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Pregnancy and Pregnancy Outcomes in Couples with High and Low Homocystein

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Abstract: It is known that an elevated level of homocysteine in the blood is one of the important markers of various obstetric pathologies. As a result of hyperhomocysteinemia, microcirculation processes in the placenta are disturbed. This, in turn, can lead to implantation defects, resulting in miscarriage.

Keywords: IVF programs, hyperhomocysteinemia, vitamin B12, placentation.

Relevance. Among the many laboratory markers of endothelial dysfunction in polycystic ovary syndrome, homocysteine occupies one of the leading places [3, 4].

Homocysteine (HC) is a sulfur-containing amino acid that is a homologue of the amino acid cysteine. Homocysteine is a product of the conversion of methionine.

As a chemical derivative, it was described in 1932 by chemists Butz and Vigneaud as a product obtained by the reaction of high concentration acid methionine.

Homocysteine does not enter the body with food, therefore, under physiological conditions, 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. The cofactors of the enzymes of the metabolic pathways of methionine in the body are vitamins: folic acid, riboflavin (vitamin B1), pyridoxine (vitamin B6), cyanocobalamin (vitamin B12) [1, 5].

Factors that cause an increase in the level of homocysteine are: genetic - gene mutations that encode the synthesis of an enzyme involved in the formation of homocysteine; non-genetic - autoimmune processes, the use of large amounts of caffeine, kidney disease, smoking, insufficient intake of vitamins B1, B6, B12, folic acid. Homocysteine levels can also rise in the elderly.

Homocysteine has a toxic effect on the cell. To prevent the cell from damaging its action, special mechanisms are triggered to remove it from the cell into the blood. Therefore, if it increases in the body, it begins to accumulate in the blood, and the main site of its damaging effect is the inner surface of the vessels. Under the influence of an increased content of homocysteine, there is an increase in the expression of pro-inflammatory cytokines, a change in the bioavailability of nitric oxide, induction of oxidative stress, activation of apoptosis and defective methylation. Insufficient supply of deoxyribonucleic acid (DNA) methyl groups, protein and lipid methylation impairs the proliferation and differentiation of granulosa cells , thereby inhibiting oocyte and follicular maturation , as well as steroidogenesis in the ovaries.

Homocysteine is a metabolite that has both thrombovascular and atherosclerotic effects. With an increase in the concentration of homocysteine in the blood, it has a damaging effect on the inner wall of the arteries. In these damaged areas, cholesterol begins to be deposited, gradually forming

atherosclerotic plaques. As a result, atherosclerosis develops, and the likelihood of blood clots increases. Microthrombus formation and microcirculation disorders lead to impaired placentation , which can cause infertility as a result of implantation defects in the embryo.

The problem of miscarriage (NB) occupies one of the leading places in modern obstetrics. The frequency of this pathology reaches 20-25% of all pregnancies [5, 9, 10]. In recent years, the issue of the role of hyperhomocysteinemia (HHC) in the pathogenesis of recurrent fetal loss has been widely discussed [13].

Homocysteine (HC) is an essential amino acid that was synthesized at the beginning of the 20th century (De Vigneaud, 1932). In 1962 Carson et al . published for the first time data on disorders of HC metabolism in patients with mental retardation. From that moment began the "era of homocysteine". In 1969 Mudd et al . established the genetic cause of the increase in HC. In 1975 Kilmer McCully confirmed the association of HHC with the development of severe vascular disease. These studies formed the basis of the homocysteine theory of atherosclerosis he proposed . Studies over the past 20 years have expanded the understanding of the role of HHC in the development of vascular disorders in various diseases: thrombovascular disease, myocardial infarction, deep and superficial vein thrombosis, carotid artery thrombosis, Crohn's disease, epilepsy, Parkinson's disease, etc. [1 2] . There are data on the association of HHC with the development of Down syndrome [10].

It is known that HHC negatively affects the reproductive function of both women and men and, as a result, the course of pregnancy [4]. It has been proven *in vitro* that a high level of HC has a direct toxic effect on the endothelium, with an increase in platelet adhesion, deposition of low density lipoproteins in the arterial wall, activation of the coagulation cascade, and disruption of the normal balance of redox reactions [18]. The fundamental role of HC in the processes of cell division and the developing embryo has been proven. Freely penetrating the fetoplacental barrier, HC can lead to the development of secondary autoimmune reactions, thereby provoking the occurrence of various complications of pregnancy, including habitual miscarriages, preeclampsia, premature detachment of a normally located placenta, neural tube defects in the fetus, placental insufficiency, intrauterine growth retardation fetus [14]. HC is one of the main fertility markers of a married couple [15].

According to some sources, the rate of blood HC levels ranges from 10-11 μmol / l. In other sources, the level of HC in blood plasma should be in the range of 5-15 μmol /l. At the same time, it should be noted that the concentration of HC in the blood during life tends to increase, which is associated with a reduced excretory function of the kidneys [5, 6].

In young women with PCOS, a high frequency of hyperhomocyteinemia (HHC) is a risk factor for an increase in the level of ET-1 (endothelin-1) and the formation of endothelial dysfunction, which can lead to impaired blood supply to the pelvic organs, impaired folliculogenesis in the ovaries, anovulation and contribute to the development of long-term somatic complications of this pathology [2]. When studying the marker of endothelial dysfunction ET-1, some authors found that an increase in the concentration of ET-1 occurred in 64 (80%) patients with PCOS, and its content in blood serum (2.4±0.4 fmol/ml) exceeded ($p<0.05$) average indicators of healthy women (0.83±0.2 fmol /ml) [2].

According to some studies, the study of the content of HC in the blood serum showed that its levels in women of the control group averaged 8.1 ± 0.2 μmol / l and coincide with a number of some authors who believe that the normal concentration in the blood of HC in women of reproductive age should not exceed 8-10 μmol /l [11]. In patients with PCOS, the level of HC averaged 10.3±0.4 μmol /l and significantly ($p< 0.05$) exceeded that of healthy women, 8.1±0.2 μmol /l. In 33 (50.8%) patients with PCOS, the blood levels of HC exceeded 10 μmol /l and averaged (12.2±0.3 μmol /l). Consequently, half of the women with PCOS had HHC [2].

However, there are conflicting data on the concentration of HC in patients with PCOS: while some researchers speak of an increase in the concentration of HC compared with healthy women [7], others deny the presence of hyperhomocysteinemia (HHC) in this group of patients [9].

Data on elevated serum levels of HC in women with PCOS, leading to disruption of follicle development and egg maturation, continue to be debated [8, 10].

Thus, an increase in the concentration of homocysteine in women with PCOS is a reason for further in-depth study of its secretion in order to improve treatment methods.

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