THE SIGNIFICANCE OF FOLIC ACID, HOMOCYSTEIN AND ENDOTHELIN-1 IN THE DEVELOPMENT OF POLYCYSTIC OVARIAN SYNDROME IN WOMEN OF REPRODUCTIVE AGE

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ANNOTATION

The article studies the relationship of folic acid with homocysteine and endothelin-1 in women with polycystic ovary syndrome. Much attention is paid to the role of folic acid in reproductive health in women. According to a study by some authors, it was found that young women with polycystic ovary syndrome have folic acid deficiency, hyperhomocysteinemia, and endothelial dysfunction [2].

Key words: polycystic ovary syndrome, homocysteine, folic acid, endothelin-1, endothelial dysfunction.

ЗНАЧЕНИЕ ФОЛИЕВОЙ КИСЛОТЫ, ГОМОЦИСТЕИНА И ЭНДОТЕЛИНА-1 ПРИ РАЗВИТИИ СИНДРОМА ПОЛИКИСТОЗНЫХ ЯИЧНИКОВ У ЖЕНЩИН РЕПРОДУКТИВНОГО ВОЗРАСТА

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АННОТАЦИЯ

В статье изучается взаимосвязь фолиевой кислоты с гомоцистеином и эндотелином-1 при синдроме поликистозных яичников у женщин. Большое внимание уделено на роль фолиевой кислоты в репродуктивном здоровье у женщин. По исследованию некоторых авторов установлено, что у молодых женщин с синдромом поликистозных яичников имеется дефицит фолиевой кислоты, гипергомоцистеинемия и эндотелиальная дисфункция [2].

Ключевые слова: синдром поликистозных яичников, гомоцистеин, фолиевая кислота, эндотелин-1, эндотелиальная дисфункция.

INTRODUCTION

One of the common ailments in women of reproductive age is the so-called polycystic ovary syndrome, which is an endocrine disease and is accompanied by impaired function of the ovaries, pancreas, adrenal cortex, hypothalamus and pituitary [12, 17].

According to some authors, women with polycystic ovary syndrome have folic acid deficiency of varying severity, as well as increased levels of homocysteine and endothelin-1 [2, 18]. In this regard, the relevance of this problem for further study, identification and elimination of factors contributing to the development of pathology increases.

In the late 1960s, the question of the effect of folic acid on ovarian function was discussed. Then it was proved in animals that an excess or deficiency of folic acid partially suppresses ovulation [16].

Folic acid is a water-soluble vitamin essential for the growth and development of the circulatory and immune systems. Folic acid deficiency can be caused not only by inadequate intake of vitamin B9 from food or malabsorption, but also by specific mutations in MTHFR 677TT, leading to disruption of the folate cycle.

Folate is also considered a vitamin. It is not folic acid itself that exhibits biological activity in the body, but its derivatives, folates, which are converted in the body into their reduced active form [4, 5, 7, 19].

After the intake of folates or folic acid in the body, these compounds are converted by the enzyme 5,10-methylenetetrahydrofolate reductase (MTHFR) into a biologically active form - levomethylfolate. It functions as a methyl group donor. The methyl group attaches to DNA and affects the expression of the MTHFR gene. When a homocysteine molecule receives a methyl group, it is converted into methionine [1, 20, 25].

Homocysteine is an amino acid that is a metabolic product of dietary methionine. Homocysteine does not enter the body with food, so the only source of homocysteine in the body is the conversion of methionine. In excess, homocysteine accumulated in the body can be converted back into methionine.

With a deficiency of folate and folic acid or a violation of their metabolism, the process of converting homocysteine to methionine is disrupted. This leads to the fact that methionine, having worked out in methylation reactions, turns into homocysteine, and has no possibility of recovery. Thus, a deficiency of folic acid in the blood serum is a risk of developing hyperhomocysteinemia, which has toxic effects on a number of tissues in the body [6, 22, 26]. Genetic defects deserve special attention in the development of hyperhomocysteinemia. Mutations in the gene that codes for MTHFR reduce the conversion of folate and folic acid to levomethylfolate. Among some

authors, the possible reasons for the increase in homocysteine levels in polycystic ovary syndrome and its relationship with folic acid continue to be debated [14, 15, 21].

An increase in blood homocysteine occupies one of the leading places among the many factors of endothelial dysfunction. Large concentrations of homocysteine have a toxic effect on cells. Therefore, to prevent cells from damaging its action, it begins to accumulate in the blood, and its main site of damaging action is the inner surface of blood vessels, which leads to the risk of increasing endothelin-1 and the formation of endothelial dysfunction [8]. Endothelin-1 is one of the most powerful vasoconstrictors secreted by the vascular endothelium. The degree of endothelial damage plays an important role in the pathogenesis of polycystic ovary syndrome and possibly affects the development and prognosis [11, 23, 24].

It is known that angiogenesis and folliculogenesis depend on the characteristics of the endothelium [13]. Endothelial dysfunction leads to impaired follicle development and egg maturation, and prevents pregnancy in women with polycystic ovary syndrome [3, 9, 10].

Hence the conclusion that the deficiency of folic acid in the blood serum in patients with polycystic ovary syndrome is a risk of developing hyperhomocysteinemia, which leads to an increase in endothelin-1 and the formation of endothelial dysfunction. And endothelial dysfunction, in turn, leads to impaired blood supply in the pelvic organs, impaired folliculogenesis in the ovaries, anovulation and contributes to the development of long-term complications of this pathology.

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