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PECULIARITIES OF THYROID FUNCTION IN PREGNANT WOMEN WITH IODINE DEFICIENCY

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Abstract: Iodine is a key element for thyroid hormones synthesis. Adequate iodine provision is essential for thyroid gland normal structure and function. Thyroid dysfunction in pregnant women, including those with iodine deficiency, can lead to gestisis, chronic intrauterine hypoxia, the threat of termination of pregnancy, fetal abnormalities. Physiological requirement of iodine during pregnancy, lactation, in childhood is increasing, and the body needs more iodine supply. In such cases, individual iodine prophylaxis with iodine drugs should be provided.

Key words: thyroid gland, pregnancy, pregnant woman, iodine preparations.

Iodine deficiency diseases (IDD) - all pathological states that develop in a population as a result of iodine deficiency, which can be prevented with normal iodine intake. It has been proven that starting from the 11th–12th week of pregnancy, thyroid gland (TG) of the fetus acquires ability to accumulate iodine, synthesize and secrete thyroid hormones, that is, by the 4th month of embryogenesis, it mainly reaches structural and functional maturity. First three months of intrauterine development fetal thyroid hormones are carried out mainly by the mother. How much the fetus will be fully provided with thyroid hormones mother directly depends on whether she experiences she is deficient in iodine in the diet. Iodine is an essential element for the synthesis of hormones thyroid. Adequate iodine supply throughout the whole life of a person - in fact - a guarantee of normal small structure and function of the thyroid gland.

Impaired thyroid function in pregnant women menstrual women, including those with iodine deficiency can lead to preeclampsia, chronic intrauterine hypoxia of the fetus, the threat of abortion mentality, fetal developmental anomalies (D. Glinoer, 2003), disturbances in the formation of the central nervous system in the fetus, the main structures of which are formed before 10-12 weeks of pregnancy, that is, when providing iodine to the fetus depends entirely on maternal iodine intake. [1,3]

Physiological changes in the thyroid gland during pregnancy.

The conditions for the functioning of the thyroid gland are already changing from the first weeks of pregnancy, which is associated with influence of certain factors. First of all with the onset and development of pregnancy, in the first half of it, the production of chorionic gonadotropin (CG) by the placenta, especially pronounced naya with multiple pregnancy. CG, analog flexible in тstructure to the -subunit of thyrotropic [4].

Increased estrogen production during pregnancy stimulates the production of thyroxine by the liver binding globulin (TSG). TSH binds additional a significant amount of free fractions of thyroid hormones and due to a transient decrease in their level causes stimulation of TSH to maintain normal function of the thyroid gland. At the same time, the level of common fractions thyroid hormones will stably remain lofty. Increased

use of iodine in fetoplacental complex for the synthesis of fetal thyroid hormones and an increase in renal clearance of iodine cause additional additional stimulation of the thyroid gland of a pregnant woman. The thyroid hormones of a pregnant woman themselves are subject to while being metabolized in the placenta, they can also become Additional source of iodine for hormone synthesis Fetal thyroid gland, which is especially important in conditions of deficiency iodine. [2,3,5]

The most severe consequence of iodine deficiency in the perinatal period is endemic (neurological) cretinism is an extreme degree of support for mental and physical development. Endemic cretinism, as a rule, is characteristic for regions with severe iodine deficiency. In re-regions of moderate iodine deficiency are observed subclinical disorders of intellectual development orgy. Differences in IQ scores between populations eat living in iodine-deficient and iodine-deficient liver regions, average 13.5% points.

Thus, the consequences of reduced iodine intake during pregnancy are:

- chronic stimulation of the thyroid gland in both women and and in the fetus with the subsequent formation of goiter;
- relative hypothyroxinemia;
- violations of the formation of the central nervous system in the fetus;
- problems of the course of pregnancy and childbirth;
- Fetal anomalies.

Results of research conducted by FGU ENTS in 2006–2008 show:

- diffuse non-toxic goiter is the most common thyroid disease among pregnant women living in the territory riah with the presence of iodine deficiency;
- frequency of occurrence of diffuse goiter in pregnant changeable women in the surveyed regions of the Russian Federation varied from 8.9 to 35.9%;
- The level of median ioduria in pregnant women is practically chesky everywhere did not exceed 100 mcg/l and varied within 72.5–95.5 µg/l, which indicates inadequate intake of iodine by pregnant women.

The first factor is the need to support during pregnancy, the concentration of thyroxion, the daily production of which, with physiological during the course of pregnancy increases to 75–150 mcg, which corresponds to 50–100 mcg of iodine. The next factor is due to the fact that the fetus, according to as it develops, needs more and more quantities of iodides for the synthesis of their own thyreoid hormones. The number of transfers from the organization maternal fetal thyroxin before the onset of thyroid gland of the fetus is not determined divided, however, about 40% of this hormone, measured leg in the umbilical cord at birth, it turned out maternal origin. Number of transmission the amount of iodide administered to the fetus is also difficult to establish, since as the content of iodine in the thyroid gland of the fetus before 17 weeks less than 2 mcg, then increased to 300 mcg to the moment of birth, and the production of thyroxin in the middle it is 500 mcg / day. Necessary additional a significant amount of thyroxin in newborns with reduced function of the thyroid gland 50–75 mcg/day, on the basis of which it is possible to make conclude that the amount transmitted per day iodide is about 50 mcg. According to In Institute of Medicine, American Academy of Sciences this figure was 75 mcg/day. The third factor contributing to the increase in the needs of a woman's body during pregnancy iodine content is associated with an increase in their renal blood flow, which increases excretion iodine in the urine. It should also be emphasized that there is no data on possible deposition and withdrawal iodide in the placenta. [10,11]

With iodine deficiency during pregnancy in women a goiter may form in the chest (in 10-20%). Presumably positively, this is one of the reasons for the higher distribution strangeness of thyroid diseases in females

compared to men. Thyroid pathology in an average of 17% of women before pregnancy leads to disorders menstrual cycle, reduced fertility, development of the syndrome of hyperprolactinemic hypogonadism (in parallel with iodine deficiency increases production of thyreoliberin and prolactin, which leads to hypogonadism). The presence of thyroid pathology during pregnancy often leads to miscarriage, the threat of interruption pregnancy, early toxicosis, preeclampsia develops in 54.5% of pregnant women, fetal distress, hypo- fetal trophy - in 22.7%, birth anomalies occur activities such as uncoordinated labor activity (in 35.2%), weakness of labor activity, preterm birth (18.2% of cases) [7, 8]. In mothers with thyroid diseases, developmental anomalies fetuses occur quite often and account for 18-25%. Most often, lesions are observed on the part of the central tral nervous (hydrocephalus, microcephaly, Down's disease, functional disorders) and endocrine systems (congenital hypothyroidism, thyrotoxicosis). Diffuse toxic goiter (DTG) during pregnancy ness occurs in 0.05–3% of cases. It's autoimmune a disease that is caused by excessive secretion thyroid hormones and diffuse increase thyroid. The diagnosis of DTG is confirmed by the corresponding clinical picture, ultrasound of the thyroid gland (diffuse organ), high blood levels of free T3 and T4, the presence of thyroid-stimulating immunoglobulins. The disease of pregnant women with DTG has no negative impact on the course of pregnancy and on fetus: increased risk of stillbirth, premature birth and the development of preeclampsia; increased neonatal mortality and the likelihood the birth of a child with low body weight. Occurs the risk of developing a thyrotoxic crisis during contractions and in the pressing period, an increased risk of fetal malformations. Perhaps the development of intrauterine thyrotoxic goat after 22–25 weeks of gestation, since during this period one function of the mature thyroid gland of the fetus is stimulated by a large number of thyroid-stimulating immunoglobulins new from mother's blood. Fetal thyrotoxicosis diagrated based on an increase in heart ratefetal contractions (more than 160 per minute), increased thyroid hormone levels in amniotic fluidsti, the presence of goiter in the fetus with ultrasound.[8,12]

Lack of adequate treatment for thyrotoxicosis, development of the disease up to 20 years of age, lasting the duration of the disease for more than 10 years, its presence with gestational age over 30 weeks of gestation - osnew risk factors for the birth of children with small soy. In children of mothers with untreated thyrotoxicosis an increase in the incidence of neonatal thyrotoxicosis, which is clinically manifested tachycardia, growth retardation, increased bone age, goiter, premature cranioctenosis. Compensation for thyrotoxicosis in a future mother any stage of pregnancy is necessary condition for the successful completion of pregnancy for both mother and fetus. DTZ in some casesteas tends to spontaneous remission with pregnancy due to immunosuppressive effect of pregnancy. In severe forms of thyroid toxicosis pregnancy is contraindicated [11, 12].

No less urgent problem is the presence which causes primary hypothyroidism in a pregnant woman, causes which are most often AIT and the state after thyroid resection for DTG or euthyroid ultrasound goiter. Hypothyroidism due to AIT is usually detected and compensated before pregnancy, although often diagnosed during pregnancy. Due to the immunosuppressive action of the remission of AIT for this period is possible and relapse after childbirth. Uncompensated hypothyroidism during pregnancy has a negativeinfluence on the body of a woman and a fetus. Maybe the development of such complications of pregnancy as anestheticmia, preeclampsia, premature detachment of normal poorly located placenta, postpartum blood leakage, dysfunction of the cardiovascular systems. Since there is a transplacental transfer of blocking antibodies to the receptors to the fetus TSH, fetal and neonatal hypotension developsrheosis [13].

Fetal hypothyroidism presents with growth retardation fetus, bradycardia, delayed appearance of nuclei of the eye stenosis, violation of the processes of development of the central fetal nervous system. Neonatal hypothyroidism long-term 1–4 months, corresponds to the half-life of ma-Therin antibodies to fetal thyroid. Signs of neonatal leg hypothyroidism are post-term pregnancy (greater than 42 weeks), high birth weight

(more than 4 kg), macroglossia, peripheral cyanosis, swelling, shortness of breath, rough voice during crying and screaming time, drowsiness, decreased activity, hypothermia, dry skin, low levels of tirheoid hormones in the blood of the newborn. There is a pathology of the thyroid gland, which does not depend on iodine supply (it is 5%), - after- generic thyroiditis. Finally, the etiology arose This state of affairs has not been elucidated. However, to risk factors include the presence of autoantibodies to tissue thyroid gland, the presence of HbA markers and lymphocytic in- thyroid filtration, as well as a belated decrease in immune suppression, decreased activity of thyrocytes. For normal fetal development throughout pregnancy, and especially at the early stages of embryogenesis, a normal level of vein of thyroid hormones in the maternal body. At the same time, adequate and timely correction treatment of various pathological conditions of the thyroid gland in pain In most cases, it allows normal function regulation of the female reproductive system and pregnancy with minimal risk of developing pafetal tology. Diagnosis and study of thyroid pathology during pregnancy faces certain difficultiesties that are associated with changes in metabolism thyroid hormones during pregnancy and dynamic by the interaction of the pituitary-thyroid systems maternal and fetal stems. There is no consensus on the recommended change of applied doses for substitution therapy PII in the pathology of the thyroid gland during pregnancy. Not-The question of the possible damaging effect of antithyroid autoantibodies on the thyroid of the fetus and newborn. According to one data, antibodies to TPO, penetrating the placenta, can call hypofunction of the thyroid gland, according to others – transient hyperthyroxinemia [2,8].

Prevention at the scale of groups of increased risk for the development of IDD (children, pregnant and lactating women) is carried out by taking lekarstny preparations containing physio-dose of potassium iodide. Fundamentally important is that it is allowed to use exactly pharmacological preparations containing precisely standardized dose of iodine, not biologically active iodine supplements. In addition, in the above among certain groups of the population, there is a particularly high distribution strangeness of diffuse iodine deficiency goiter and, therefore, taking drugs with an accurate dosage which potassium iodide has not only preventive sskoe, but also medicinal value. Thus, all women of reproductive age living in a region with natural iodine deficiency, it is recommended to use iodized salt in the diet. In pregnant women living in conditions of pensioned natural iodine deficiency, in a set of measures for managing pregnancy it is necessary to include diagnostics of the state of the thyreoid system using ultrasonic follow-up of the thyroid gland and laboratory control of its function according to the levels of TSH, St. T4 in thekah genetic screening of the condition of the fetus, for detection of goiter and hypothyroxinemia as factors risk of obstetric and perinatal complications. To improve obstetric and perinatal outcomes dows in pregnant women living in the region of natural iodine deficiency, it is necessary to carry out iodine preventive measures during pregnancy and all lactation period, which provides for a dailydaily intake of 250 micrograms of potassium iodide Recommended doses of potassium iodide for prevention in groups of increased risk for the development of IDD [10,14]

- Children under 12:

Potassium iodide orally 50–100 mcg once a day for a long seriously.

- Teenagers and adults:

Potassium iodide inside 100–200 mcg once a day for a long seriously.

- Pregnant and lactating women:

Potassium iodide orally 250 mcg once a day (all period of pregnancy and lactation).

Complications and side effects from the use the above prophylactic dosing there is no potassium iodide.

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