitive impairment in diabetes mellitus [8], cognitive impairment in neurodegenerative diseases [9 ], epilepsy [10], perinatal lesions of the nervous system [11; 12], including for postoperative cerebral complications [13; 14, 15].

It is assumed that the ideal marker of brain damage should have the following characteristics:

(1) high specificity for damage to brain matter;

(2) high sensitivity;

(3) is released exclusively in cases of irreversible damage to cerebral neurons and should provide information about the nature of damage to the brain substance;

(4) is found in the blood or cerebrospinal fluid within a short period of time following the injury and correlates with its severity;

(5) is released at a well-known time following injury. Moreover, he must be

(6) age - and gender independent ;

(7) readily detectable in blood because frequent CSF sampling is impractical;

(8) the concentration should be easily measurable by laboratory tests and simple procedures;

(9) reflect the dynamics of the disease and the effectiveness of treatment [13; 15; 16].

Among biochemical markers, the determination of the level of neurospecific proteins is being actively explored. Most of them are autoantigens , getting into the bloodstream, they can cause the appearance of autoantibodies , which, if the function of the blood - brain barrier is impaired, from the blood vessel enter the brain and cause morphological changes, destructive processes in neurons, as well as the development of nonspecific acute -phase reactions such as edema or inflammation [13; 16].

*The S100B protein* is a glial biomarker , the most studied and included in laboratory diagnostics due to its neurospecificity . This protein was first isolated by B. Moor in 1965. It mainly contains glutamic and aspartic acids, phenylalanine , and a small amount of tryptophan, tyrosine, and proline [2]. These proteins are low molecular weight Ca -binding proteins up to 21 kDa and have three known subtypes consisting of α- and β-chains. Various combinations of subunits divide the S100 family into homodimeric (α-α, β-β) and heterodimeric (α–β) forms. Protein S100β has a molecular weight of 10-12 kDa and consists of β-β, α-β forms [15]. It is found in the cytoplasm of astrocytes , Schwann cells, adipocytes , chondrocytes , melanocytes . Given that this protein is widely present in cells of various types, it is presumably considered a marker of generalized damage to the blood-brain barrier, rather than isolated damage to glia [16]. At low concentrations, S100β exhibits neuroprotective properties by blocking NMDA receptors and acting as a growth and differentiation factor for neurons and glia . And at a high concentration, it triggers the synthesis of pro-inflammatory cytokines and leads to neuronal apoptosis [1].

The S100 protein is the most studied and is often used as a marker of brain damage in various studies. Thus, its increase is noted in strokes [1; 2], TBI [4; 5; 16], CHIM [6], diabetes mellitus [8; 9], brain tumors [7], perinatal injuries of the nervous system [11; 12].

This protein is actively used to analyze brain damage during various operations, including coronary artery bypass grafting. The content of S100 protein in blood serum is normally less than 0.2 μg/L. The development of cerebral complications in the patient in the postoperative period is evidenced by its content of more than 0.5 μg/l [18]. It is also noted that the lowest concentration of S100 in the blood serum is observed immediately

before induction into anesthesia and increases significantly during EC, reaching maximum values by the end of it, and then decreases on the first day of surgery [14]. According to some reports, the level of this peptide returns to its original value as early as 18 hours after surgery [19].

Also, S100β is used to evaluate the neuroprotective properties of anesthetics along with neuropsychological testing in studies evaluating the effect of general anesthetics on cognitive impairment in the postoperative period of CABG. So, when comparing anesthesia with propofol and sevoflurane , a smaller increase in the concentration of S100β was observed in the 2nd group, both in the first hours and in the subsequent ones. The same patients showed the best neurodynamic parameters. Comparison of these data with indicators of cerebral oxygenation and the study of the cognitive status of patients made it possible to draw conclusions about the better neuroprotective properties of sevoflurane compared to propofol [14]. There are also similar studies of desflurane and propofol and other anesthetics [20; 21].

*Neuron-specific enolase (NSE)* is a glycolytic enzyme 2-phospho-D-glycerate hydrolase, which belongs to the enolase family and is involved in the last stage of glycolysis - catalyzes the conversion of 2-phospho-O-glyceric acid to 2-phosphoenolpyruvate [24; 25]. This enzyme was first isolated in the 1970s-1980s. [26; 27]. It has a molecular weight of 78 kDa , a half-life of 24 h, and exists in various dimer variants , consisting of three subunits: α, β, γ. At the same time, the α-subunit of enolase is secreted in various tissues, the β-subunit is found only in the heart and striated muscles. For neurons, the specific isoform is α-γ, γ-γ, and they are called NSE or γ -enolase . They were initially found in high concentrations in neurons and endocrine cells, as well as in tumors derived from these cells [3; 28].

Determining the level of NSE in ischemic damage to the CNS makes it possible to judge the severity of neuronal damage and impaired membrane function of the blood-brain barrier [3; eleven; 27]. Currently, this marker is used to diagnose acute conditions characterized by cerebral ischemia and cerebral hypoxia, as well as to study the pathogenesis of neurological diseases. Its significance has been proven in various pathologies of the nervous system, such as epilepsy [10], Parkinson's disease [27], senile dementia, Alzheimer's disease [27, 28, 29], perinatal brain damage [11; 25], primary hypothyroidism [26], brain tumors [7], TBI [4; 5].

The study of neurospecific proteins makes it possible to judge the nature of brain damage in trauma, cerebrovascular accidents, diseases of the nervous system, and after surgical interventions. Thus, an increase in S100 reflects damage to glial cells, NSE neurons, and MBP oligodendrocytes .

Despite the fact that ideal markers of brain damage have not yet been found, an increase in the content of neurospecific proteins in blood and other biological fluids can be associated with signs of early neurological disorders, the extent of brain damage, early clinical deterioration, and prognostic signs of disease outcome. Although the determination of individual neuropeptides may not have sufficient diagnostic significance necessary for an accurate diagnosis of brain damage, however, the simultaneous determination of several markers is diagnostically significant.

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