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MEDICINE

PEDAGOGY

THE ROLE OF HYPERGOMOCYSTEINEMIA IN CHRONIC ISCHEMIC STROKE

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Abstract. Recent studies have shown that homocysteine is a more informative indicator of the development of cardiovascular diseases than total cholesterol, and is an independent factor in the formation of both stenocclusive lesions of the main arteries, deep vein thrombosis and microangiopathy, and subsequent cerebrovascular events, especially in patients with diseases coronary arteries, kidneys, patients with type 2 diabetes mellitus.

Keywords: C-reactive protein, hypercomocysteinemia, cognitive impairment, homocysteine..

Hyperhomocysteinuria and hyperhomocysteinemia are associated with defects in the molecules of cystathionine beta synthase and methylenetetrahydrofolate reductase.

The methylenetetrahydrofolate reductase (C677T) gene replaces cytosine with thymidine at position 677, which leads to the replacement of alanine with valine in the apoprotein of this enzyme. This is the most studied variant of the MTHFR gene polymorphism, in which homocysteine in the blood increases. Defects M5, M10-MTHFR in adulthood are observed in 54% of cases among all thrombophilic disorders and lead to hyperhomocysteinemia of intermediate and medium levels (more than 15 μmol/l). However, according to some data, the association of this mutation in the development of cerebrovascular diseases was noted in 16% of cases [3].

The methylenetetrahydrofolate reductase gene (A1298C) is a variant of the MTHFR gene polymorphism with the replacement of adenine by cytosine at position 1298, which is not accompanied by an increase in the level of homocysteine in the blood. However, the combination of heterozygosity for 677T and 1298C alleles is accompanied by an increase in plasma homocysteine levels, a decrease in folate levels, and a decrease in MTHFR enzyme activity.

Genotype differences: The difference between TT and CC MTHFR genotypes results in an average homocysteine difference of 2 μmol/L, which in turn has a 20% difference in stroke risk according to studies. The independent difference between TT and CC genotypes for stroke is 26% [1].

Recent studies have shown that homocysteine is a more informative indicator of the development of cardiovascular diseases than total cholesterol, and is an independent factor in the formation of both stenocclusive lesions of the main arteries, deep vein thrombosis and microangiopathy, and subsequent cerebrovascular events, especially in patients with diseases coronary arteries, kidneys, patients with type 2 diabetes mellitus.

A decrease in the level of pyridoxine, cyanocobalamin, and folic acid in food causes hyperhomocysteinemia not only in homozygous carriers, but also in people without mutations in the homocysteine metabolism genes (low-protein nutrition leads to increased homocysteine remethylation pathways and inhibition of transsulfonation reactions) [2]. A significant role in the development of secondary hyperhomocysteinemia is assigned to nutritional factors, since a diet low in vitamins can lead to blockade of the corresponding metabolic pathways. Concomitant factors are lifestyle, various diseases, taking drugs that lead to changes in the concentration of vitamins in blood plasma, changes in enzyme activity, and kidney function [3].

The use of d-penicylamine, n-acetylcysteine (disulfide replacement), adenosine analogs (inhibit adhomocysteine hydrolase), estrogen (in menopause), simvastatin (to the end unknown mechanism) leads to a decrease in plasma homocysteine. In 20% of patients with ischemic stroke, there is also a decrease in the concentration of homocysteine in the blood plasma.

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