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Biochemical Markers in The Diagnosis and Prognosis of Cerebral Stroke

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Abstract
acute stroke is one of the serious problems of modern medicine and Neurology. The death or disability of patients caused by a cerebral stroke has a negative impact on healthy morals and the state economy. According to the World Federation of neurologists (WFN), 14-16 million people around the world are registered with acute cerebral stroke every year, of which 5 million are fatal, 5mln patients are registered for disability. In Russia, 400-450000 people a year are sick with a stroke, which in every 1.5 minutes one patient is sick with a stroke.

Keywords: acute stroke, blood vessels, Matrix metaloprotease, nerve tissue.

Introduction

The search for new diagnostic and treatment methods for cerebral stroke, which lead to a decrease in medical and social complications of the disease, is one of the priorities of modern Neurology. In the diagnosis of cerebral stroke, MRI, CT and various clinical diagnostic methods are taken as a basis. In recent times, the diagnosis of various pathological conditions has been focused on the use of laboratory methods, that is, it is introduced as a new approach in the study of the pathogenesis of the disease. Ways of metabolism in Haki, the expansion of concepts and biomarkers involved in pathological processes lead to an increase in the amount of therapeutic targets and the opening of new doors in the future in medicine. According to the data of modern literature, many laboratory tests with a short diagnostic and prognostic purpose, as well as the priorities of clinical laboratory diagnostics, lead to an increase in their analytical reliability, as well as newly developed or implemented biochemical markers – biological active proteins. They perform a special function for the nervous system [1,2,3,4].

Markers in the human body are classified according to localization or structure: neuronal, glial, membrane associated, cytoplasmic. Also divided into markers found in the norm or pathology. Biomarkers found in cerebral stroke include S100 protein, neuronal specific enolase (NSE), NMDA receptor oxide or antibodies, basic myelin protein (MBP), s reactive iqsil (SRP), interleukin 6 (IL6), Matrix metalloprotease (MMR2 and MMR9), and X. The production of an endothelial growth factor in the vascular endothelium increases when the brain infartis lacks oxygen to the tissue. In response to the activated growth factor, the ischemized nerve tissue increases the

transport of nutrients and oxygen, that is, the content of small-caliber blood vessels, which is a new dressing. According to the results of studies carried out, the growth factor activates the process of neurogenesis and microgenesis, in acute ischemic stroke, the neurons and glia of the brain are chemically worn. The growth factor is the predictor of cerebral radiotherapy [1,2,3,4,6,7].

Matrix metaloprotease is a proteinase in addition to the family cell, with the exception of MMR – 2 and MMR–9 cells, the Matrix breaks down all types of proteins, plays Mukhim rol in cell remodeling, is involved in angiogenesis, proliferation, cell migration and differentiation, MMR–2 tissue development and regeneration vaccine, has a mesenchymal cell dressing, as well as neutrophil, macrophil and from monocytes, the dressing blurs. MMP-9 is a proteolytic enzyme that is activated IL-6 and TNF-α in the treatment of ischemic stroke or predicted for transmural hemorrhagic stroke that is lysobbed from muxime markers [1,2,3,7,8,9].

The enzyme MMR is a bulib in the zinc and calcium-binding endopeptidase family, primarily companentin of the extratcellular Matrix. It has the property that the enzyme burns: it is involved in the remodeling of the extratcellurar matrix, is involved in the breakdown of collagen and elastin, fibrinectin, glucosaminglicans, is secreted like a proferment, and needs proteolytic degradation for its activation. MMR is synthesized in a physiological state in a small amount of microns, secreted under the influence of cytokines. MMR dressing blurs in cup micdor in the involution of fat cells, T lymphocytes and macrophages against the background of constant flaking. MMR-9 develops cells of inflammation and is mobilized to damage 28 arteries. The maximum activity of MMR is anicized in the areas of inflammation in cardiovascular diseases, that is, with the rupture of plaques and the development of Atherothrombosis, participation in the process of bond remodeling serves as the basis of Haki. The enzymes hydrolyze the gelatins and are known as gelatinase. Ikala gelatinase (MMR-2 I MMR-9.) collagen changes to type IV, which is located in the basal membrane of the arteries. As well as the attachment of a series of skull collagens and skull oxyls to matrices, including elastin dressing blurs. Gelatinose A and V (MMR-2 I MMR-9.) actively participates in the process of destructure of the ciliary muscle and collagen structure of the cardiac vascular system, that is, an increase in the activity of MMR-9 is a direct connection with the violation of the integrity of the cardiovascular system and the development of acute coronary syndrome .

currently, despite the pathophysiological mechanism of development of cerebral infarction and the occurrence of risk factors, the necessary methods in diagnosing the disease are not always Ham effective and also the development and implementation of new modern diagnostic methods for predicting the severity of cerebral stroke remains an actual task. As a result, we should pay attention to practical neurology only for the purpose of adequate treatment of the disease to prevent cerebral vascular diseases

without using only methods that affect the formation of neurological deficits in the ham.

Conclusion:

1. There is a linkage between complications of a cerebral stroke and the expectation of micdorin of MMR-2 and MMR-9 enzymes in the mine.
2. The use of biomarkers in the diagnosis and diagnosis of cerebral stroke has an important effect.
3. The use of the enzymes MMR-2 and MMR-9 in prognosis in Tserebro vascular diseases is moofic to maxad.

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