The Effect and Significance of Vitamin D on the Course and Effects of Pregnancy

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ABSTRACT

Background. The high prevalence and negative effects of vitamin D deficiency is a problem for a wide range of specialists worldwide. Pregnant and lactating women are at risk for vitamin D deficiency, as evidenced by the results of many epidemiological studies. Vitamin D plays an important role in providing the mother-placenta-fetus system, including implantation, placenta formation, embryogenesis, intra- and postnatal periods. Vitamin D deficiency leads to placental insufficiency, low birth weight, gestational diabetes, premature birth, preeclampsia, and an increase in the inflammatory response.

The purpose of the study: an analytical review of the role and importance of vitamin D during pregnancy in modern scientific literature.

Materials and methods. An analysis of 18 foreign literary sources on this topic was carried out using vitamin D [6,7]. This indicates a high level of SAMR. It suppresses the synthesis of protivovospalitelnogo

Conclusion. Vitamin D plays an important role in the system of pregnant women, the placenta and the fetus, especially during implantation, placenta formation, embryogenesis, intra- and postnatal period. According to the literature review: Low level 25 (ON) D has an adverse effect on the course of pregnancy, outcome, fetal development, and infant health. Eliminating vitamin D deficiency has a positive effect on pregnancy outcomes and baby’s health, i.e. serious complications during pregnancy - preeclampsia, gestational diabetes, placental insufficiency, chronic infection and other diseases are significantly reduced.

KEYWORDS: 25 (OH) 2D, 1.25 (OH) 2D, vitamin D deficiency, preeclampsia, gestational diabetes

INTRODUCTION

High prevalence and negative consequences of vitamin D deficiency is a problem for many professionals in the world. Vitamin D deficiency is associated with health risks, including cardiovascular disease, cancer, metabolic syndrome, bone marrow, autism, multiple sclerosis, diabetes and mental health. It is proved that vitamin D deficiency causes infertility, affects the longevity of pregnancy and good health (in the womb and in the late adulthood). [1]

Pregnant and breastfeeding women are exposed to the risk of vitamin D deficiency, which is confirmed by the results of many epidemiological studies. Vitamin D plays an important role in supporting the maternal-placental-fetal system, including implantation, formation of the placenta, embryogenesis, intra and postnatal periods. Deficiency of vitamin D leads to placental insufficiency, birth of children with low body mass, gestational diabetes, preterm birth, preeclampsia, inflammatory reactions. [2]

Materials and methods

An analysis of 18 sources of foreign literature on this topic was carried out. Influence of vitamin D on satellites, especially on trophoblast cells. The placenta contains all the components of the signaling system of vitamin D (VDR, RXR, CYP27B1 and CYP24A1). Weisman et al. [2]

It was found that detsiudalnya a tissue in the placenta is able to synthesize 1,25 (OH) 2D and 24,25 (OH) 2D, which predispose to the formation of cytokines, granulocytes, macrophages 2 (GMCSF-2), stimulating colony a (TNF-). a, suppresses the synthesis of protivovospalitelnogo interleykina-6 (IL-6) and increases the indicators of TsAMF in detsiudalnyx cells and cytotrophoblastax [3, 5, 6].

In the case of using vitamin D after exposure to E. coli on the third row of trophoblast cell cultures, a relatively low infection rate was confirmed compared to the case without using vitamin D [6,7]. This indicates a high level of SAMR. It follows that adding vitamin D to the diet with a serum 25 (OH) D level <30 ng/ml (vitamin D deficiency or deficiency) during pregnancy may reduce infectious processes.

Functions of vitamin D during pregnancy

The satellite has been proven to be capable of synthesizing 1.25 (OH) 2D. Placental product - 1.25 (PO) 2D. During pregnancy, against the background of massive synthesis of this vitamin, the rate of entry of this vitamin into the kidneys increases, which leads to the accumulation of ego [7].

Observations have shown that the level of 1.25 (OH) 2D in a recent series in the third trimester of pregnancy was twice as high as in non-pregnant women and in the postpartum period [8, 9]. 1.25 (OH) 2D is a link in the auto- and paracrine immune systems and has been shown to be synthesized during pregnancy [10]. 1.25 (OH) 2D inhibits the release of Th1 cytokines and increases the amount of Th2 (Th2 cytokines predominate during implantation) [10, 11]. This modulation of the immune system prevents the implanted embryo from separating. 1.25 (OH) 2D also promotes the transformation of the endometrium into decidual cells [12, 13]. An important gene for embryonic implantation and myeloid differentiation in early gestational age increases the synthesis of HOXA10 [13, 14]. It was reported that 1.25 (OH) 2D-synctiotrophoblasts control secretion, secretion and increase the synthesis of steroid
hormones in the placenta by human chorionic gonadotropin [4]. It has been shown that calcitriol promotes calcium transport to the placenta, increases the secretion of lactogen in endometrial stromal cells. As a result of this process, the development of the endometrium and the improvement of the implantation process are ensured [4]. These data confirm that 1,25 (OH) 2D promotes implantation, maintains normal pregnancy, promotes fetal growth through calcium delivery, controls the secretion of non-colonic formulations, and acts on hormones.

Vitamin D deficiency leads to a number of negative complications: secondary hypertension, gestosis [15, 17], an increase in the frequency of caesarean section and spontaneous premature birth, the development of bacterial vaginosis in early pregnancy [19], and an increase in the incidence of diseases such as gestational diabetes. Gestosis is one of the most common obstetric complications and plays an important role in maternal and infant mortality. At the heart of gestosis are mechanisms such as impaired trophoblast invasion, decreased placental perfusion, endothelial dysfunction, and oxidative stress. The presence of vitamin D and its receptors in the placenta, as well as the participation of vitamin D in the processes of immune, inflammatory and vascular resistance justify its role in the pathogenesis of gestosis in pregnant women [14, 16, 17]. In women, high vitamin D levels reduce the incidence of gestosis, and when it occurs, blood pressure readings are lower. The dose of 25 (OH) D below 20 ng / ml during pregnancy is four times higher, and less than 15 ng / ml is five times higher than with the development of severe gestosis. M. Haugen et al. A study of 23,423 women in labor in Norway showed that the risk of developing the disease was 27% lower in the group of women who received 400-600 IU of vitamin D per day than in the group that did not receive it. Lack of vitamin D leads to a deficiency of sex hormones, a violation of the ratio of body fat (prolactin, insulin, cortisol) and fat-breaking factors (somatotropin, catecholamines, sex hormones, thyroid hormones) factors [18].

One large study found an inverse relationship between vitamin D supplementation in a pregnant woman’s diet and spontaneous preterm labor: High doses of vitamin D reduced the likelihood of miscarriage [20]

**Conclusion**

Vitamin D plays an important role in the pregnant woman-placenta-fetus system, especially during implantation, placenta formation, embryogenesis, intra- and postnatal period. Literature review Low levels of 25 (ON) D indicate adverse effects on pregnancy, outcome, fetal development and infant health. Elimination of vitamin D deficiency has a positive effect on the outcome of pregnancy and the health of the baby, that is, on serious complications of pregnancy - preeclampsia, gestational diabetes, placental insufficiency, chronic infection and others. The incidence is significantly reduced.

**Literature**

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