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Influence of Vitamin D Deficiency on a Woman's Health State and its Reproductive Function

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Vitamin D plays a huge role in the life of the female body:

- vitamin D regulates the synthesis and secretion of progesterone, as well as sensitivity to follicle-stimulating hormone;
- the optimal level of vitamin D in the blood 30ng/25(OH)D ensures the optimal thickness of the endometrium for implantation of the embryo
- the optimal level of vitamin D in the blood provides local immunity and is a preventive measure for bacterial vaginosis and increases the effectiveness of its treatment;
- the optimal level of vitamin D in the blood is necessary for the normal reproductive function of women.

A high percentage of women of reproductive age are deficient in vitamin D, and pregnant women are at an even higher risk of deficiency.

It has been proven that vitamin D deficiency causes infertility, and also affects the course of pregnancy and the health of offspring (intrauterine and later life).

Vitamin D deficiency during pregnancy is associated with placental insufficiency, increased risk of emergency cesarean section, low birth weight, risk of gestational diabetes, preterm birth, and preeclampsia. Vitamin D deficiency in the fetus depends on maternal vitamin D levels.

4000 IU of vitamin D per day is most effective in improving the health status of pregnant women with vitamin D deficiency, with circulating vitamin D levels as high as 40 ng/mL.

Vitamin D metabolism is increased during pregnancy and lactation. From the 4th week of pregnancy, vitamin D is transferred through the placenta and the concentration of 25 (OH) D in the umbilical cord blood of the fetus correlates with its concentration in the mother. Vitamin D regulates the main target genes associated with the implantation of the fetal egg, plays a role in the formation of immunotolerance.

The level of vitamin D in the blood serum of women in the 3rd trimester of pregnancy is 2 times higher than in non-pregnant women.

Vitamin D deficiency is associated with poor pregnancy outcomes. Hypertension during pregnancy and especially preeclampsia (PE) are the most studied complications of pregnancy associated with vitamin D deficiency in women. Vitamin D3 levels less than 20 ng/mL at 16 weeks of gestation are associated with a 2.7-fold increased risk of developing gestational diabetes in late pregnancy.

Polycystic ovary syndrome and vitamin D

A group of Italian researchers (Ferrari et al. 2014) concluded that vitamin D (D-hormone) deficiency may be the cause of IVF failure. The authors observed 335 women treated with IVF in 2012. After analyzing blood levels of vitamin D, the researchers identified 154 women in the vitamin D deficient group and 181 women in the vitamin D adequate group. The authors considered normal levels of

vitamin D to be twice as likely to become pregnant after IVF compared to women with vitamin D deficiency.

The researchers concluded that sufficient levels of vitamin D lead to the “maturation” of quality eggs in the ovaries and more successful implantation of embryos, which are of higher quality in women with adequate levels of the vitamin.

Infertility in Women and Vitamin D Deficiency

Polycystic ovary syndrome (PCOS, Stein-Leventhal syndrome, hyperandrogenic ovarian dysfunction) is a condition characterized by chronic anovulation or menstrual irregularities and ovarian hyperandrogenism in the absence of other causes of androgen hyperproduction, the development of polycystic ovaries.

The incidence of PCOS is 3-8% among all diseases of the reproductive system. In the structure of anovulatory infertility, the frequency of PCOS reaches 50%, with menstrual disorders - 12%, with hyperandrogenism - 80-90%.

The peak incidence occurs at the age of 18-30 years. In 1935, Stein and Leventhal described the classic form of "sclerocystic ovaries" as a syndrome of amenorrhea and enlarged ovaries, combined with hirsutism, infertility, and obesity.

With PCOS, there is a risk of developing certain diseases and conditions:

- ✓ insulin resistance, impaired glucose tolerance or overt diabetes mellitus;
- ✓ visceral obesity;
- ✓ non-alcoholic fatty liver disease;
- ✓ obstructive sleep apnea syndrome and daytime sleepiness;
- ✓ depressive states;
- ✓ development of cardiovascular pathology with an increased risk of hypertension, coronary artery disease, including myocardial infarction and circulatory failure;
- ✓ risk of developing endometrial and ovarian cancer.

The main clinical manifestations of PCOS are: violation of the menstrual cycle; infertility; hypertrichosis; obesity; baldness; "black acanthosis" (skin changes in the back of the neck, armpits, under the mammary glands, characterized by an ash-gray or brownish color, accentuated skin pattern, its folding and fringed growths, sometimes the appearance of papillomas); numerous acne; impaired glucose tolerance; insulin resistance.

The development of PCOS is based on excessive production of androgens by ovarian tissue. Androgens are synthesized in the theca cells of the follicles and in the stroma. They regulate the synthesis of androgens LH (luteinizing hormone), IGF-1 (insulin-like growth factor-1), the enzyme P450c17 (17 a-hydroxylase and C20-22-lyase) is a key enzyme in the synthesis of androgens in the ovaries and adrenal glands. Currently, the leading theory of the pathogenesis of PCOS is the theory of insulin resistance and hyperinsulinemia.

Insulin and IGF-1 stimulate the synthesis of LH and increase the sensitivity of the ovaries to it, contributing to an increase in LH-dependent androgen synthesis in the theca and stroma cells. Insulin can also activate ACTH - dependent cytochrome P450a in the adrenal glands, increase the synthesis of a sex hormone-binding protein by the liver, which leads to an increase in free androgen fractions in the blood. The synergism of the action of insulin and LH causes stimulation of theca cells, hyperandrogenism and cystic atresia of the follicles.

There is now evidence of an important role of vitamin D deficiency in the development of PCOS. Vitamin D deficiency contributes to the development of insulin resistance, which plays a leading role in the pathogenesis of PCOS. Patients with PCOS showed an inverse correlation between vitamin D levels and insulin resistance, body mass index, blood triglycerides, total testosterone and

dehydroepiandrosterone, and a positive correlation with insulin sensitivity. It can be assumed that vitamin D levels affect luteinizing hormone and insulin resistance.

A positive effect of vitamin D on insulin secretion, lipid profile, decrease in glucose and C-peptide levels, menstrual cycle and follicle development has been shown. Vitamin D deficiency and the beneficial effects of vitamin D treatment are clearly seen in the presence of obesity in PCOS.

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